DNALYSIS Biotechnology

PATIENT INFORMATION
NAME: John Doe
DOB: 04/Mar/1976
SEX AT BIRTH: Male

SPECIMEN DETAILS
BARCODE: DNADUMMY2ZA
SAMPLE ID: DUMMY2
TYPE: DBS
COLLECTED: 04/Apr/2025

ORDERED BY
Private
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This pharmacogenetic information is based on best evidence compiled from guidelines and databases including the FDA Table of Pharmacogenetic Associations and the Clinical Pharmacogenetics Implementation Consortium (CPIC). In some cases, PharmGKB and the Dutch Pharmacogenetics Working Group (DPWG) may also be referenced.
Please refer to the Methods, Limitations, and Liability Disclaimer at the end of this report.

Current Medications

The medications listed below indicate the patient's **Current Medications**.

1 Mild/no drug-gene or Minor drug-drug interaction

2 Moderate drug-gene or drug-drug interaction

3 Serious drug-gene or Major drug-drug interaction

Allopurinol	Phenotype	Genetic Test	Results	Source/Evidence Level
Aloprim Lopurin Zyloprim	Increased risk of adverse drug reactions	HLA-B*58:01	Positive	CPIC A[42]; FDA 2[47]

TreatGx
ReviewGx

Drug-Gene Interactions:

3 CPIC – Implication: Significantly increased risk of allopurinol-induced severe cutaneous adverse reaction (SCAR).

3 CPIC – Strong Recommendation: Allopurinol is contraindicated.

Clopidogrel	Phenotype	Genetic Test	Results	Source/Evidence Level
Plavix	Rapid metabolizer	CYP2C19	*1/*17	CPIC A[27]; FDA 1[47]

TreatGx
ReviewGx

Drug-Gene Interactions:

1 CPIC – Implication: Normal or increased clopidogrel active metabolite formation; normal or lower on-treatment platelet reactivity; no association with higher bleeding risk.

1 CPIC – Strong Recommendation for acute coronary syndrome (ACS) and/or percutaneous coronary intervention (PCI): If considering clopidogrel, use at standard dose (75 mg/day).
No recommendation for non-ACS, non-PCI cardiovascular indications. ACS and/or PCI includes patients undergoing PCI for an ACS or non-ACS (elective) indication. Non-ACS, non-PCI cardiovascular indications include peripheral arterial disease and stable coronary artery disease following a recent myocardial infarction outside the setting of PCI.

Drug-Drug Interactions:

2 (Codeine)-(Clopidogrel) Codeine can cause a decrease in the absorption of Clopidogrel resulting in a reduced serum concentration and potentially a decrease in efficacy.

Codeine	Phenotype	Genetic Test	Results	Source/Evidence Level
Codeine Contin Tylenol with Codeine No. 2/3/4	Intermediate metabolizer	CYP2D6	*2.019/*4.001	CPIC A[13]; FDA 1[47]; FDA 2[47]

TreatGx
ReviewGx

Drug-Gene Interactions:

1 CPIC – Implication: Reduced morphine formation.

1 CPIC – Moderate Recommendation: Use codeine label recommended age-specific or weight-specific dosing. If no response and opioid use is warranted, consider a non-tramadol opioid.

Drug-Drug Interactions:

2 (Codeine)-(Clopidogrel) Codeine can cause a decrease in the absorption of Clopidogrel resulting in a reduced serum concentration and potentially a decrease in efficacy.

Medication Summary Table

The Medication Summary Table lists medications with pharmacogenetic associations, organized by therapeutic area and drug-gene interaction severity. Some medications appear in multiple columns within moderate drug-gene interactions due to various possible effects or associations with multiple genes. The highest severity is prioritized (moderate/severe) from all relevant sources and genes for a medication.
Warfarin appears in multiple columns because its dosing cannot be predicted based on PGx alone and other factors may increase or reduce dose requirement. See Medication Report for full details.

	1 Mild or no known drug-gene interaction	2 Moderate drug-gene interaction: Consider alternative medications	2 Moderate drug-gene interaction: May require an increased dose	2 Moderate drug-gene interaction: May require a reduced dose	2 Moderate drug-gene interaction: May reduce efficacy	2 Moderate drug-gene interaction: May increase adverse events	3 Serious drug-gene interaction: avoid/select alternative
Analgesia	Carisoprodol Celecoxib Codeine Flurbiprofen Hydrocodone Ibuprofen Meloxicam Oliceridine Oxcarbazepine Piroxicam Tenoxicam Tramadol Venlafaxine	Amitriptyline Imipramine		Alfentanil Desipramine Fentanyl Morphine Nortriptyline	Amitriptyline Fentanyl Imipramine	Amitriptyline Desipramine Imipramine Lamotrigine Nortriptyline	Carbamazepine
Anesthesia	Desflurane Isoflurane Methoxyflurane Sevoflurane Succinylcholine						
Autoimmune	Azathioprine Etanercept Mercaptopurine Siponimod Thioguanine		Tacrolimus	Cyclosporine		Methotrexate	
Cancer	Capecitabine Erdafitinib Fluorouracil Gefitinib Irinotecan Mercaptopurine Pazopanib Thioguanine	Tamoxifen	Tamoxifen		Tamoxifen	Methotrexate	
Cardiovascular							

	1 Mild or no known drug-gene interaction	2 Moderate drug-gene interaction: Consider alternative medications	2 Moderate drug-gene interaction: May require an increased dose	2 Moderate drug-gene interaction: May require a reduced dose	2 Moderate drug-gene interaction: May reduce efficacy	2 Moderate drug-gene interaction: May increase adverse events	3 Serious drug-gene interaction: avoid/select alternative
	Acetylsalicylic acid Atorvastatin Carvedilol Clopidogrel Fluvastatin Lovastatin Mavacamten Metoprolol Nebivolol Pitavastatin Pravastatin Propranolol Rosuvastatin Simvastatin	Propafenone	Warfarin	Flecainide Propafenone Warfarin	Flecainide Propafenone Warfarin	Flecainide Propafenone Warfarin	
Endocrinology	Nateglinide						
Gastroenterology	Dronabinol Esomeprazole Metoclopramide Ondansetron Rabeprazole		Dexlansoprazole Lansoprazole Omeprazole Pantoprazole	Meclizine	Dexlansoprazole Lansoprazole Meclizine Omeprazole Pantoprazole	Meclizine Methotrexate Prochlorperazine Promethazine	
Infection	Abacavir Atazanavir Dapsone Nitrofurantoin Primaquine Tafenoquine			Efavirenz	PEG-interferon alpha	Efavirenz	Voriconazole
Mental Health	Alprazolam Amoxapine Amphetamine Bromazepam Chlordiazepoxide Clonazepam Clorazepate Diazepam Fluoxetine Flurazepam Lofexidine	Amitriptyline Citalopram Clomipramine Doxepin Escitalopram Imipramine Trimipramine	Atomoxetine Citalopram Escitalopram	Desipramine Nortriptyline Paroxetine Zuclopenthixol	Amitriptyline Atomoxetine Bupropion Citalopram Clomipramine Doxepin Escitalopram Imipramine Methylphenidate Olanzapine Risperidone	Amitriptyline Aripiprazole Aripiprazole lauroxil Asenapine Brexpiprazole Cariprazine Chlorpromazine Clomipramine Clozapine Desipramine Doxepin	Carbamazepine

	1 Mild or no known drug-gene interaction	2 Moderate drug-gene interaction: Consider alternative medications	2 Moderate drug-gene interaction: May require an increased dose	2 Moderate drug-gene interaction: May require a reduced dose	2 Moderate drug-gene interaction: May reduce efficacy	2 Moderate drug-gene interaction: May increase adverse events	3 Serious drug-gene interaction: avoid/select alternative
	Lorazepam Nicotine replacement therapy Nitrazepam Oxazepam Oxcarbazepine Protriptyline Sertraline Temazepam Triazolam Venlafaxine Viloxazine				Trimipramine	Flupentixol Fluphenazine Fluvoxamine Haloperidol Iloperidone Imipramine Lamotrigine Loxapine Lurasidone Methotrimeprazine Molindone Nortriptyline Olanzapine Paliperidone Paroxetine Perphenazine Pimozide Quetiapine Risperidone Thioridazine Thiothixene Trifluoperazine Trimipramine Vortioxetine Ziprasidone Zuclopenthixol	
Neurology	Brivaracetam Clobazam Clonazepam Deutetrabenazine Diazepam Donepezil Fosphenytoin Galantamine Metoprolol Oxcarbazepine Phenytoin Pitolisant Propranolol Tetrabenazine Valbenazine	Amitriptyline			Amitriptyline	Amitriptyline Lamotrigine	Carbamazepine

	1 Mild or no known drug-gene interaction	2 Moderate drug-gene interaction: Consider alternative medications	2 Moderate drug-gene interaction: May require an increased dose	2 Moderate drug-gene interaction: May require a reduced dose	2 Moderate drug-gene interaction: May reduce efficacy	2 Moderate drug-gene interaction: May increase adverse events	3 Serious drug-gene interaction: avoid/select alternative
	Venlafaxine						
Respiratory	Salmeterol						
Rheumatology	Azathioprine Celecoxib Etanercept Flurbiprofen Ibuprofen Meloxicam Pegloticase Piroxicam Rasburicase Tenoxicam					Methotrexate	Allopurinol
Urology	Darifenacin Fesoterodine Mirabegron Tamsulosin Tolterodine				Sildenafil		
Other	Abrocitinib Avatrombopag Cevimeline Elagolix Eltrombopag Flibanserin Lusutrombopag Methylene blue Oral contraceptives						Eliglustat

Medication Summary

The Medication Summary lists medications with pharmacogenetic associations, organized by therapeutic area and drug-gene interaction severity. The highest severity is prioritized (moderate/severe) from all relevant sources and genes for a medication. See Medication Report for full details.

1 Mild or no known drug-gene interaction

2 Moderate drug-gene interaction

3 Serious drug-gene interaction: avoid/select alternative

Analgesia

1
Carisoprodol
Celecoxib
Codeine
Flurbiprofen
Hydrocodone
Ibuprofen
Meloxicam
Oliceridine
Oxcarbazepine
Piroxicam
Tenoxicam
Tramadol
Venlafaxine

2
Alfentanil
Amitriptyline
Desipramine
Fentanyl
Imipramine
Lamotrigine
Morphine
Nortriptyline

3
Carbamazepine

Anesthesia

1
Desflurane
Isoflurane
Methoxyflurane
Sevoflurane
Succinylcholine

Autoimmune

1
Azathioprine
Etanercept
Mercaptopurine
Siponimod
Thioguanine

2
Cyclosporine
Methotrexate
Tacrolimus

Cancer

1
Capecitabine
Erdafitinib
Fluorouracil
Gefitinib
Irinotecan
Mercaptopurine
Pazopanib
Thioguanine

...Cancer

2
Methotrexate
Tamoxifen

Cardiovascular

1
Acetylsalicylic acid
Atorvastatin
Carvedilol
Clopidogrel
Fluvastatin
Lovastatin
Mavacamten
Metoprolol
Nebivolol
Pitavastatin
Pravastatin
Propranolol
Rosuvastatin
Simvastatin

2
Flecainide
Propafenone
Warfarin

Endocrinology

1
Nateglinide

Gastroenterology

1
Dronabinol

...Gastroenterology

1
Esomeprazole
Metoclopramide
Ondansetron
Rabeprazole

2
Dexlansoprazole
Lansoprazole
Meclizine
Methotrexate
Omeprazole
Pantoprazole
Prochlorperazine
Promethazine

Infection

1
Abacavir
Atazanavir
Dapsone
Nitrofurantoin
Primaquine
Tafenoquine

2
Efavirenz
PEG-interferon alpha

3
Voriconazole

Mental Health

1

Alprazolam
Amoxapine
Amphetamine
Bromazepam
Chlordiazepoxide
Clonazepam
Clorazepate
Diazepam
Fluoxetine
Flurazepam
Lofexidine
Lorazepam
Nicotine replacement therapy
Nitrazepam
Oxazepam
Oxcarbazepine
Protriptyline
Sertraline
Temazepam
Triazolam
Venlafaxine
Viloxazine

2

Amitriptyline
Aripiprazole
Aripiprazole lauroxil
Asenapine
Atomoxetine
Brexpiprazole
Bupropion
Cariprazine
Chlorpromazine
Citalopram

...Mental Health

2

Clomipramine
Clozapine
Desipramine
Doxepin
Escitalopram
Flupentixol
Fluphenazine
Fluvoxamine
Haloperidol
Iloperidone
Imipramine
Lamotrigine
Loxapine
Lurasidone
Methotrimeprazine
Methylphenidate
Molindone
Nortriptyline
Olanzapine
Paliperidone
Paroxetine
Perphenazine
Pimozide
Quetiapine
Risperidone
Thioridazine
Thiothixene
Trifluoperazine
Trimipramine
Vortioxetine
Ziprasidone
Zuclopenthixol

3

Carbamazepine

Neurology

1

Brivaracetam
Clobazam
Clonazepam
Deutetrabenazine
Diazepam
Donepezil
Fosphenytoin
Galantamine
Metoprolol
Oxcarbazepine
Phenytoin
Pitolisant
Propranolol
Tetrabenazine
Valbenazine
Venlafaxine

2

Amitriptyline
Lamotrigine

3

Carbamazepine

Respiratory

1

Salmeterol

Rheumatology

1

Azathioprine
Celecoxib
Etanercept
Flurbiprofen
Ibuprofen
Meloxicam
Pegloticase

...Rheumatology

1

Piroxicam
Rasburicase
Tenoxicam

2

Methotrexate

3

Allopurinol

Urology

1

Darifenacin
Fesoterodine
Mirabegron
Tamsulosin
Tolterodine

2

Sildenafil

Other

1

Abrocitinib
Avatrombopag
Cevimeline
Elagolix
Eltrombopag
Flibanserin
Lusutrombopag
Methylene blue
Oral contraceptives

3

Eliglustat

Overview

This pharmacogenetic information is based on best evidence compiled from guidelines and databases including the FDA Table of Pharmacogenetic Associations and the Clinical Pharmacogenetics Implementation Consortium (CPIC). In some cases, PharmGKB and the Dutch Pharmacogenetics Working Group (DPWG) may also be referenced.

This document includes:
1. Medication Summary: A list of medications organized by their therapeutic area of use and sorted based on their drug-gene interaction severity.
2. Medication Report: Provides information about factors affecting medication response.
3. Guidelines: A table of guidelines used to produce each interpretation.
4. References: Sources of information used to create this report.
5. Laboratory Report: Contains genetic test results in a technical table.

TreatGx and ReviewGx are clinical decision support tools that expand on the contents on this report.

TreatGx

TreatGx is clinical decision support software for precision prescribing that identifies condition-specific medication options based on multiple patient factors.

ReviewGx

ReviewGx uses patient factors including pharmacogenetics to highlight medication safety issues, help optimize medications, and identify deprescribing opportunities.

Components of the Medication Report

For all medications, clinical factors, medical conditions, lab values, drug-gene and drug-drug interactions may contribute to medication response and should be evaluated for each patient. The kidney and liver icon notations are intended for informational purposes only. The patient's kidney/liver function are not used for the purposes of displaying this information, and the potential interactions for that specific medication may not apply. TreatGx and ReviewGx help integrate this information to support precision prescribing and comprehensive medication management. The final genotype/phenotype call is at the discretion of the laboratory director. Medication changes should only be initiated at the discretion of the patient's healthcare provider after a full assessment.

Example:

Generic Name → Codeine

Brand Names → Codeine Contin Tylenol with Codeine No. 2/3/4

Potential Kidney or Liver Interaction → TreatGx ReviewGx

Codeine	Phenotype	Genetic Test	Results	Source/Evidence Level
Codeine Contin Tylenol with Codeine No. 2/3/4	Poor metabolizer	CYP2D6	*3/*6	CPIC A[6]; FDA 1[34]

2 CYP2D6 poor metabolizer: greatly reduced metabolism of Codeine may result in decreased response

3 Avoid Codeine use

Source/Evidence Level for Drug-Gene Interactions:

For each medication, a source is listed for each drug-gene interaction. This report prioritizes guidance from CPIC if the drug-gene pair is assigned a CPIC Level of A or B. This is the threshold that CPIC defines as having sufficient evidence for at least one prescribing action to be recommended. See cpicpgx.org/prioritization for a full explanation of CPIC Levels for Genes/Drugs.

Pharmacogenetic information from FDA-approved drug labels or the FDA Table of Pharmacogenetic Associations (https://www.fda.gov/medical-devices/precision-medicine/table-pharmacogenetic-associations) is included when available.

If there is no CPIC guideline (level A or B) or FDA guidance, other sources may be referenced, such as DPWG guidelines, PharmGKB clinical annotations, and in some instances, clinical studies. See https://www.pharmgkb.org/page/clinAnnLevels for a full explanation of PharmGKB levels of evidence. Use of any of this information is at the discretion of the health professional.

* Other clinical factors, medical conditions and drug-drug interactions may contribute to medication response.

Medication Report

The **Medication Report** provides information on how pharmacogenetic results affect each medication.

Use TreatGx and ReviewGx to explore personalized medication treatment options, dosing information and medication optimization.

Abacavir	Phenotype	Genetic Test	Results	Source/Evidence Level
Ziagen ReviewGx	Reduced risk of adverse drug reactions	HLA-B*57:01	Negative	CPIC A[30]; FDA 1[47]

Drug-Gene Interactions:

1 CPIC – Implication: Low or reduced risk of abacavir hypersensitivity.

1 CPIC – Strong Recommendation: Use abacavir per standard dosing guidelines.

Abrocitinib	Phenotype	Genetic Test	Results	Source/Evidence Level
Cibinqo ReviewGx	Rapid metabolizer	CYP2C19	*1/*17	FDA 1[47]; Product monograph (actionable)[37]

Drug-Gene Interactions:

1 FDA PGx Table: No recommended changes or information for this phenotype in the FDA PGx Table

Acetylsalicylic acid	Phenotype	Genetic Test	Results	Source/Evidence Level
Aspirin Entrophen TreatGx ReviewGx	Decreased, but not absent, risk for non-response	PTGS1 rs10306114	A/A	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 PharmGKB – Clinical Annotation (Level 3 Efficacy): Patients with the PTGS1 rs10306114 A/A genotype who are treated with ASA may have a decreased, but not absent, risk for non-response to ASA as compared to patients with the A/G or G/G genotype. Other genetic and clinical factors may also influence response to ASA.

Alfentanil	Phenotype	Genetic Test	Results	Source/Evidence Level
Alfenta ReviewGx	Increased analgesic response	OPRM1 rs1799971	A/A	PharmGKB 3[48,49]

Drug-Gene Interactions:

2 PharmGKB – Clinical Annotation (Level 3 Efficacy): Patients with the OPRM1 rs1799971 A/A genotype may have an increased analgesic response to alfentanil as compared to patients with the A/G or G/G genotypes. Note that one study reported a non-significant association. This drug-variant pair has been assigned a "no recommendation" by CPIC, as it was determined to be not clinically actionable. Other genetic or clinical factors may also affect a patient's response to alfentanil.
PharmGKB – Clinical Annotation (Level 3 Dosage): Patients with the OPRM1 rs1799971 A/A genotype may have reduced alfentanil dose requirements as compared to patients with the A/G or G/G genotypes. This drug-variant pair has been assigned a "no recommendation" by CPIC, as it was determined to be not clinically actionable. Other genetic or clinical factors may also affect a alfentanil dose requirements.

Allopurinol	Phenotype	Genetic Test	Results	Source/Evidence Level
Aloprim Lopurin Zyloprim TreatGx ReviewGx	Increased risk of adverse drug reactions	HLA-B*58:01	Positive	CPIC A[42]; FDA 2[47]

Drug-Gene Interactions:

3 CPIC – Implication: Significantly increased risk of allopurinol-induced severe cutaneous adverse reaction (SCAR).

3 CPIC – Strong Recommendation: Allopurinol is contraindicated.

Alprazolam	Phenotype	Genetic Test	Results	Source/Evidence Level
Xanax	Normal metabolizer	CYP2C9	*1/*1	Clinical studies[21]

ReviewGx

Drug-Gene Interactions:

1 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*1 homozygotes using benzodiazepines had no statistically significant increased fall risk compared to those not taking benzodiazepines (HR = 1.14; 95% CI: 0.90-1.45), whereas those carrying a CYP2C9*2 or *3 allele had a 45% increased fall risk (HR = 1.45; 95% CI: 1.21-1.73). Because the exact role of CYP2C9 in benzodiazepine metabolism is still unclear, additional research is warranted. Additionally, due to sample size, the authors were not able to perform sub-analyses of different benzodiazepine classes or investigate individual benzodiazepines in order to assess a potential class or individual drug effect.

Amitriptyline	Phenotype	Genetic Test	Results	Source/Evidence Level
Elavil Levate	Intermediate metabolizer	CYP2D6	*2.019/*4.001	CPIC A[23]; FDA 3[47]
	Rapid metabolizer	CYP2C19	*1/*17	CPIC A[23]

TreatGx
ReviewGx

Drug-Gene Interactions:

2 CPIC – CYP2D6 Implication: Reduced metabolism of TCAs to less active compounds compared to normal metabolizers. Higher plasma concentrations of active drug will increase the probability of side effects.

2 CPIC – CYP2C19 Implication: Increased metabolism of tertiary amines compared to normal metabolizers. Greater conversion of tertiary amines to secondary amines may affect response or side effects.

2 CPIC – Optional Recommendation: Consider alternative drug not metabolized by CYP2C19. If use is warranted, utilize therapeutic drug monitoring to guide dose adjustment (strongly recommended for a patient with CYP2D6 ultrarapid, intermediate, or poor metabolism in combination with CYP2C19 ultrarapid, intermediate, or poor metabolism). TCAs without major CYP2C19 metabolism include the secondary amines nortriptyline and desipramine.
Recommendations above only apply to higher initial doses of TCAs for treatment of conditions such as depression. Lower dosages are often used for neuropathic pain compared to depressive disorders.
Because of the lower dosage, it is less likely that CYP2D6 intermediate metabolizers will experience adverse effects due to supratherapeutic plasma concentrations. Therefore, CPIC recommends no dose modifications for intermediate metabolizers when prescribed a lower dose for treatment of neuropathic pain, but these patients should be monitored closely for side effects. If larger doses are warranted, CPIC recommends following the gene-based guidelines presented above.
There are limited data to support dose recommendations for CYP2C19*17 carriers who are prescribed TCAs at lower doses for neuropathic pain treatment.

Amoxapine	Phenotype	Genetic Test	Results	Source/Evidence Level
	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 3[47]

ReviewGx

Drug-Gene Interactions:

1 FDA PGx Table Section 3 – Potential Impact on Pharmacokinetic Properties Only: May alter systemic concentrations.

Amphetamine	Phenotype	Genetic Test	Results	Source/Evidence Level
Adderall Adzenys Dyanavel Evekeo Mydayis	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]

TreatGx
ReviewGx

Drug-Gene Interactions:

1 CYP2D6 alleles do not indicate changes from recommended dose

Aripiprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Abilify TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]; Product monograph (actionable)[43]
	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain compared to A/A	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1. FDA PGx Table: No recommended changes or information for this CYP2D6 phenotype in the FDA PGx Table
1. 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.
1. PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have a decreased likelihood of experiencing weight gain when treated with aripiprazole as compared to patients with the A/A genotype. Other genetic and clinical factors may also influence the likelihood of experiencing weight gain when treated with aripiprazole.
2. 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Aripiprazole lauroxil	Phenotype	Genetic Test	Results	Source/Evidence Level
Aristada TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]; Product monograph (actionable)[3]
	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain compared to A/A	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1. FDA PGx Table: No recommended changes or information for this CYP2D6 phenotype in the FDA PGx Table
1. 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.
1. PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have a decreased likelihood of experiencing weight gain when treated with aripiprazole as compared to patients with the A/A genotype. Other genetic and clinical factors may also influence the likelihood of experiencing weight gain when treated with aripiprazole. *PharmGKB clinical annotation is based on aripiprazole; aripiprazole lauroxil is the prodrug, effects may vary.
2. 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Asenapine	Phenotype	Genetic Test	Results	Source/Evidence Level
Saphris TreatG ReviewG	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Atazanavir	Phenotype	Genetic Test	Results	Source/Evidence Level
Reyataz ReviewG	Normal/Extensive metabolizer	UGT1A1	*1/*1	CPIC A[17]

Drug-Gene Interactions:

1 CPIC – Implication: Reference UGT1A1 activity; very low likelihood of bilirubin-related discontinuation of atazanavir.

1 CPIC – Strong Recommendation: There is no need to avoid prescribing of atazanavir based on UGT1A1 genetic test result. Inform the patient that some patients stop atazanavir because of jaundice (yellow eyes and skin), but that this patient's genotype makes this unlikely (less than about a 1 in 20 chance of stopping atazanavir because of jaundice).
All studies correlating UGT1A1 genotypes with atazanavir adverse events have involved ritonavir boosting. However, concentration-time profiles are equivalent when boosted with either cobicistat or ritonavir (PMID 23532097), and bilirubin-related adverse events including discontinuation of atazanavir occur in a similar percentage of patients prescribed atazanavir with cobicistat or ritonavir (PMID 23532097). Associations between UGT1A1 genotype, bilirubin elevations, and atazanavir/ritonavir discontinuation therefore almost certainly translate to atazanavir/cobicistat.

Atomoxetine	Phenotype	Genetic Test	Results	Source/Evidence Level
Strattera	Intermediate metabolizer (AS 1.0)	CYP2D6 (Activity Score)	*2.019/*4.001	CPIC A[10]; FDA 1[47]

TreatGx
ReviewGx

Drug-Gene Interactions:

2 CPIC – Implication: Possibly higher atomoxetine concentrations as compared to normal metabolizers but questionable clinical significance. Intermediate metabolizers with an activity score of 1 may be at an increased risk of discontinuation as compared to poor metabolizers.

2 ADULTS: CPIC – Moderate Recommendation: Initiate with a dose of 40 mg/day and increase to 80 mg/day after 3 days. If no clinical response and in the absence of adverse events after 2 weeks, consider increasing dose to 100 mg/day. If no clinical response observed after 2 weeks, consider obtaining a peak plasma concentration (1 to 2 hours after dose administered). If <200 ng/mL, consider a proportional increase in dose to approach 400 ng/mL. Dosages greater than 100 mg/day may be needed to achieve target concentrations.
PEDIATRICS: CPIC – Moderate Recommendation: Initiate with a dose of 0.5 mg/kg/day and increase to 1.2 mg/kg/day after 3 days. If no clinical response and in the absence of adverse events after 2 weeks, consider obtaining a peak plasma concentration (1 to 2 hours after dose administered). If < 200 ng/mL, consider a proportional increase in dose to approach 400 ng/mL.
Notes (Adult & Pediatrics): Therapeutic range of 200 to 1000 ng/mL has been proposed (PMID 29493375). Limited data are available regarding the relationship between atomoxetine plasma concentrations and clinical response. Available information suggests that clinical response is greater in poor metabolizers (PMs) compared to non-PMs and may be related to the higher plasma concentrations 1 to 1.5 hours after dosing in PMs compared to non-PMs administered a similar dose. Furthermore, modest improvement in response, defined as reduction in ADHD-rating scale, is observed at peak concentrations greater than 400 ng/mL. Doses above 120 mg/day have not been evaluated.

Atorvastatin	Phenotype	Genetic Test	Results	Source/Evidence Level
Lipitor	Normal function	SLCO1B1	*1/*1	CPIC A[12]; FDA 3[47]

TreatGx
ReviewGx

Drug-Gene Interactions:

1 CPIC – Implication: Typical myopathy risk and Atorvastatin exposure.

1 CPIC – Strong Recommendation: Prescribe desired starting dose and adjust doses based on disease-specific guidelines.
The potential for drug-drug interactions and dose limits based on renal and hepatic function and ancestry should be evaluated prior to initiating a statin.

Avatrombopag	Phenotype	Genetic Test	Results	Source/Evidence Level
Doptelet	Normal metabolizer	CYP2C9	*1/*1	FDA 3[47]
ReviewGx	Normal Factor II	Factor II rs1799963	G/G	Product monograph (actionable)[1]
	Normal Factor V Leiden	Factor V rs6025	C/C	Product monograph (actionable)[1]

Drug-Gene Interactions:

1 FDA PGx Table: No recommended changes or information for this CYP2C9 phenotype in the FDA PGx Table

1 FDA Product Monograph: No recommended changes or information for normal Factor II (i.e. Prothrombin 20210A mutation absent) in the FDA Product Monograph.

1 FDA Product Monograph: No recommended changes or information for normal Factor V in the FDA Product Monograph.

Azathioprine	Phenotype	Genetic Test	Results	Source/Evidence Level
Azasan Imuran	Normal metabolizer	TPMT	*1/*1	CPIC A[40,45]; FDA 1[47]
	Normal metabolizer	NUDT15	*1/*1	CPIC A[40,45]; FDA 1[47]

TreatG
ReviewG

Drug-Gene Interactions:

1 CPIC – TPMT Implication: Lower concentrations of thioguanine nucleotides metabolites, higher metabolites of thiopurine methyltransferase, this is the 'normal' pattern. Normal risk of thiopurine-related leukopenia, neutropenia, myelosuppression.

1 CPIC – NUDT15 Implication: Normal risk of thiopurine-related leukopenia, neutropenia, myelosuppression.

1 CPIC – Strong Recommendation: Start with normal starting dose (e.g., 2-3 mg/kg/day) and adjust doses of azathioprine based on disease-specific guidelines. Allow 2 weeks to reach steady-state after each dose adjustment.
Normal starting doses vary by race/ethnicity and treatment regimens.

Brexpiprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Rexulti	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]; Product monograph (actionable)[4]
	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

TreatG
ReviewG

Drug-Gene Interactions:

1 FDA PGx Table: No recommended changes or information for this CYP2D6 phenotype in the FDA PGx Table

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Brivaracetam	Phenotype	Genetic Test	Results	Source/Evidence Level
Briviact Brivlera	Rapid metabolizer	CYP2C19	*1/*17	FDA 1[47]

ReviewG

Drug-Gene Interactions:

1 CYP2C19 alleles do not indicate changes from recommended dose

Bromazepam	Phenotype	Genetic Test	Results	Source/Evidence Level
ReviewGx	Normal metabolizer	CYP2C9	*1/*1	Clinical studies[21]

Drug-Gene Interactions:

1 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*1 homozygotes using benzodiazepines had no statistically significant increased fall risk compared to those not taking benzodiazepines (HR = 1.14; 95% CI: 0.90-1.45), whereas those carrying a CYP2C9*2 or *3 allele had a 45% increased fall risk (HR = 1.45; 95% CI: 1.21-1.73). Because the exact role of CYP2C9 in benzodiazepine metabolism is still unclear, additional research is warranted. Additionally, due to sample size, the authors were not able to perform sub-analyses of different benzodiazepine classes or investigate individual benzodiazepines in order to assess a potential class or individual drug effect.

Bupropion	Phenotype	Genetic Test	Results	Source/Evidence Level
Wellbutrin Zyban	Less likely to quit smoking compared to G/G	ANKK1/DRD2 rs1800497	A/G	PharmGKB 3[48,49]

TreatGx ReviewGx

Drug-Gene Interactions:

2 PharmGKB – Clinical Annotation (Level 3 Efficacy): Patients with the ANKK1 rs1800497 A/G genotype who are treated with bupropion may be less likely to quit smoking as compared to patients with the G/G genotype, however contradictory findings about abstinence exist. Other genetic and clinical factors may also influence a patient's chance for quitting smoking.

Capecitabine	Phenotype	Genetic Test	Results	Source/Evidence Level
Xeloda	Normal metabolizer	DPYD	*1/*1	CPIC A[5]; FDA 1[47]

ReviewGx

Drug-Gene Interactions:

1 DPYD alleles indicate normal DPD activity and typical risk for Capecitabine toxicity

1 DPYD alleles do not indicate changes from recommended dose

Carbamazepine	Phenotype	Genetic Test	Results	Source/Evidence Level
Carbatrol Epitol Equetro Tegretol Teril	Increased risk of adverse drug reactions	HLA-A*31:01	Positive	CPIC A[39]; FDA 2[47]
	Reduced risk of adverse drug reactions	HLA-B*15:02	Negative	CPIC A[39]; FDA 1[47]

TreatGx ReviewGx

Drug-Gene Interactions:

3 CPIC – HLA-A*31:01 Implication: Greater risk of carbamazepine-induced Stevens-Johnson syndrome and toxic epidermal necrolysis (SJS/TEN), drug reaction with eosinophilia and systemic symptoms (DRESS), and maculopapular exanthema (MPE).

3 CPIC – HLA-A*31:01 Strong Recommendation: If patient is carbamazepine-naïve and alternative agents are available, do not use carbamazepine.
CPIC – Optional Recommendation: If patient is carbamazepine-naïve and alternative agents are not available, consider the use of carbamazepine with increased frequency of clinical monitoring. Discontinue therapy at first evidence of a cutaneous adverse reaction.
CPIC – Optional Recommendation: The latency period for cutaneous adverse drug reactions is variable depending on phenotype; however, all usually occur within three months of regular dosing. Therefore, if the patient has previously used carbamazepine consistently for longer than three months without incidence of cutaneous adverse reactions, cautiously consider use of carbamazepine.
Other aromatic anticonvulsants have very limited evidence, if any, linking SJS/ TEN, DRESS, and/or MPE with the HLA-A*31:01 allele, and thus no recommendation can be made with respect to choosing another aromatic anticonvulsant as an alternative agent. Previous tolerance of carbamazepine is not indicative of tolerance to other aromatic anticonvulsants. Aromatic anticonvulsants include carbamazepine, oxcarbazepine, eslicarbazepine, lamotrigine, phenytoin, fosphenytoin, and phenobarbital.

Cariprazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Vraylar	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
TreatGx ReviewGx	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Carisoprodol	Phenotype	Genetic Test	Results	Source/Evidence Level
ReviewGx	Rapid metabolizer	CYP2C19	*1/*17	FDA 3[47]

Drug-Gene Interactions:

1 CYP2C19 alleles do not indicate changes from recommended dose

Carvedilol	Phenotype	Genetic Test	Results	Source/Evidence Level
Coreg	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 2[47]
TreatGx ReviewGx				

Drug-Gene Interactions:

1 CYP2D6 alleles do not indicate changes from recommended dose

Celecoxib	Phenotype	Genetic Test	Results	Source/Evidence Level
Celebrex	Normal metabolizer	CYP2C9 (Star Alleles)	*1/*1	CPIC A[46]; FDA 1[47]
TreatGx ReviewGx				

Drug-Gene Interactions:

1 CYP2C9 alleles do not indicate changes from recommended dose

Cevimeline	Phenotype	Genetic Test	Results	Source/Evidence Level
Evoxac ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 2[47]

Drug-Gene Interactions:

1 CYP2D6 alleles do not indicate changes from recommended dose

Chlordiazepoxide	Phenotype	Genetic Test	Results	Source/Evidence Level
Librium ReviewGx	Normal metabolizer	CYP2C9	*1/*1	Clinical studies[21]

Drug-Gene Interactions:

1 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*1 homozygotes using benzodiazepines had no statistically significant increased fall risk compared to those not taking benzodiazepines (HR = 1.14; 95% CI: 0.90-1.45), whereas those carrying a CYP2C9*2 or *3 allele had a 45% increased fall risk (HR = 1.45; 95% CI: 1.21-1.73). Because the exact role of CYP2C9 in benzodiazepine metabolism is still unclear, additional research is warranted. Additionally, due to sample size, the authors were not able to perform sub-analyses of different benzodiazepine classes or investigate individual benzodiazepines in order to assess a potential class or individual drug effect.

Chlorpromazine	Phenotype	Genetic Test	Results	Source/Evidence Level
TreatGx ReviewGx	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Citalopram	Phenotype	Genetic Test	Results	Source/Evidence Level
Celexa TreatGx ReviewGx	Rapid metabolizer	CYP2C19	*1/*17	CPIC A[9]; FDA 1[47]

Drug-Gene Interactions:

2 Increase in metabolism of citalopram and escitalopram to less active compounds when compared with CYP2C19 normal metabolizers. Lower plasma concentrations decrease the probability of clinical benefit.

2 Initiate therapy with recommended starting dose. If patient does not adequately respond to recommended maintenance dosing, consider titrating to a higher maintenance dose or switching to a clinically appropriate alternative antidepressant not predominantly metabolized by CYP2C19 (per CPIC optional recommendation).

Clobazam	Phenotype	Genetic Test	Results	Source/Evidence Level
Onfi Sympazan	Rapid metabolizer	CYP2C19	*1/*17	FDA 1[47]; Product monograph (actionable)[29]
ReviewGx	Normal metabolizer	CYP2C9	*1/*1	Clinical Studies[21]

Drug-Gene Interactions:

1 FDA PGx Table: No recommended changes or information for this CYP2C19 phenotype in the FDA PGx Table

1 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*1 homozygotes using benzodiazepines had no statistically significant increased fall risk compared to those not taking benzodiazepines (HR = 1.14; 95% CI: 0.90-1.45), whereas those carrying a CYP2C9*2 or *3 allele had a 45% increased fall risk (HR = 1.45; 95% CI: 1.21-1.73). Because the exact role of CYP2C9 in benzodiazepine metabolism is still unclear, additional research is warranted. Additionally, due to sample size, the authors were not able to perform sub-analyses of different benzodiazepine classes or investigate individual benzodiazepines in order to assess a potential class or individual drug effect.

Clomipramine	Phenotype	Genetic Test	Results	Source/Evidence Level
Anafranil	Intermediate metabolizer	CYP2D6	*2.019/*4.001	CPIC B[23]; FDA 3[47]
ReviewGx	Rapid metabolizer	CYP2C19	*1/*17	CPIC B[23]

Drug-Gene Interactions:

2 CPIC – CYP2D6 Implication: Reduced metabolism of TCAs to less active compounds compared to normal metabolizers. Higher plasma concentrations of active drug will increase the probability of side effects.

2 CPIC – CYP2C19 Implication: Increased metabolism of tertiary amines compared to normal metabolizers. Greater conversion of tertiary amines to secondary amines may affect response or side effects.

2 CPIC – Optional Recommendation: Consider alternative drug not metabolized by CYP2C19. If use is warranted, utilize therapeutic drug monitoring to guide dose adjustment (strongly recommended for a patient with CYP2D6 ultrarapid, intermediate, or poor metabolism in combination with CYP2C19 ultrarapid, intermediate, or poor metabolism). TCAs without major CYP2C19 metabolism include the secondary amines nortriptyline and desipramine.
Recommendations above only apply to higher initial doses of TCAs for treatment of conditions such as depression. Lower dosages are often used for neuropathic pain compared to depressive disorders.
Because of the lower dosage, it is less likely that CYP2D6 intermediate metabolizers will experience adverse effects due to supratherapeutic plasma concentrations. Therefore, CPIC recommends no dose modifications for intermediate metabolizers when prescribed a lower dose for treatment of neuropathic pain, but these patients should be monitored closely for side effects. If larger doses are warranted, CPIC recommends following the gene-based guidelines presented above.
There are limited data to support dose recommendations for CYP2C19*17 carriers who are prescribed TCAs at lower doses for neuropathic pain treatment.

Clonazepam	Phenotype	Genetic Test	Results	Source/Evidence Level
Klonopin Rivotril	Normal metabolizer	CYP2C9	*1/*1	Clinical studies[21]

TreatGx
ReviewGx

Drug-Gene Interactions:

1 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*1 homozygotes using benzodiazepines had no statistically significant increased fall risk compared to those not taking benzodiazepines (HR = 1.14; 95% CI: 0.90-1.45), whereas those carrying a CYP2C9*2 or *3 allele had a 45% increased fall risk (HR = 1.45; 95% CI: 1.21-1.73). Because the exact role of CYP2C9 in benzodiazepine metabolism is still unclear, additional research is warranted. Additionally, due to sample size, the authors were not able to perform sub-analyses of different benzodiazepine classes or investigate individual benzodiazepines in order to assess a potential class or individual drug effect.

Clopidogrel	Phenotype	Genetic Test	Results	Source/Evidence Level
Plavix TreatGx ReviewGx	Rapid metabolizer	CYP2C19	*1/*17	CPIC A[27]; FDA 1[47]

Drug-Gene Interactions:

1 CPIC – Implication: Normal or increased clopidogrel active metabolite formation; normal or lower on-treatment platelet reactivity; no association with higher bleeding risk.

1 CPIC – Strong Recommendation for acute coronary syndrome (ACS) and/or percutaneous coronary intervention (PCI): If considering clopidogrel, use at standard dose (75 mg/day).
No recommendation for non-ACS, non-PCI cardiovascular indications. ACS and/or PCI includes patients undergoing PCI for an ACS or non-ACS (elective) indication. Non-ACS, non-PCI cardiovascular indications include peripheral arterial disease and stable coronary artery disease following a recent myocardial infarction outside the setting of PCI.

Clorazepate	Phenotype	Genetic Test	Results	Source/Evidence Level
Gen-Xene Tranxene ReviewGx	Normal metabolizer	CYP2C9	*1/*1	Clinical studies[21]

Drug-Gene Interactions:

1 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*1 homozygotes using benzodiazepines had no statistically significant increased fall risk compared to those not taking benzodiazepines (HR = 1.14; 95% CI: 0.90-1.45), whereas those carrying a CYP2C9*2 or *3 allele had a 45% increased fall risk (HR = 1.45; 95% CI: 1.21-1.73). Because the exact role of CYP2C9 in benzodiazepine metabolism is still unclear, additional research is warranted. Additionally, due to sample size, the authors were not able to perform sub-analyses of different benzodiazepine classes or investigate individual benzodiazepines in order to assess a potential class or individual drug effect.

Clozapine	Phenotype	Genetic Test	Results	Source/Evidence Level
Clozaril Fazaclo ODT Versacloz TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]
	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased risk of developing metabolic syndrome	HTR2C rs1414334	G/G	PharmGKB 3[48,49]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 FDA PGx Table: No recommended changes or information for this CYP2D6 phenotype in the FDA PGx Table

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with two X-chromosomes and the HTR2C rs1414334 G/G genotype, or one X-chromosome and the G genotype who are treated with Clozapine may have a decreased risk of developing metabolic syndrome as compared to patients with the C/C, C/G, or C genotype. Other genetic and clinical factors may also influence a patient's risk for developing metabolic syndrome.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Codeine	Phenotype	Genetic Test	Results	Source/Evidence Level
Codeine Contin Tylenol with Codeine No. 2/3/4 TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	CPIC A[13]; FDA 1[47]; FDA 2[47]

Drug-Gene Interactions:

1 CPIC – Implication: Reduced morphine formation.

1 CPIC – Moderate Recommendation: Use codeine label recommended age-specific or weight-specific dosing. If no response and opioid use is warranted, consider a non-tramadol opioid.

Cyclosporine	Phenotype	Genetic Test	Results	Source/Evidence Level
Neoral Sandimmune ReviewGx	Poor metabolizer	CYP3A5	*3/*3	PharmGKB 3[48,49]

Drug-Gene Interactions:

2 PharmGKB – Clinical Annotation (Level 3 Dosage): Patients who are recipients of a kidney transplant and who carry the *3 allele in combination with another no function allele may have decreased cyclosporine dose requirements as compared to patients carrying two normal function alleles or a normal function allele in combination with a no function allele. However, conflicting evidence has been reported. Other genetic and clinical factors may also affect cyclosporine dose requirements. (PharmGKB does not provide information about other poor metabolizer diplotypes without *3 i.e. *6/*6, *7/*7, *6/*7).

Dapsone	Phenotype	Genetic Test	Results	Source/Evidence Level
Aczone ReviewGx	Normal phenotype	G6PD	B (reference)/B (reference)	CPIC A[18]

Drug-Gene Interactions:

1 CPIC – Implication: Low risk of acute hemolytic anemia.

1 CPIC – Strong Recommendation: No reason to avoid based on G6PD status.

Darifenacin	Phenotype	Genetic Test	Results	Source/Evidence Level
Enablex TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 3[47]

Drug-Gene Interactions:

1 CYP2D6 alleles do not indicate changes from recommended dose

Desflurane	Phenotype	Genetic Test	Results	Source/Evidence Level
ReviewGx	Negative	CACNA1S rs1800559	C/C	CPIC A[20]
	Negative	CACNA1S rs772226819	G/G	CPIC A[20]
	Negative	RYR1 rs111888148	G/G	CPIC A[20]
	Negative	RYR1 rs112563513	G/G	CPIC A[20]
	Negative	RYR1 rs118192122	G/G	CPIC A[20]
	Negative	RYR1 rs118192124	C/C	CPIC A[20]
	Negative	RYR1 rs118192161	C/C	CPIC A[20]
	Negative	RYR1 rs118192167	A/A	CPIC A[20]
	Negative	RYR1 rs118192168	G/G	CPIC A[20]
	Negative	RYR1 rs118192170	T/T	CPIC A[20]
	Negative	RYR1 rs118192172	C/C	CPIC A[20]
	Negative	RYR1 rs118192175	C/C	CPIC A[20]
	Negative	RYR1 rs118192176	G/G	CPIC A[20]
	Negative	RYR1 rs121918593	G/G	CPIC A[20]
	Negative	RYR1 rs121918594	G/G	CPIC A[20]
	Negative	RYR1 rs121918595	C/C	CPIC A[20]
	Negative	RYR1 rs144336148	G/G	CPIC A[20]
	Negative	RYR1 rs193922747	T/T	CPIC A[20]
	Negative	RYR1 rs193922748	C/C	CPIC A[20]
	Negative	RYR1 rs193922753	G/G	CPIC A[20]
	Negative	RYR1 rs193922770	C/C	CPIC A[20]
	Negative	RYR1 rs193922802	G/G	CPIC A[20]
	Negative	RYR1 rs193922803	C/C	CPIC A[20]
	Negative	RYR1 rs193922807	G/G	CPIC A[20]
	Negative	RYR1 rs193922809	G/G	CPIC A[20]
	Negative	RYR1 rs193922816	C/C	CPIC A[20]
	Negative	RYR1 rs193922818	G/G	CPIC A[20]
	Negative	RYR1 rs193922832	G/G	CPIC A[20]
	Negative	RYR1 rs193922843	G/G	CPIC A[20]
	Negative	RYR1 rs193922876	C/C	CPIC A[20]
	Negative	RYR1 rs193922878	C/C	CPIC A[20]
	Negative	RYR1 rs28933396	G/G	CPIC A[20]
	Negative	RYR1 rs28933397	C/C	CPIC A[20]
	Negative	RYR1 rs63749869	G/G	CPIC A[20]

Drug-Gene Interactions:

1 Uncertain risk of developing malignant hyperthermia (MH)

1 Results do not eliminate malignant hyperthermia susceptibility. Clinical findings, family history, further genetic testing, and other laboratory data should guide use of Desflurane.

Desipramine	Phenotype	Genetic Test	Results	Source/Evidence Level
Norpramin TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	CPIC B[23]; FDA 3[47]

Drug-Gene Interactions:

2 CPIC – CYP2D6 Implication: Reduced metabolism of TCAs to less active compounds compared to normal metabolizers. Higher plasma concentrations of active drug will increase the probability of side effects.

2 CPIC – Optional Recommendation: Consider a 25% reduction of recommended starting dose. Patients may receive an initial low dose of a tricyclic, which is then increased over several days to the recommended steady-state dose. The starting dose in this guideline refers to the recommended steady-state dose. Utilize therapeutic drug monitoring to guide dose adjustments. Titrate dose to observed clinical response with symptom improvement and minimal (if any) side effects.
Recommendations above only apply to higher initial doses of TCAs for treatment of conditions such as depression. Lower dosages are often used for neuropathic pain compared to depressive disorders.
Because of the lower dosage, it is less likely that CYP2D6 intermediate metabolizers will experience adverse effects due to supratherapeutic plasma concentrations. Therefore, CPIC recommends no dose modifications for intermediate metabolizers when prescribed a lower dose for treatment of neuropathic pain, but these patients should be monitored closely for side effects. If larger doses are warranted, CPIC recommends following the gene-based guidelines presented above.

Deutetrabenazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Austedo ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]

Drug-Gene Interactions:

1 CYP2D6 alleles do not indicate changes from recommended dose

Dexlansoprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Dexilant TreatGx ReviewGx	Rapid metabolizer	CYP2C19	*1/*17	CPIC B[28]; FDA 3[47]

Drug-Gene Interactions:

2 CPIC – Implication: Decreased plasma concentrations of PPIs compared with CYP2C19 NMs; increased risk of therapeutic failure.

2 CPIC – Moderate Recommendation: Initiate standard starting daily dose. Consider increasing dose by 50–100% for the treatment of Helicobacter pylori infection and erosive esophagitis. Daily dose may be given in divided doses. Monitor for efficacy.

Diazepam	Phenotype	Genetic Test	Results	Source/Evidence Level
Diastat Valium TreatGx ReviewGx	Rapid metabolizer	CYP2C19	*1/*17	FDA 3[47]
	Normal metabolizer	CYP2C9	*1/*1	Clinical Studies[21]

Drug-Gene Interactions:

1 FDA PGx Table: No recommended changes or information for this CYP2C19 phenotype in the FDA PGx Table

1 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*1 homozygotes using benzodiazepines had no statistically significant increased fall risk compared to those not taking benzodiazepines (HR = 1.14; 95% CI: 0.90-1.45), whereas those carrying a CYP2C9*2 or *3 allele had a 45% increased fall risk (HR = 1.45; 95% CI: 1.21-1.73). Because the exact role of CYP2C9 in benzodiazepine metabolism is still unclear, additional research is warranted. Additionally, due to sample size, the authors were not able to perform sub-analyses of different benzodiazepine classes or investigate individual benzodiazepines in order to assess a potential class or individual drug effect.

Donepezil	Phenotype	Genetic Test	Results	Source/Evidence Level
Aricept TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 3[47]

Drug-Gene Interactions:

1 CYP2D6 alleles do not indicate changes from recommended dose

Doxepin	Phenotype	Genetic Test	Results	Source/Evidence Level
Silenor Sinequan	Intermediate metabolizer	CYP2D6	*2.019/*4.001	CPIC B[23]; FDA 3[47]
	Rapid metabolizer	CYP2C19	*1/*17	CPIC B[23]; FDA 3[47]

TreatGx
ReviewGx

Drug-Gene Interactions:

2 CPIC – CYP2D6 Implication: Reduced metabolism of TCAs to less active compounds compared to normal metabolizers. Higher plasma concentrations of active drug will increase the probability of side effects.

2 CPIC – CYP2C19 Implication: Increased metabolism of tertiary amines compared to normal metabolizers. Greater conversion of tertiary amines to secondary amines may affect response or side effects.

2 CPIC – Optional Recommendation: Consider alternative drug not metabolized by CYP2C19. If use is warranted, utilize therapeutic drug monitoring to guide dose adjustment (strongly recommended for a patient with CYP2D6 ultrarapid, intermediate, or poor metabolism in combination with CYP2C19 ultrarapid, intermediate, or poor metabolism). TCAs without major CYP2C19 metabolism include the secondary amines nortriptyline and desipramine.
Recommendations above only apply to higher initial doses of TCAs for treatment of conditions such as depression. Lower dosages are often used for neuropathic pain compared to depressive disorders.
Because of the lower dosage, it is less likely that CYP2D6 intermediate metabolizers will experience adverse effects due to supratherapeutic plasma concentrations. Therefore, CPIC recommends no dose modifications for intermediate metabolizers when prescribed a lower dose for treatment of neuropathic pain, but these patients should be monitored closely for side effects. If larger doses are warranted, CPIC recommends following the gene-based guidelines presented above.
There are limited data to support dose recommendations for CYP2C19*17 carriers who are prescribed TCAs at lower doses for neuropathic pain treatment.

Dronabinol	Phenotype	Genetic Test	Results	Source/Evidence Level
Marinol Syndros	Normal metabolizer	CYP2C9	*1/*1	FDA 1[47]

ReviewGx

Drug-Gene Interactions:

1 CYP2C9 alleles do not indicate changes from recommended dose

Efavirenz	Phenotype	Genetic Test	Results	Source/Evidence Level
Sustiva	Intermediate metabolizer	CYP2B6	*1/*6	CPIC A[14]; FDA 2[47]

ReviewGx

Drug-Gene Interactions:

2 CPIC – Implication: Higher dose-adjusted trough concentrations of efavirenz compared with normal metabolizers; increased risk of CNS adverse events.

2 CPIC – Moderate Recommendation: Consider initiating efavirenz with decreased dose of 400 mg/day (in children ≥40 kg and adult patients).
If therapeutic drug monitoring is available and a decreased efavirenz dose is prescribed, consider obtaining steady-state plasma efavirenz concentrations to ensure concentrations are in the suggested therapeutic range (~1 to 4 µg/mL). To prescribe efavirenz at a decreased dose of 400 mg/day or 200 mg/day in a multidrug regimen may require prescribing more than one pill once daily. If so, the provider should weigh the potential benefit of reduced dose against the potential detrimental impact of increased pill number.

Elagolix	Phenotype	Genetic Test	Results	Source/Evidence Level
Orilissa	Normal function	SLCO1B1	*1/*1	FDA 3[47]

ReviewGx

Drug-Gene Interactions:

1 SLCO1B1 alleles indicate a typical response to Elagolix

Eliglustat	Phenotype	Genetic Test	Results	Source/Evidence Level
Cerdelga	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]

ReviewGx

Drug-Gene Interactions:

- 2 CYP2D6 intermediate metabolizer: reduced metabolism of Eliglustat to less active compounds
- 2 Higher plasma concentrations of active drug may increase the risk of adverse drug reactions
- 3 Concurrent use of a strong or moderate CYP3A inhibitor, use of both a moderate or strong CYP2D6 inhibitor and a moderate or strong CYP3A inhibitor, or use of a strong CYP3A inducer: Avoid Eliglustat use
- 2 Concurrent use of a moderate or strong CYP2D6 inhibitor: Consider reducing eliglustat dose, refer to drug monograph or FDA labelling for dosing recommendations
- 1 No concurrent use of interacting drugs: CYP2D6 alleles do not indicate changes from recommended dose, refer to drug monograph or FDA labelling for dosing recommendations

Eltrombopag	Phenotype	Genetic Test	Results	Source/Evidence Level
Promacta Revolade	Normal Factor V Leiden	Factor V rs6025	C/C	Product monograph (actionable)[36]

ReviewGx

Drug-Gene Interactions:

- 1 FDA Product Monograph: No recommended changes or information for normal Factor V in the FDA Product Monograph.

Erdafitinib	Phenotype	Genetic Test	Results	Source/Evidence Level
Balversa	Normal metabolizer	CYP2C9 (Star Alleles)	*1/*1	FDA 1[47]

ReviewGx

Drug-Gene Interactions:

- 1 FDA PGx Table: No recommended changes or information for this CYP2C9 star allele result in the FDA PGx Table

Escitalopram	Phenotype	Genetic Test	Results	Source/Evidence Level
Cipralex Lexapro	Rapid metabolizer	CYP2C19	*1/*17	CPIC A[9]; FDA 3[47]

TreatGx ReviewGx

Drug-Gene Interactions:

- 2 Increase in metabolism of citalopram and escitalopram to less active compounds when compared with CYP2C19 normal metabolizers. Lower plasma concentrations decrease the probability of clinical benefit.
- 2 Initiate therapy with recommended starting dose. If patient does not adequately respond to recommended maintenance dosing, consider titrating to a higher maintenance dose or switching to a clinically appropriate alternative antidepressant not predominantly metabolized by CYP2C19 (per CPIC optional recommendation).

Esomeprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Nexium	Rapid metabolizer	CYP2C19	*1/*17	FDA 3[47]

TreatGx ReviewGx

Drug-Gene Interactions:

- 1 FDA PGx Table: No recommended changes or information for this phenotype in the FDA PGx Table

Etanercept	Phenotype	Genetic Test	Results	Source/Evidence Level
Enbrel Brenzys	Increased response	TNF-alpha rs1800629	G/G	PharmGKB 2B[48,49]

TreatGx ReviewGx

Drug-Gene Interactions:

- 1 PharmGKB – Clinical Annotation (Level 2B Efficacy): Patients with the TNF rs1800629 G/G genotype may have increased response to etanercept as compared to patients with the A/A or A/G genotype (for psoriatic arthritis, rheumatoid arthritis, Crohn's disease, inflammation, psoriasis, and ankylosing spondylitis). Other genetic and clinical factors may also influence response to etanercept.

Fentanyl	Phenotype	Genetic Test	Results	Source/Evidence Level
Actiq Duragesic Fentora Sublimaze ReviewGx	Decreased analgesic response	OPRM1 rs1799971	A/A	PharmGKB 3[48,49]

Drug-Gene Interactions:

2 PharmGKB – Clinical Annotation (Level 3 Efficacy): Patients with the OPRM1 rs1799971 A/A genotype may have a decreased analgesic response to fentanyl as compared to patients with the A/G or G/G genotypes. However, conflicting evidence has been reported. This drug-variant pair has been assigned a "no recommendation" by CPIC, as it was determined to be not clinically actionable. Other genetic or clinical factors may also affect response to fentanyl.

PharmGKB – Clinical Annotation (Level 3 Dosage): Patients with the OPRM1 rs1799971 A/A genotype may have decreased fentanyl dose requirements as compared to patients with the G/G genotype. However, conflicting evidence has been reported. This drug-variant pair has been assigned a "no recommendation" by CPIC, as it was determined to be not clinically actionable. Other genetic or clinical factors may also affect fentanyl dose requirements.

Fesoterodine	Phenotype	Genetic Test	Results	Source/Evidence Level
Toviaz TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 3[47]

Drug-Gene Interactions:

1 CYP2D6 alleles do not indicate changes from recommended dose

Flecainide	Phenotype	Genetic Test	Results	Source/Evidence Level
Tambocor TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	DPWG[16]

Drug-Gene Interactions:

2 CYP2D6 intermediate metabolizer: reduced metabolism of Flecainide to less active compounds

2 Higher plasma concentrations of active drug may increase the risk of adverse drug reactions

2 Reduce the standard dose by 25%, record electrocardiogram, and monitor plasma concentration

Flibanserin	Phenotype	Genetic Test	Results	Source/Evidence Level
Addyi ReviewGx	Rapid metabolizer	CYP2C19	*1/*17	FDA 1[47]

Drug-Gene Interactions:

1 CYP2C19 alleles do not indicate changes from recommended dose

Fluorouracil	Phenotype	Genetic Test	Results	Source/Evidence Level
Carac Efudex Fluoroplex Tolak ReviewGx	Normal metabolizer	DPYD	*1/*1	CPIC A[5]; FDA 1[47]

Drug-Gene Interactions:

1 DPYD alleles indicate normal DPD activity and typical risk for Fluorouracil toxicity

1 DPYD alleles do not indicate changes from recommended dose

Fluoxetine	Phenotype	Genetic Test	Results	Source/Evidence Level
Prozac TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	Product monograph (actionable)[11]

Drug-Gene Interactions:

1 FDA Product Monograph: No recommended changes or information for this phenotype in the FDA Product Monograph

Flupentixol	Phenotype	Genetic Test	Results	Source/Evidence Level
Fluanxol TreatG ReviewG	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Fluphenazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Modecate TreatG ReviewG	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Flurazepam	Phenotype	Genetic Test	Results	Source/Evidence Level
TreatGx ReviewGx	Normal metabolizer	CYP2C9	*1/*1	Clinical studies[21]

Drug-Gene Interactions:

1 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*1 homozygotes using benzodiazepines had no statistically significant increased fall risk compared to those not taking benzodiazepines (HR = 1.14; 95% CI: 0.90-1.45), whereas those carrying a CYP2C9*2 or *3 allele had a 45% increased fall risk (HR = 1.45; 95% CI: 1.21-1.73). Because the exact role of CYP2C9 in benzodiazepine metabolism is still unclear, additional research is warranted. Additionally, due to sample size, the authors were not able to perform sub-analyses of different benzodiazepine classes or investigate individual benzodiazepines in order to assess a potential class or individual drug effect.

Flurbiprofen	Phenotype	Genetic Test	Results	Source/Evidence Level
Ansaid	Normal metabolizer	CYP2C9 (Star Alleles)	*1/*1	CPIC A[46]; FDA 1[47]

TreatGx ReviewGx

Drug-Gene Interactions:

1 CYP2C9 alleles do not indicate changes from recommended dose

Fluvastatin	Phenotype	Genetic Test	Results	Source/Evidence Level
Lescol	Normal metabolizer	CYP2C9	*1/*1	CPIC A[12]
	Normal function	SLCO1B1	*1/*1	CPIC A[12]

TreatGx ReviewGx

Drug-Gene Interactions:

1 CPIC – CYP2C9 Implication: Normal exposure.

1 CPIC – SLCO1B1 Implication: Typical myopathy risk and Fluvastatin exposure.

1 CPIC – Strong Recommendation: Prescribe desired starting dose and adjust doses of fluvastatin based on disease-specific guidelines.
The potential for drug-drug interactions and dose limits based on renal and hepatic function and ancestry should be evaluated prior to initiating a statin.

Fluvoxamine	Phenotype	Genetic Test	Results	Source/Evidence Level
Luvox	Intermediate metabolizer	CYP2D6	*2.019/*4.001	CPIC B[9]; FDA 3[47]

TreatGx ReviewGx

Drug-Gene Interactions:

2 Reduced metabolism of fluvoxamine to less active compounds when compared with CYP2D6 normal metabolizers. Higher plasma concentrations may increase the probability of side effects.

2 Initiate therapy with recommended starting dose (per CPIC moderate recommendation).

Fosphenytoin	Phenotype	Genetic Test	Results	Source/Evidence Level
Cerebyx	Reduced risk of adverse drug reactions	HLA-B*15:02	Negative	CPIC A[26]; FDA 1[47]
	Normal metabolizer	CYP2C9	*1/*1	CPIC A[26]; FDA 1[47]

ReviewGx

Drug-Gene Interactions:

1 CPIC – CYP2C9 Implication: Normal Fosphenytoin metabolism

1 CPIC – Strong Recommendation: No adjustments needed from typical dosing strategies. Subsequent doses should be adjusted according to therapeutic drug monitoring, response, and side effects. An HLA-B*15:02 negative test does not eliminate the risk of Fosphenytoin-induced Stevens-Johnson syndrome and toxic epidermal necrolysis (SJS/TEN), and patients should be carefully monitored according to standard practice.

Galantamine	Phenotype	Genetic Test	Results	Source/Evidence Level
Razadyne	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 3[47]

TreatGx ReviewGx

Drug-Gene Interactions:

1 CYP2D6 alleles do not indicate changes from recommended dose

Gefitinib	Phenotype	Genetic Test	Results	Source/Evidence Level
Iressa	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]

ReviewGx

Drug-Gene Interactions:

1 FDA PGx Table: No recommended changes or information for this phenotype in the FDA PGx Table

Haloperidol	Phenotype	Genetic Test	Results	Source/Evidence Level
Haldol	Intermediate metabolizer	CYP2D6	*2.019/*4.001	DPWG[16]
	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of side effects e.g. weight gain and hypertriglyceridemia compared to A/A	MC4R rs489693	C/C	PharmGKB 3[48,49]

TreatGx ReviewGx

Drug-Gene Interactions:

1 DPWG – Description: The CYP2D6 genetic variation results in a higher plasma concentration, but the effect is small and no clinically significant effects were found.
DPWG – CYP2D6 Recommendation: No action is required for this gene-drug interaction.

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have a decreased likelihood of experiencing side effects, such as weight gain and hypertriglyceridemia, when treated with haloperidol as compared to patients with the A/A genotype. Other genetic and clinical factors may also influence the likelihood of side effects when treated with haloperidol.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Hydrocodone	Phenotype	Genetic Test	Results	Source/Evidence Level
Hysingla Zohydro	Intermediate metabolizer	CYP2D6	*2.019/*4.001	CPIC B[13]

TreatGx ReviewGx

Drug-Gene Interactions:

1 CPIC – Implication: Minimal evidence for pharmacokinetic or clinical effect.

1 CPIC – Optional Recommendation: Use hydrocodone label recommended age-specific or weight-specific dosing. If no response and opioid use is warranted, consider non-codeine or non-tramadol opioid.

Ibuprofen	Phenotype	Genetic Test	Results	Source/Evidence Level
Advil Caldolor Duexis Motrin IB NeoProfen TreatGx ReviewGx	Normal metabolizer	CYP2C9 (Star Alleles)	*1/*1	CPIC A[46]; FDA 3[47]
	Drug-Gene Interactions: 1 CYP2C9 alleles do not indicate changes from recommended dose			

Iloperidone	Phenotype	Genetic Test	Results	Source/Evidence Level
Fanapt TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]
	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]
	Drug-Gene Interactions: 1 FDA PGx Table: No recommended changes or information for this CYP2D6 phenotype in the FDA PGx Table 1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary. 1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary. 2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.			

Imipramine	Phenotype	Genetic Test	Results	Source/Evidence Level
Tofranil TreatG ReviewG	Intermediate metabolizer	CYP2D6	*2.019/*4.001	CPIC B[23]; FDA 3[47]
	Rapid metabolizer	CYP2C19	*1/*17	CPIC B[23]

Drug-Gene Interactions:

2 CPIC – CYP2D6 Implication: Reduced metabolism of TCAs to less active compounds compared to normal metabolizers. Higher plasma concentrations of active drug will increase the probability of side effects.

2 CPIC – CYP2C19 Implication: Increased metabolism of tertiary amines compared to normal metabolizers. Greater conversion of tertiary amines to secondary amines may affect response or side effects.

2 CPIC – Optional Recommendation: Consider alternative drug not metabolized by CYP2C19. If use is warranted, utilize therapeutic drug monitoring to guide dose adjustment (strongly recommended for a patient with CYP2D6 ultrarapid, intermediate, or poor metabolism in combination with CYP2C19 ultrarapid, intermediate, or poor metabolism). TCAs without major CYP2C19 metabolism include the secondary amines nortriptyline and desipramine.
Recommendations above only apply to higher initial doses of TCAs for treatment of conditions such as depression. Lower dosages are often used for neuropathic pain compared to depressive disorders.
Because of the lower dosage, it is less likely that CYP2D6 intermediate metabolizers will experience adverse effects due to supratherapeutic plasma concentrations. Therefore, CPIC recommends no dose modifications for intermediate metabolizers when prescribed a lower dose for treatment of neuropathic pain, but these patients should be monitored closely for side effects. If larger doses are warranted, CPIC recommends following the gene-based guidelines presented above.
There are limited data to support dose recommendations for CYP2C19*17 carriers who are prescribed TCAs at lower doses for neuropathic pain treatment.

Irinotecan	Phenotype	Genetic Test	Results	Source/Evidence Level
Camptosar Onivyde ReviewG	Normal/Extensive metabolizer	UGT1A1	*1/*1	DPWG[16]; FDA 1[47]; Product monograph (actionable)[24,38]

Drug-Gene Interactions:

1 DPWG: no recommendation for this UGT1A1 phenotype.

1 FDA PGx Table: No recommended changes or information for this UGT1A1 phenotype in the FDA PGx Table

1 FDA Product Monograph: Consider UGT1A1 genotype testing for the *28 and *6 alleles to determine UGT1A1 metabolizer status. No recommended changes or information for this diplotype in the FDA Product Monograph

Isoflurane	Phenotype	Genetic Test	Results	Source/Evidence Level
Forane ReviewGx	Negative	CACNA1S rs1800559	C/C	CPIC A[20]
	Negative	CACNA1S rs772226819	G/G	CPIC A[20]
	Negative	RYR1 rs111888148	G/G	CPIC A[20]
	Negative	RYR1 rs112563513	G/G	CPIC A[20]
	Negative	RYR1 rs118192122	G/G	CPIC A[20]
	Negative	RYR1 rs118192124	C/C	CPIC A[20]
	Negative	RYR1 rs118192161	C/C	CPIC A[20]
	Negative	RYR1 rs118192167	A/A	CPIC A[20]
	Negative	RYR1 rs118192168	G/G	CPIC A[20]
	Negative	RYR1 rs118192170	T/T	CPIC A[20]
	Negative	RYR1 rs118192172	C/C	CPIC A[20]
	Negative	RYR1 rs118192175	C/C	CPIC A[20]
	Negative	RYR1 rs118192176	G/G	CPIC A[20]
	Negative	RYR1 rs121918593	G/G	CPIC A[20]
	Negative	RYR1 rs121918594	G/G	CPIC A[20]
	Negative	RYR1 rs121918595	C/C	CPIC A[20]
	Negative	RYR1 rs144336148	G/G	CPIC A[20]
	Negative	RYR1 rs193922747	T/T	CPIC A[20]
	Negative	RYR1 rs193922748	C/C	CPIC A[20]
	Negative	RYR1 rs193922753	G/G	CPIC A[20]
	Negative	RYR1 rs193922770	C/C	CPIC A[20]
	Negative	RYR1 rs193922802	G/G	CPIC A[20]
	Negative	RYR1 rs193922803	C/C	CPIC A[20]
	Negative	RYR1 rs193922807	G/G	CPIC A[20]
	Negative	RYR1 rs193922809	G/G	CPIC A[20]
	Negative	RYR1 rs193922816	C/C	CPIC A[20]
	Negative	RYR1 rs193922818	G/G	CPIC A[20]
	Negative	RYR1 rs193922832	G/G	CPIC A[20]
	Negative	RYR1 rs193922843	G/G	CPIC A[20]
	Negative	RYR1 rs193922876	C/C	CPIC A[20]
	Negative	RYR1 rs193922878	C/C	CPIC A[20]
	Negative	RYR1 rs28933396	G/G	CPIC A[20]
	Negative	RYR1 rs28933397	C/C	CPIC A[20]
	Negative	RYR1 rs63749869	G/G	CPIC A[20]

Drug-Gene Interactions:

1 Uncertain risk of developing malignant hyperthermia (MH)

1 Results do not eliminate malignant hyperthermia susceptibility. Clinical findings, family history, further genetic testing, and other laboratory data should guide use of Isoflurane.

Lamotrigine	Phenotype	Genetic Test	Results	Source/Evidence Level
Lamictal	Reduced risk of adverse drug reactions	HLA-B*15:02	Negative	DPWG[16]
TreatG ReviewG	Increased risk of adverse drug reactions	HLA-B*58:01	Positive	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 DPWG: no recommendation for this HLA-B*15:02 phenotype.

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with one or two copies of the HLA-B*58:01 allele have an increased risk of hypersensitivity reactions, such as Stevens-Johnson Syndrome, toxic epidermal necrolysis or maculopapular eruption, when treated with lamotrigine as compared to patients with no HLA-B*58:01 alleles or negative for the HLA-B*58:01 test. Other genetic and clinical factors may also influence a patient's risk of lamotrigine-induced adverse reactions.

Lansoprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Prevacid	Rapid metabolizer	CYP2C19	*1/*17	CPIC A[28]; FDA 3[47]
TreatG ReviewG				

Drug-Gene Interactions:

2 CPIC – Implication: Decreased plasma concentrations of PPIs compared with CYP2C19 NMs; increased risk of therapeutic failure.

2 CPIC – Moderate Recommendation: Initiate standard starting daily dose. Consider increasing dose by 50–100% for the treatment of Helicobacter pylori infection and erosive esophagitis. Daily dose may be given in divided doses. Monitor for efficacy.

Lofexidine	Phenotype	Genetic Test	Results	Source/Evidence Level
Lucemyra	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]
ReviewG				

Drug-Gene Interactions:

1 CYP2D6 alleles do not indicate changes from recommended dose

Lorazepam	Phenotype	Genetic Test	Results	Source/Evidence Level
Ativan	Normal metabolizer	CYP2C9	*1/*1	Clinical studies[21]
ReviewG				

Drug-Gene Interactions:

1 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*1 homozygotes using benzodiazepines had no statistically significant increased fall risk compared to those not taking benzodiazepines (HR = 1.14; 95% CI: 0.90-1.45), whereas those carrying a CYP2C9*2 or *3 allele had a 45% increased fall risk (HR = 1.45; 95% CI: 1.21-1.73). Because the exact role of CYP2C9 in benzodiazepine metabolism is still unclear, additional research is warranted. Additionally, due to sample size, the authors were not able to perform sub-analyses of different benzodiazepine classes or investigate individual benzodiazepines in order to assess a potential class or individual drug effect.

Lovastatin	Phenotype	Genetic Test	Results	Source/Evidence Level
Altoprev	Normal function	SLCO1B1	*1/*1	CPIC A[12]
TreatG ReviewG				

Drug-Gene Interactions:

1 CPIC – Implication: Typical myopathy risk and Lovastatin exposure.

1 CPIC – Strong Recommendation: Prescribe desired starting dose and adjust doses based on disease-specific guidelines.
The potential for drug-drug interactions and dose limits based on renal and hepatic function and ancestry should be evaluated prior to initiating a statin.

Loxapine	Phenotype	Genetic Test	Results	Source/Evidence Level
Adasuve Loxapac TreatGx ReviewGx	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Lurasidone	Phenotype	Genetic Test	Results	Source/Evidence Level
Latuda TreatGx ReviewGx	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Lusutrombopag	Phenotype	Genetic Test	Results	Source/Evidence Level
Mupleta ReviewGx	Normal Factor II	Factor II rs1799963	G/G	Product monograph (actionable)[44]
	Normal Factor V Leiden	Factor V rs6025	C/C	Product monograph (actionable)[44]

Drug-Gene Interactions:

1. FDA Product Monograph: No recommended changes or information for normal Factor II (i.e. Prothrombin 20210A mutation absent) in the FDA Product Monograph.
1. FDA Product Monograph: No recommended changes or information for normal Factor V in the FDA Product Monograph.

Mavacamten	Phenotype	Genetic Test	Results	Source/Evidence Level
Camzyos ReviewGx	Rapid metabolizer	CYP2C19	*1/*17	FDA 2[47]

Drug-Gene Interactions:

1. FDA PGx Table: No recommended changes or information for this phenotype in the FDA PGx Table

Meclizine	Phenotype	Genetic Test	Results	Source/Evidence Level
Antivert ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]

Drug-Gene Interactions:

2. CYP2D6 intermediate metabolizer: reduced metabolism of Meclizine to less active compounds
2. Higher plasma concentrations of active drug may increase the risk of adverse drug reactions
2. This drug has an FDA therapeutic recommendation, refer to drug monograph or FDA labelling for dosing recommendations

Meloxicam	Phenotype	Genetic Test	Results	Source/Evidence Level
Anjeso Mobic Qmiiz ODT Vivlodex TreatGx ReviewGx	Normal metabolizer	CYP2C9 (Star Alleles)	*1/*1	CPIC A[46]; FDA 1[47]

Drug-Gene Interactions:

1. CYP2C9 alleles do not indicate changes from recommended dose

Mercaptopurine	Phenotype	Genetic Test	Results	Source/Evidence Level
Purinethol Purixan TreatGx ReviewGx	Normal metabolizer	TPMT	*1/*1	CPIC A[40,45]; FDA 1[47]
	Normal metabolizer	NUDT15	*1/*1	CPIC A[40,45]; FDA 1[47]

Drug-Gene Interactions:

1. CPIC – TPMT Implication: Lower concentrations of thioguanine nucleotides metabolites, higher metabolites of thiopurine methyltransferase, this is the 'normal' pattern. Normal risk of thiopurine-related leukopenia, neutropenia, myelosuppression.
1. CPIC – NUDT15 Implication: Normal risk of thiopurine-related leukopenia, neutropenia, myelosuppression.
1. CPIC – Strong Recommendation: Start with normal starting dose (e.g., 75 mg/m2/day or 1.5 mg/kg/day) and adjust doses of mercaptopurine (and of any other myelosuppressive therapy) without any special emphasis on mercaptopurine compared to other agents. Allow at least 2 weeks to reach steady-state after each dose adjustment.
Normal starting doses vary by race/ethnicity and treatment regimens.

Methotrexate	Phenotype	Genetic Test	Results	Source/Evidence Level
Metoject Otrexup Rasuvo Trexall Xatmep TreatGx ReviewGx	Increased risk of toxicity compared to G/G or decreased compared to A/A	MTHFR rs1801133	A/G	PharmGKB 2A[48,49]

Drug-Gene Interactions:

2 PharmGKB – Clinical Annotation (Level 2A Toxicity): Patients with the MTHFR rs1801133 A/G genotype and cancer or arthritis who are treated with methotrexate may have an increased risk of toxicity as compared to patients with the G/G genotype, or may have a decreased risk of adverse events as compared to patients with the A/A genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence methotrexate toxicity. This drug-variant pair has been assigned a "no recommendation" by DPWG, as it was determined to be not clinically actionable.

Methotrimeprazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Nozinan TreatGx ReviewGx	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Methoxyflurane	Phenotype	Genetic Test	Results	Source/Evidence Level
Penthrox	Negative	CACNA1S rs1800559	C/C	CPIC A[20]
TreatG	Negative	CACNA1S rs772226819	G/G	CPIC A[20]
ReviewG	Negative	RYR1 rs111888148	G/G	CPIC A[20]
	Negative	RYR1 rs112563513	G/G	CPIC A[20]
	Negative	RYR1 rs118192122	G/G	CPIC A[20]
	Negative	RYR1 rs118192124	C/C	CPIC A[20]
	Negative	RYR1 rs118192161	C/C	CPIC A[20]
	Negative	RYR1 rs118192167	A/A	CPIC A[20]
	Negative	RYR1 rs118192168	G/G	CPIC A[20]
	Negative	RYR1 rs118192170	T/T	CPIC A[20]
	Negative	RYR1 rs118192172	C/C	CPIC A[20]
	Negative	RYR1 rs118192175	C/C	CPIC A[20]
	Negative	RYR1 rs118192176	G/G	CPIC A[20]
	Negative	RYR1 rs121918593	G/G	CPIC A[20]
	Negative	RYR1 rs121918594	G/G	CPIC A[20]
	Negative	RYR1 rs121918595	C/C	CPIC A[20]
	Negative	RYR1 rs144336148	G/G	CPIC A[20]
	Negative	RYR1 rs193922747	T/T	CPIC A[20]
	Negative	RYR1 rs193922748	C/C	CPIC A[20]
	Negative	RYR1 rs193922753	G/G	CPIC A[20]
	Negative	RYR1 rs193922770	C/C	CPIC A[20]
	Negative	RYR1 rs193922802	G/G	CPIC A[20]
	Negative	RYR1 rs193922803	C/C	CPIC A[20]
	Negative	RYR1 rs193922807	G/G	CPIC A[20]
	Negative	RYR1 rs193922809	G/G	CPIC A[20]
	Negative	RYR1 rs193922816	C/C	CPIC A[20]
	Negative	RYR1 rs193922818	G/G	CPIC A[20]
	Negative	RYR1 rs193922832	G/G	CPIC A[20]
	Negative	RYR1 rs193922843	G/G	CPIC A[20]
	Negative	RYR1 rs193922876	C/C	CPIC A[20]
	Negative	RYR1 rs193922878	C/C	CPIC A[20]
	Negative	RYR1 rs28933396	G/G	CPIC A[20]
	Negative	RYR1 rs28933397	C/C	CPIC A[20]
	Negative	RYR1 rs63749869	G/G	CPIC A[20]

Drug-Gene Interactions:

1 Uncertain risk of developing malignant hyperthermia (MH)

1 Results do not eliminate malignant hyperthermia susceptibility. Clinical findings, family history, further genetic testing, and other laboratory data should guide use of Methoxyflurane.

Methylene blue	Phenotype	Genetic Test	Results	Source/Evidence Level
Provayblue	Normal phenotype	G6PD	B (reference)/B (reference)	CPIC A[18]
ReviewG				

Drug-Gene Interactions:

1 CPIC – Implication: Low risk of acute hemolytic anemia.

1 CPIC – Strong Recommendation: No reason to avoid ased on G6PD status.

Methylphenidate	Phenotype	Genetic Test	Results	Source/Evidence Level
Aptensio Concerta Cotempla Daytrana Jornay Metadate Methylin Quillichew Quillivant Relexxiii Ritalin TreatGx ReviewGx	Poorer response	ADRA2A rs1800544	C/C	PharmGKB 4[48,49]
	No significant association to response	COMT rs4680	G/G	PharmGKB 4[48,49]
	Drug-Gene Interactions: 1 PharmGKB – Clinical Annotation (Level 4 Efficacy): The current evidence base suggests that there is no significant association between the COMT rs4680 G/G genotype and response to methylphenidate. However, conflicting evidence has been reported. This drug-variant pair has been assigned a "no recommendation" by DPWG, as it was determined to be not clinically actionable. Other genetic and clinical factors may also influence response to methylphenidate. 2 PharmGKB – Clinical Annotation (Level 4 Efficacy): Patients (mainly pediatric patients) with the ADRA2A rs1800544 C/C genotype and attention deficit hyperactivity disorder (ADHD) may have a poorer response to methylphenidate treatment as compared to patients with the C/G or G/G genotype. However, contradictory evidence exists for this association. Studies used different scales to analyze improvement, e.g. CGI-I, ARS-IV, and other. Other genetic and clinical factors may also influence response to methylphenidate.			

Metoclopramide	Phenotype	Genetic Test	Results	Source/Evidence Level
Metonia Reglan TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]
	Drug-Gene Interactions: 1 CYP2D6 alleles do not indicate changes from recommended dose			

Metoprolol	Phenotype	Genetic Test	Results	Source/Evidence Level
Kapspargo Sprinkle Lopressor Toprol-XL TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	CPIC B[15]
	Drug-Gene Interactions: 1 CPIC – Implication: Decreased metabolism of metoprolol leading to increased drug concentrations; however, this does not appear to translate into clinically significant changes in heart rate, blood pressure, or clinical outcomes. 1 CPIC – Moderate Recommendation: Initiate standard dosing.			

Mirabegron	Phenotype	Genetic Test	Results	Source/Evidence Level
Myrbetriq TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 3[47]
	Drug-Gene Interactions: 1 CYP2D6 alleles do not indicate changes from recommended dose			

Molindone	Phenotype	Genetic Test	Results	Source/Evidence Level
Moban TreatGx ReviewGx	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Morphine	Phenotype	Genetic Test	Results	Source/Evidence Level
Kadian M-Eslon Morphabond ER MS Contin MS-IR Statex TreatGx ReviewGx	Increased analgesic response	OPRM1 rs1799971	A/A	PharmGKB 3[48,49]

Drug-Gene Interactions:

2 PharmGKB – Clinical Annotation (Level 3 Efficacy): Patients with the OPRM1 rs1799971 A/A genotype may have an increased analgesic response to morphine as compared to patients with the A/G or G/G genotypes. However, conflicting evidence has been reported. This drug-variant pair has been assigned a "no recommendation" by CPIC, as it was determined to be not clinically actionable. Other genetic or clinical factors may also affect response to morphine.
PharmGKB – Clinical Annotation (Level 3 Dosage): Patients with the OPRM1 rs1799971 A/A genotype may have decreased morphine dose requirements as compared to patients with the A/G or G/G genotypes. However, conflicting evidence has been reported. This drug-variant pair has been assigned a "no recommendation" by CPIC, as it was determined to be not clinically actionable. Other genetic or clinical factors may also affect morphine dose requirements.

Nateglinide	Phenotype	Genetic Test	Results	Source/Evidence Level
ReviewGx	Normal metabolizer	CYP2C9	*1/*1	FDA 1[47]

Drug-Gene Interactions:

1 FDA PGx Table: No recommended changes or information for this phenotype in the FDA PGx Table

Nebivolol	Phenotype	Genetic Test	Results	Source/Evidence Level
Bystolic TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 3[47]

Drug-Gene Interactions:

1 CYP2D6 alleles do not indicate changes from recommended dose

Nicotine replacement therapy	Phenotype	Genetic Test	Results	Source/Evidence Level
Nicorette Nicotrol Habitrol Nicoderm Thrive TreatGx ReviewGx	Increased likelihood of smoking cessation compared to G/G	ANKK1/DRD2 rs1800497	A/G	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 PharmGKB – Clinical Annotation (Level 3 Efficacy): Patients with the ANKK1 rs1800497 A/G genotype may have an increased likelihood of smoking cessation when treated with nicotine replacement therapy as compared to patients with the G/G genotype. However, contradictory findings have been reported. Other genetic and clinical factors may influence a patient's likelihood of smoking cessation.

Nitrazepam	Phenotype	Genetic Test	Results	Source/Evidence Level
Mogadon ReviewGx	Normal metabolizer	CYP2C9	*1/*1	Clinical studies[21]

Drug-Gene Interactions:

1 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*1 homozygotes using benzodiazepines had no statistically significant increased fall risk compared to those not taking benzodiazepines (HR = 1.14; 95% CI: 0.90-1.45), whereas those carrying a CYP2C9*2 or *3 allele had a 45% increased fall risk (HR = 1.45; 95% CI: 1.21-1.73). Because the exact role of CYP2C9 in benzodiazepine metabolism is still unclear, additional research is warranted. Additionally, due to sample size, the authors were not able to perform sub-analyses of different benzodiazepine classes or investigate individual benzodiazepines in order to assess a potential class or individual drug effect.

Nitrofurantoin	Phenotype	Genetic Test	Results	Source/Evidence Level
Macrobid Macrodantin Furadantin TreatGx ReviewGx	Normal phenotype	G6PD	B (reference)/B (reference)	CPIC A[18]

Drug-Gene Interactions:

1 CPIC – Implication: Low risk of acute hemolytic anemia.

1 CPIC – Strong Recommendation: No reason to avoid based on G6PD status.

Nortriptyline	Phenotype	Genetic Test	Results	Source/Evidence Level
Aventyl Pamelor TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	CPIC A[23]; FDA 3[47]

Drug-Gene Interactions:

2 CPIC – CYP2D6 Implication: Reduced metabolism of TCAs to less active compounds compared to normal metabolizers. Higher plasma concentrations of active drug will increase the probability of side effects.

2 CPIC – Moderate Recommendation: Consider a 25% reduction of recommended starting dose. Patients may receive an initial low dose of a tricyclic, which is then increased over several days to the recommended steady-state dose. The starting dose in this guideline refers to the recommended steady-state dose. Utilize therapeutic drug monitoring to guide dose adjustments. Titrate dose to observed clinical response with symptom improvement and minimal (if any) side effects.
Recommendations above only apply to higher initial doses of TCAs for treatment of conditions such as depression. Lower dosages are often used for neuropathic pain compared to depressive disorders.
Because of the lower dosage, it is less likely that CYP2D6 intermediate metabolizers will experience adverse effects due to supratherapeutic plasma concentrations. Therefore, CPIC recommends no dose modifications for intermediate metabolizers when prescribed a lower dose for treatment of neuropathic pain, but these patients should be monitored closely for side effects. If larger doses are warranted, CPIC recommends following the gene-based guidelines presented above.

Olanzapine	Phenotype	Genetic Test	Results	Source/Evidence Level
Zyprexa TreatG ReviewG	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	May have increased time until response	DRD2 rs1799978	T/T	PharmGKB 3[48,49]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

2 PharmGKB – Clinical Annotation (Level 3 Response): Patients with the DRD2 rs1799978 T/T genotype and schizophrenia who are treated with olanzapine or risperidone may have increased time until response as compared to patients with the C/C or C/T genotype. Other genetic and clinical factors may also influence a patient's response to olanzapine or risperidone.

Oliceridine	Phenotype	Genetic Test	Results	Source/Evidence Level
Olinvyk	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]
ReviewG				

Drug-Gene Interactions:

1 FDA PGx Table: No recommended changes or information for this phenotype in the FDA PGx Table

Omeprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Losec Olex Prilosec	Rapid metabolizer	CYP2C19	*1/*17	CPIC A[28]; FDA 3[47]
TreatG ReviewG				

Drug-Gene Interactions:

2 CPIC – Implication: Decreased plasma concentrations of PPIs compared with CYP2C19 NMs; increased risk of therapeutic failure.

2 CPIC – Moderate Recommendation: Initiate standard starting daily dose. Consider increasing dose by 50–100% for the treatment of Helicobacter pylori infection and erosive esophagitis. Daily dose may be given in divided doses. Monitor for efficacy.

Ondansetron	Phenotype	Genetic Test	Results	Source/Evidence Level
Zofran Zuplenz	Intermediate metabolizer	CYP2D6	*2.019/*4.001	CPIC A[7]
ReviewG				

Drug-Gene Interactions:

1 CPIC – Implication: Very limited data available for CYP2D6 intermediate metabolizers.

1 CPIC – No Recommendation: Insufficient evidence demonstrating clinical impact based on CYP2D6 genotype. Initiate therapy with recommended starting dose.
Drug-drug interactions and other patient characteristics (e.g., age, renal function, and liver function) should be considered when selecting alternative therapy.

Oral contraceptives	Phenotype	Genetic Test	Results	Source/Evidence Level
ReviewGx	Decreased risk for DVT	Factor II rs1799963	G/G	PharmGKB 2B[48,49]
	Decreased risk of thrombosis (normal Factor V)	Factor V rs6025	C/C	PharmGKB 2B[48,49]

Drug-Gene Interactions:

1. PharmGKB – Clinical Annotation (Level 2B Toxicity): Patients with the Factor II rs1799963 G/G genotype who are taking oral contraceptives may have a decreased risk for deep vein thrombosis (DVT), as compared to patients with the A/A or A/G genotypes or those who are not taking oral contraceptives. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence risk for DVT in patients taking oral contraceptives.

1. PharmGKB – Clinical Annotation (Level 2B Toxicity): Patients with the rs6025 C/C genotype (normal Factor V) may have a decreased risk of experiencing thrombosis when receiving oral contraceptives as compared to patients with the C/T or T/T genotype (carriers of Factor V Leiden). However, conflicting evidence has been reported. Both Factor V Leiden and oral contraceptives have been found to independently increase the risk for thrombosis, but together they may have a cumulative effect on thrombosis risk. Other genetic and clinical factors may also influence risk of thrombosis.

Oxazepam	Phenotype	Genetic Test	Results	Source/Evidence Level
ReviewGx	Normal metabolizer	CYP2C9	*1/*1	Clinical studies[21]

Drug-Gene Interactions:

1. Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*1 homozygotes using benzodiazepines had no statistically significant increased fall risk compared to those not taking benzodiazepines (HR = 1.14; 95% CI: 0.90-1.45), whereas those carrying a CYP2C9*2 or *3 allele had a 45% increased fall risk (HR = 1.45; 95% CI: 1.21-1.73). Because the exact role of CYP2C9 in benzodiazepine metabolism is still unclear, additional research is warranted. Additionally, due to sample size, the authors were not able to perform sub-analyses of different benzodiazepine classes or investigate individual benzodiazepines in order to assess a potential class or individual drug effect.

Oxcarbazepine	Phenotype	Genetic Test	Results	Source/Evidence Level
Oxtellar XR Trileptal	Reduced risk of adverse drug reactions	HLA-B*15:02	Negative	CPIC A[39]; FDA 2[47]
TreatGx ReviewGx				

Drug-Gene Interactions:

1. CPIC – Implication: Normal risk of oxcarbazepine-induced Stevens-Johnson syndrome and toxic epidermal necrolysis (SJS/TEN).

1. CPIC – Strong Recommendation: Use oxcarbazepine per standard dosing guidelines.

Paliperidone	Phenotype	Genetic Test	Results	Source/Evidence Level
Invega TreatGx ReviewGx	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain compared to A/A	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1. PharmGKB – Clinical Annotation (Level 3 Efficacy): Patients with the MC4R rs489693 C/C genotype may be at a decreased risk of experiencing weight gain when treated with paliperidone as compared to patients with the A/A genotype. Other genetic and clinical factors may also influence risk of experiencing weight gain when treated with paliperidone.
1. 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.
2. 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Pantoprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Pantoloc Protonix Tecta TreatGx ReviewGx	Rapid metabolizer	CYP2C19	*1/*17	CPIC A[28]; FDA 1[47]

Drug-Gene Interactions:

2. CPIC – Implication: Decreased plasma concentrations of PPIs compared with CYP2C19 NMs; increased risk of therapeutic failure.
2. CPIC – Moderate Recommendation: Initiate standard starting daily dose. Consider increasing dose by 50–100% for the treatment of Helicobacter pylori infection and erosive esophagitis. Daily dose may be given in divided doses. Monitor for efficacy.

Paroxetine	Phenotype	Genetic Test	Results	Source/Evidence Level
Brisdelle Paxil Pexeva TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	CPIC A[9]; FDA 3[47]

Drug-Gene Interactions:

2. Reduced metabolism of paroxetine to less active compounds when compared with CYP2D6 normal metabolizers when starting treatment or at lower doses. Higher plasma concentrations may increase the probability of side effects. Paroxetine-associated phenoconversion of intermediate metabolizers to poor metabolizers due to CYP2D6 autoinhibition may occur and is dose-dependent and greater at steady-state concentrations.
2. Consider a lower starting dose and slower titration schedule as compared with normal metabolizers (per CPIC optional recommendation).

Pazopanib	Phenotype	Genetic Test	Results	Source/Evidence Level
Votrient ReviewGx	Reduced risk of adverse drug reactions	HLA-B*57:01	Negative	FDA 2[47]

Drug-Gene Interactions:

1. FDA PGx Table: No recommended changes or information for this HLA-B *57:01 phenotype in the FDA PGx Table

PEG-interferon alpha	Phenotype	Genetic Test	Results	Source/Evidence Level
Alferon N Injection Intron A Pegasys Pegintron Sylatron ReviewGx	Reduced response	IFNL3 rs12979860	T/T	CPIC A[33]

Drug-Gene Interactions:

- 2 IFNL3 alleles indicate decreased likelihood of sustained virologic response (SVR) when treating HCV with PEG-IFN-alpha and ribavirin (approximately 30% after 48 weeks)
- 2 The reduced likelihood of SVR is more prominent in HCV genotypes 1 and 4 (OR = 0.21; 95% CI 0.19-0.25) than in HCV genotypes 2 and 3 (OR = 0.77; 95% CI 0.60-0.98)
- 2 The likelihood of SVR after 24-48 weeks of treatment is approximately 60% when a protease inhibitor is added
- 1 Consider implications before initiating PEG-IFN alpha and ribavirin containing regimens

Pegloticase	Phenotype	Genetic Test	Results	Source/Evidence Level
Krystexxa ReviewGx	Normal phenotype	G6PD	B (reference)/B (reference)	CPIC A[18]

Drug-Gene Interactions:

- 1 CPIC – Implication: Low risk of acute hemolytic anemia.
- 1 CPIC – Strong Recommendation: No reason to avoid based on G6PD status.

Perphenazine	Phenotype	Genetic Test	Results	Source/Evidence Level
TreatGx ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 2[47]
	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

- 1 FDA PGx Table: No recommended changes or information for this CYP2D6 phenotype in the FDA PGx Table
- 1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.
- 1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.
- 2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Phenytoin	Phenotype	Genetic Test	Results	Source/Evidence Level
Dilantin Tremytoine Phenytek	Reduced risk of adverse drug reactions	HLA-B*15:02	Negative	CPIC A[26]; FDA 1[47]
	Normal metabolizer	CYP2C9	*1/*1	CPIC A[26]; FDA 1[47]

ReviewGx

Drug-Gene Interactions:

1 CPIC – CYP2C9 Implication: Normal Phenytoin metabolism

1 CPIC – Strong Recommendation: No adjustments needed from typical dosing strategies. Subsequent doses should be adjusted according to therapeutic drug monitoring, response, and side effects. An HLA-B*15:02 negative test does not eliminate the risk of Phenytoin-induced Stevens-Johnson syndrome and toxic epidermal necrolysis (SJS/TEN), and patients should be carefully monitored according to standard practice.

Pimozide	Phenotype	Genetic Test	Results	Source/Evidence Level
Orap	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]
	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

TreatGx
ReviewGx

Drug-Gene Interactions:

1 FDA PGx Table: No recommended changes or information for this CYP2D6 phenotype in the FDA PGx Table

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Piroxicam	Phenotype	Genetic Test	Results	Source/Evidence Level
Feldene	Normal metabolizer	CYP2C9 (Star Alleles)	*1/*1	CPIC A[46]; FDA 1[47]

TreatGx
ReviewGx

Drug-Gene Interactions:

1 CYP2C9 alleles do not indicate changes from recommended dose

Pitavastatin	Phenotype	Genetic Test	Results	Source/Evidence Level
Livalo Zypitamag	Normal function	SLCO1B1	*1/*1	CPIC A[12]

Drug-Gene Interactions:

1 CPIC – Implication: Typical myopathy risk and Pitavastatin exposure.

TreatGx
ReviewGx

1 CPIC – Strong Recommendation: Prescribe desired starting dose and adjust doses based on disease-specific guidelines.
The potential for drug-drug interactions and dose limits based on renal and hepatic function and ancestry should be evaluated prior to initiating a statin.

Pitolisant	Phenotype	Genetic Test	Results	Source/Evidence Level
Wakix ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]; Product monograph (actionable)[22]

Drug-Gene Interactions:

1 FDA PGx Table: No recommended changes or information for this phenotype in the FDA PGx Table

Pravastatin	Phenotype	Genetic Test	Results	Source/Evidence Level
Pravachol TreatGx ReviewGx	Normal function	SLCO1B1	*1/*1	CPIC A[12]

Drug-Gene Interactions:

1 CPIC – Implication: Typical myopathy risk and Pravastatin exposure.

1 CPIC – Strong Recommendation: Prescribe desired starting dose and adjust doses based on disease-specific guidelines.
The potential for drug-drug interactions and dose limits based on renal and hepatic function and ancestry should be evaluated prior to initiating a statin.

Primaquine	Phenotype	Genetic Test	Results	Source/Evidence Level
Aralen ReviewGx	Normal phenotype	G6PD	B (reference)/B (reference)	CPIC A[18]

Drug-Gene Interactions:

1 CPIC – Implication: Low risk of acute hemolytic anemia.

1 CPIC – Strong Recommendation: No reason to avoid based on G6PD status.

Prochlorperazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Compro TreatGx ReviewGx	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Promethazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Phenadoz Promethegan TreatG ReviewG	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Propafenone	Phenotype	Genetic Test	Results	Source/Evidence Level
Rythmol TreatG ReviewG	Intermediate metabolizer	CYP2D6	*2.019/*4.001	DPWG[16]; FDA 1[47]

Drug-Gene Interactions:

2 CYP2D6 intermediate metabolizer: reduced metabolism of Propafenone to less active compounds

2 Higher plasma concentrations of active drug may increase the risk of adverse drug reactions

2 Adjust dose in response to plasma concentration and record electrocardiogram or select an alternative drug

Propranolol	Phenotype	Genetic Test	Results	Source/Evidence Level
Inderal Innopran TreatG ReviewG	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 3[47]

Drug-Gene Interactions:

1 CYP2D6 alleles do not indicate changes from recommended dose

Protriptyline	Phenotype	Genetic Test	Results	Source/Evidence Level
Vivactil ReviewG	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 3[47]

Drug-Gene Interactions:

1 CYP2D6 alleles do not indicate changes from recommended dose

Quetiapine	Phenotype	Genetic Test	Results	Source/Evidence Level
Seroquel TreatGx ReviewGx	Normal metabolizer	CYP3A4	*1/*1	DPWG[16]
	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of side effects e.g. weight gain and hypertriglyceridemia compared to A/A	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 DPWG: no recommendation for this CYP3A4 phenotype.

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have a decreased likelihood of experiencing side effects, such as weight gain and hypertriglyceridemia, when treated with quetiapine as compared to patients with the A/A genotype. Other genetic and clinical factors may also influence the likelihood of side effects when treated with quetiapine.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Rabeprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Aciphex Pariet TreatGx ReviewGx	Rapid metabolizer	CYP2C19	*1/*17	FDA 3[47]

Drug-Gene Interactions:

1 FDA PGx Table: No recommended changes or information for this phenotype in the FDA PGx Table

Rasburicase	Phenotype	Genetic Test	Results	Source/Evidence Level
Elitek Fasturtec ReviewGx	Normal phenotype	G6PD	B (reference)/B (reference)	CPIC A[18]

Drug-Gene Interactions:

1 CPIC – Implication: Low risk of acute hemolytic anemia.

1 CPIC – Strong Recommendation: No reason to avoid based on G6PD status.

Risperidone	Phenotype	Genetic Test	Results	Source/Evidence Level
Perseris Risperdal	Intermediate metabolizer	CYP2D6	*2.019/*4.001	DPWG[16]; FDA 3[47]
	Increased prolactin compared to G/G	ANKK1/DRD2 rs1800497	A/G	PharmGKB 3[48,49]
	May have increased time until response	DRD2 rs1799978	T/T	PharmGKB 3[48,49]
	Decreased risk of developing metabolic syndrome	HTR2C rs1414334	G/G	PharmGKB 3[48,49]
	Decreased likelihood of side effects e.g. weight gain and hypertriglyceridemia compared to A/A	MC4R rs489693	C/C	PharmGKB 3[48,49]

TreatG
ReviewG

Drug-Gene Interactions:

1. FDA PGx Table: No recommended changes or information for this CYP2D6 phenotype in the FDA PGx Table
1. DPWG – CYP2D6 Description: There is little evidence to support an increase in side effects caused by the gene variation. The gene variation may lead to a decrease in the required maintenance dose. However, as the effect on the dose is smaller than that of the normal biological variation, action is not useful.
   DPWG – CYP2D6 Recommendation: NO action is needed for this gene-drug interaction.
1. PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with two X-chromosomes and the HTR2C rs1414334 G/G genotype, or one X-chromosome and the G genotype who are treated with Risperidone may have a decreased risk of developing metabolic syndrome as compared to patients with the C/C, C/G, or C genotype. Other genetic and clinical factors may also influence a patient's risk for developing metabolic syndrome.
1. PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may be at a decreased risk of experiencing side effects, such as weight gain and hypertriglyceridemia, when treated with risperidone as compared to patients with the A/A genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence risk of experiencing side effects when treated with risperidone.
2. PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the ANKK1/DRD2 rs1800497 A/G genotype and schizophrenia may have increased prolactin when treated with risperidone as compared to patients with the G/G genotype. Other genetic and clinical factors may also influence risperidone related hyperprolactinemia.
2. PharmGKB – Clinical Annotation (Level 3 Response): Patients with the DRD2 rs1799978 T/T genotype and schizophrenia who are treated with olanzapine or risperidone may have increased time until response as compared to patients with the C/C or C/T genotype. Other genetic and clinical factors may also influence a patient's response to olanzapine or risperidone.

Rosuvastatin	Phenotype	Genetic Test	Results	Source/Evidence Level
Crestor	Normal function	SLCO1B1	*1/*1	CPIC A[12]; FDA 3[47]
	Decreased function	ABCG2 rs2231142	G/T	CPIC A[12]

TreatG
ReviewG

Drug-Gene Interactions:

1. CPIC – SLCO1B1 Implication: Typical myopathy risk and Rosuvastatin exposure.
1. CPIC – ABCG2 Implication: Increased rosuvastatin exposure as compared with normal function; unknown risk for myopathy; increased lipid-lowering effects.
1. CPIC – Moderate Recommendation: Prescribe desired starting dose and adjust doses of rosuvastatin based on disease-specific and population-specific guidelines.
   The potential for drug-drug interactions and dose limits based on renal and hepatic function and Asian ancestry should be evaluated prior to initiating rosuvastatin. The effects of drug-drug interactions may be more pronounced, resulting in a higher risk of myopathy.

Salmeterol	Phenotype	Genetic Test	Results	Source/Evidence Level
Serevent TreatGx ReviewGx	Increased response compared to A/A	ADRB2 rs1042713	G/G	PharmGKB 2A[48,49]

Drug-Gene Interactions:

1 PharmGKB – Clinical Annotation (Level 2A Efficacy): Patients with the ADRB2 rs1042713 G/G genotype and asthma may have an increased response to salmeterol as compared to patients with the A/A genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence response to salmeterol.

Sertraline	Phenotype	Genetic Test	Results	Source/Evidence Level
Zoloft	Intermediate metabolizer	CYP2B6	*1/*6	CPIC B[9]
	Rapid metabolizer	CYP2C19	*1/*17	CPIC A[9]
TreatGx ReviewGx				

Drug-Gene Interactions:

1 Reduced metabolism of sertraline to less active compounds when compared with CYP2B6 normal metabolizers.

1 Small increase in metabolism of sertraline to less active compounds when compared with CYP2C19 normal metabolizers.

1 Initiate therapy with recommended starting dose (per CPIC moderate recommendation).

Sevoflurane	Phenotype	Genetic Test	Results	Source/Evidence Level
ReviewGx	Negative	CACNA1S rs1800559	C/C	CPIC A[20]
	Negative	CACNA1S rs772226819	G/G	CPIC A[20]
	Negative	RYR1 rs111888148	G/G	CPIC A[20]
	Negative	RYR1 rs112563513	G/G	CPIC A[20]
	Negative	RYR1 rs118192122	G/G	CPIC A[20]
	Negative	RYR1 rs118192124	C/C	CPIC A[20]
	Negative	RYR1 rs118192161	C/C	CPIC A[20]
	Negative	RYR1 rs118192167	A/A	CPIC A[20]
	Negative	RYR1 rs118192168	G/G	CPIC A[20]
	Negative	RYR1 rs118192170	T/T	CPIC A[20]
	Negative	RYR1 rs118192172	C/C	CPIC A[20]
	Negative	RYR1 rs118192175	C/C	CPIC A[20]
	Negative	RYR1 rs118192176	G/G	CPIC A[20]
	Negative	RYR1 rs121918593	G/G	CPIC A[20]
	Negative	RYR1 rs121918594	G/G	CPIC A[20]
	Negative	RYR1 rs121918595	C/C	CPIC A[20]
	Negative	RYR1 rs144336148	G/G	CPIC A[20]
	Negative	RYR1 rs193922747	T/T	CPIC A[20]
	Negative	RYR1 rs193922748	C/C	CPIC A[20]
	Negative	RYR1 rs193922753	G/G	CPIC A[20]
	Negative	RYR1 rs193922770	C/C	CPIC A[20]
	Negative	RYR1 rs193922802	G/G	CPIC A[20]
	Negative	RYR1 rs193922803	C/C	CPIC A[20]
	Negative	RYR1 rs193922807	G/G	CPIC A[20]
	Negative	RYR1 rs193922809	G/G	CPIC A[20]
	Negative	RYR1 rs193922816	C/C	CPIC A[20]
	Negative	RYR1 rs193922818	G/G	CPIC A[20]
	Negative	RYR1 rs193922832	G/G	CPIC A[20]
	Negative	RYR1 rs193922843	G/G	CPIC A[20]
	Negative	RYR1 rs193922876	C/C	CPIC A[20]
	Negative	RYR1 rs193922878	C/C	CPIC A[20]
	Negative	RYR1 rs28933396	G/G	CPIC A[20]
	Negative	RYR1 rs28933397	C/C	CPIC A[20]
	Negative	RYR1 rs63749869	G/G	CPIC A[20]

Drug-Gene Interactions:

1 Uncertain risk of developing malignant hyperthermia (MH)

1 Results do not eliminate malignant hyperthermia susceptibility. Clinical findings, family history, further genetic testing, and other laboratory data should guide use of Sevoflurane.

Sildenafil	Phenotype	Genetic Test	Results	Source/Evidence Level
Viagra Revatio	Less likely to have positive erectile response compared to T/T	GNB3 rs5443	C/C	PharmGKB 3[48,49]

ReviewG

Drug-Gene Interactions:

2 PharmGKB – Clinical Annotation (Level 3 Efficacy): Patients with the GNB3 rs5443 C/C genotype and erectile dysfunction who are treated with sildenafil may be less likely to have positive erectile response as compared to patients with the T/T genotype. Other genetic and clinical factors may also influence a patient's response to sildenafil.

Simvastatin	Phenotype	Genetic Test	Results	Source/Evidence Level
Zocor Flolipid	Normal function	SLCO1B1	*1/*1	CPIC A[12]; FDA 2[47]

TreatG
ReviewG

Drug-Gene Interactions:

1 CPIC – Implication: Typical myopathy risk and Simvastatin exposure.

1 CPIC – Strong Recommendation: Prescribe desired starting dose and adjust doses based on disease-specific guidelines.
The potential for drug-drug interactions and dose limits based on renal and hepatic function and ancestry should be evaluated prior to initiating a statin.

Siponimod	Phenotype	Genetic Test	Results	Source/Evidence Level
Mayzent	Normal metabolizer	CYP2C9 (Star Alleles)	*1/*1	FDA 1[47]

ReviewG

Drug-Gene Interactions:

1 CYP2C9 alleles do not indicate changes from recommended dose

Succinylcholine	Phenotype	Genetic Test	Results	Source/Evidence Level
ReviewGx	Negative	CACNA1S rs1800559	C/C	CPIC A[20]
	Negative	CACNA1S rs772226819	G/G	CPIC A[20]
	Negative	RYR1 rs111888148	G/G	CPIC A[20]
	Negative	RYR1 rs112563513	G/G	CPIC A[20]
	Negative	RYR1 rs118192122	G/G	CPIC A[20]
	Negative	RYR1 rs118192124	C/C	CPIC A[20]
	Negative	RYR1 rs118192161	C/C	CPIC A[20]
	Negative	RYR1 rs118192167	A/A	CPIC A[20]
	Negative	RYR1 rs118192168	G/G	CPIC A[20]
	Negative	RYR1 rs118192170	T/T	CPIC A[20]
	Negative	RYR1 rs118192172	C/C	CPIC A[20]
	Negative	RYR1 rs118192175	C/C	CPIC A[20]
	Negative	RYR1 rs118192176	G/G	CPIC A[20]
	Negative	RYR1 rs121918593	G/G	CPIC A[20]
	Negative	RYR1 rs121918594	G/G	CPIC A[20]
	Negative	RYR1 rs121918595	C/C	CPIC A[20]
	Negative	RYR1 rs144336148	G/G	CPIC A[20]
	Negative	RYR1 rs193922747	T/T	CPIC A[20]
	Negative	RYR1 rs193922748	C/C	CPIC A[20]
	Negative	RYR1 rs193922753	G/G	CPIC A[20]
	Negative	RYR1 rs193922770	C/C	CPIC A[20]
	Negative	RYR1 rs193922802	G/G	CPIC A[20]
	Negative	RYR1 rs193922803	C/C	CPIC A[20]
	Negative	RYR1 rs193922807	G/G	CPIC A[20]
	Negative	RYR1 rs193922809	G/G	CPIC A[20]
	Negative	RYR1 rs193922816	C/C	CPIC A[20]
	Negative	RYR1 rs193922818	G/G	CPIC A[20]
	Negative	RYR1 rs193922832	G/G	CPIC A[20]
	Negative	RYR1 rs193922843	G/G	CPIC A[20]
	Negative	RYR1 rs193922876	C/C	CPIC A[20]
	Negative	RYR1 rs193922878	C/C	CPIC A[20]
	Negative	RYR1 rs28933396	G/G	CPIC A[20]
	Negative	RYR1 rs28933397	C/C	CPIC A[20]
	Negative	RYR1 rs63749869	G/G	CPIC A[20]

Drug-Gene Interactions:

1 Uncertain risk of developing malignant hyperthermia (MH)

1 Results do not eliminate malignant hyperthermia susceptibility. Clinical findings, family history, further genetic testing, and other laboratory data should guide use of Succinylcholine.

Tacrolimus	Phenotype	Genetic Test	Results	Source/Evidence Level
Advagraf Astagraf XL Envarsus XR Prograf Protopic ReviewGx	Poor metabolizer	CYP3A5	*3/*3	CPIC A[8]; FDA 1[47]
	Normal metabolizer	CYP3A4	*1/*1	PharmGKB 2A[48,49]

Drug-Gene Interactions:

1. CPIC – CYP3A5 Implication: Higher ("normal") dose-adjusted trough concentrations of tacrolimus and increased chance of achieving target tacrolimus concentrations.
   CPIC – CYP3A5 Strong Recommendation: Initiate therapy with standard recommended dose. Use therapeutic drug monitoring to guide dose adjustments. This recommendation includes the use of tacrolimus in kidney, heart, lung, and hematopoietic stem cell transplant patients, and liver transplant patients in which the donor and recipient genotypes are identical.
2. PharmGKB – CYP3A4 Clinical Annotation (Level 2A Dosage): Patients who are recipients of an organ transplant and carry two copies of the CYP3A4*1 allele may require an increased dose of tacrolimus as compared to patients with two copies of the *3 or *22 alleles or one copy of the 1* allele in combination with one copy of the *3 or *22 alleles. Other genetic and clinical factors may also influence tacrolimus dose.

Tafenoquine	Phenotype	Genetic Test	Results	Source/Evidence Level
Arakoda Krintafel ReviewGx	Normal phenotype	G6PD	B (reference)/B (reference)	CPIC A[18]

Drug-Gene Interactions:

1. CPIC – Implication: Low risk of acute hemolytic anemia.
1. CPIC – Strong Recommendation: No reason to avoid based on G6PD status
   Tafenoquine's safety has been established for a G6PD enzyme activity ≥70% of normal. (Inclusion criteria for clinical trials involving tafenoquine included G6PD activity ≥70%.).

Tamoxifen	Phenotype	Genetic Test	Results	Source/Evidence Level
Nolvadex Soltamox ReviewGx	Intermediate metabolizer (AS 1.0)	CYP2D6 (Activity Score)	*2.019/*4.001	CPIC A[19]; FDA 3[47]

Drug-Gene Interactions:

2. CPIC – Implication: Lower endoxifen concentrations compared to normal metabolizers; higher risk of breast cancer recurrence, event-free and recurrence-free survival compared to normal metabolizers.
2. CPIC – Optional Recommendation: Consider hormonal therapy such as an aromatase inhibitor for postmenopausal women or aromatase inhibitor along with ovarian function suppression in premenopausal women, given that these approaches are superior to tamoxifen regardless of CYP2D6 genotype (PMID 26211827). If aromatase inhibitor use is contraindicated, consideration should be given to use a higher but FDA approved tamoxifen dose (40 mg/day) (PMID 27226358). Avoid CYP2D6 strong to weak inhibitors.
1. FDA PGx Table Section 3 – Potential Impact on Pharmacokinetic Properties Only: Results in lower systemic active metabolite concentrations. The impact of CYP2D6 intermediate or poor metabolism on efficacy is not well established.

Tamsulosin	Phenotype	Genetic Test	Results	Source/Evidence Level
Flomax ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 3[47]

Drug-Gene Interactions:

1. CYP2D6 alleles do not indicate changes from recommended dose

Temazepam	Phenotype	Genetic Test	Results	Source/Evidence Level
Restoril TreatGx ReviewGx	Normal metabolizer	CYP2C9	*1/*1	Clinical studies[21]

Drug-Gene Interactions:

1. Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*1 homozygotes using benzodiazepines had no statistically significant increased fall risk compared to those not taking benzodiazepines (HR = 1.14; 95% CI: 0.90-1.45), whereas those carrying a CYP2C9*2 or *3 allele had a 45% increased fall risk (HR = 1.45; 95% CI: 1.21-1.73). Because the exact role of CYP2C9 in benzodiazepine metabolism is still unclear, additional research is warranted. Additionally, due to sample size, the authors were not able to perform sub-analyses of different benzodiazepine classes or investigate individual benzodiazepines in order to assess a potential class or individual drug effect.

Tenoxicam	Phenotype	Genetic Test	Results	Source/Evidence Level
Mobiflex	Normal metabolizer	CYP2C9 (Star Alleles)	*1/*1	CPIC A[46]

ReviewGx

Drug-Gene Interactions:

1 CYP2C9 alleles do not indicate changes from recommended dose

Tetrabenazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Austedo Nitoman Xenazine	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]

Drug-Gene Interactions:

1 CYP2D6 alleles do not indicate changes from recommended dose

ReviewGx

Thioguanine	Phenotype	Genetic Test	Results	Source/Evidence Level
Lanvis	Normal metabolizer	TPMT	*1/*1	CPIC A[40,45]; FDA 1[47]
ReviewGx	Normal metabolizer	NUDT15	*1/*1	CPIC A[40,45]; FDA 1[47]

Drug-Gene Interactions:

1 CPIC – TPMT Implication: Lower concentrations of thioguanine nucleotides (TGN) metabolites, but note that TGN after thioguanine are 5-10X higher than TGN after mercaptopurine or azathioprine. Normal risk of thiopurine-related leukopenia, neutropenia, myelosuppression.

1 CPIC – NUDT15 Implication: Normal risk of thiopurine-related leukopenia, neutropenia, myelosuppression.

1 CPIC – Strong Recommendation: Start with normal starting dose (e.g., 40-60 mg/m2/day) and adjust doses of thioguanine and of other myelosuppressive therapy without any special emphasis on thioguanine. Allow 2 weeks to reach steady-state after each dose adjustment.
Normal starting doses vary by race/ethnicity and treatment regimens.

Thioridazine	Phenotype	Genetic Test	Results	Source/Evidence Level
TreatGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]
ReviewGx	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 FDA PGx Table: No recommended changes or information for this CYP2D6 phenotype in the FDA PGx Table

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Thiothixene	Phenotype	Genetic Test	Results	Source/Evidence Level
TreatGx ReviewGx	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1. 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.
1. PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.
2. 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Tolterodine	Phenotype	Genetic Test	Results	Source/Evidence Level
Detrol	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 2[47]

Drug-Gene Interactions:

1. CYP2D6 alleles do not indicate changes from recommended dose

TreatGx ReviewGx

Tramadol	Phenotype	Genetic Test	Results	Source/Evidence Level
Conzip Durela Ralivia Ultram Zytram XL	Intermediate metabolizer	CYP2D6	*2.019/*4.001	CPIC A[13]; FDA 1[47]; FDA 2[47]

Drug-Gene Interactions:

1. CPIC – Implication: Reduced O-desmethyltramadol (active metabolite) formation.
1. CPIC – Optional Recommendation: Use tramadol label recommended age specific or weight-specific dosing. If no response and opioid use is warranted, consider non-codeine opioid.

TreatGx ReviewGx

Triazolam	Phenotype	Genetic Test	Results	Source/Evidence Level
Halcion	Normal metabolizer	CYP2C9	*1/*1	Clinical studies[21]

TreatGx ReviewGx

Drug-Gene Interactions:

1. Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*1 homozygotes using benzodiazepines had no statistically significant increased fall risk compared to those not taking benzodiazepines (HR = 1.14; 95% CI: 0.90-1.45), whereas those carrying a CYP2C9*2 or *3 allele had a 45% increased fall risk (HR = 1.45; 95% CI: 1.21-1.73). Because the exact role of CYP2C9 in benzodiazepine metabolism is still unclear, additional research is warranted. Additionally, due to sample size, the authors were not able to perform sub-analyses of different benzodiazepine classes or investigate individual benzodiazepines in order to assess a potential class or individual drug effect.

Trifluoperazine	Phenotype	Genetic Test	Results	Source/Evidence Level
TreatGx ReviewGx	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Trimipramine	Phenotype	Genetic Test	Results	Source/Evidence Level
Surmontil ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	CPIC B[23]; FDA 3[47]
	Rapid metabolizer	CYP2C19	*1/*17	CPIC B[23]

Drug-Gene Interactions:

2 CPIC – CYP2D6 Implication: Reduced metabolism of TCAs to less active compounds compared to normal metabolizers. Higher plasma concentrations of active drug will increase the probability of side effects.

2 CPIC – CYP2C19 Implication: Increased metabolism of tertiary amines compared to normal metabolizers. Greater conversion of tertiary amines to secondary amines may affect response or side effects.

2 CPIC – Optional Recommendation: Consider alternative drug not metabolized by CYP2C19. If use is warranted, utilize therapeutic drug monitoring to guide dose adjustment (strongly recommended for a patient with CYP2D6 ultrarapid, intermediate, or poor metabolism in combination with CYP2C19 ultrarapid, intermediate, or poor metabolism). TCAs without major CYP2C19 metabolism include the secondary amines nortriptyline and desipramine.
Recommendations above only apply to higher initial doses of TCAs for treatment of conditions such as depression. Lower dosages are often used for neuropathic pain compared to depressive disorders.
Because of the lower dosage, it is less likely that CYP2D6 intermediate metabolizers will experience adverse effects due to supratherapeutic plasma concentrations. Therefore, CPIC recommends no dose modifications for intermediate metabolizers when prescribed a lower dose for treatment of neuropathic pain, but these patients should be monitored closely for side effects. If larger doses are warranted, CPIC recommends following the gene-based guidelines presented above.
There are limited data to support dose recommendations for CYP2C19*17 carriers who are prescribed TCAs at lower doses for neuropathic pain treatment.

Valbenazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Ingrezza ReviewGx	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 1[47]

Drug-Gene Interactions:

1 CYP2D6 alleles do not indicate changes from recommended dose

Venlafaxine	Phenotype	Genetic Test	Results	Source/Evidence Level
Effexor XR	Intermediate metabolizer	CYP2D6	*2.019/*4.001	CPIC B[9]; FDA 1[47]

TreatGx
ReviewGx

Drug-Gene Interactions:

1 Decreased metabolism of venlafaxine to active metabolite O-desmethylvenlafaxine (desvenlafaxine) and decreased O-desmethylvenlafaxine: venlafaxine ratio as compared with CYP2D6 normal metabolizers. There is insufficient evidence supporting the clinical impact of the decreased O-desmethylvenlafaxine: venlafaxine ratio in CYP2D6 intermediate metabolizers.

1 CPIC: No action recommended based on genotype for venlafaxine because of minimal evidence regarding the impact on efficacy or side effects.

Viloxazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Qelbree	Intermediate metabolizer	CYP2D6	*2.019/*4.001	FDA 3[47]

ReviewGx

Drug-Gene Interactions:

1 FDA PGx Table: No recommended changes or information for this phenotype in the FDA PGx Table

Voriconazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Vfend	Rapid metabolizer	CYP2C19	*1/*17	CPIC A[32]; FDA 2[47]

ReviewGx

Drug-Gene Interactions:

3 CPIC – Implication: In patients for whom a rapid metabolizer genotype (*1/*17) is identified, the probability of attainment of therapeutic concentrations is: (a) for ADULTS, modest with standard dosing, and (b) for PEDIATRICS <18yo, variable.

3 ADULTS: CPIC – Moderate Recommendation: Choose an alternative agent that is not dependent on CYP2C19 metabolism as primary therapy in lieu of voriconazole. Such agents include isavuconazole, liposomal amphotericin B, and posaconazole. Further dose adjustments or selection of alternative therapy may be necessary due to other clinical factors, such as drug interactions, hepatic function, renal function, species, site of infection, therapeutic drug monitoring, and comorbidities.

2 PEDIATRICS <18yo: CPIC – Moderate Recommendation: Initiate therapy with recommended standard of care dosing. Use therapeutic drug monitoring (TDM) to titrate dose to therapeutic trough concentrations. Achieving voriconazole therapeutic concentrations in the pediatric population with ultrarapid and rapid metabolizer phenotypes in a timely manner is difficult. As critical time may be lost in achieving therapeutic concentrations, an alternative antifungal agent is recommended in order that the child receives effective antifungal therapy as soon as possible. Meticulous TDM is critical for rapid metabolizers. There is insufficient evidence to distinguish a CYP2C19*1/*17 and *1/*1 pediatric patient due to large variability in trough concentrations.

Vortioxetine	Phenotype	Genetic Test	Results	Source/Evidence Level
Trintellix	Intermediate metabolizer	CYP2D6	*2.019/*4.001	CPIC A[9]; FDA 1[47]

TreatGx
ReviewGx

Drug-Gene Interactions:

2 Reduced metabolism of vortioxetine to less active compounds when compared with CYP2D6 normal metabolizers. Higher plasma concentrations may increase the probability of side effects.

2 Initiate therapy with recommended starting dose (per CPIC moderate recommendation).

Warfarin	Phenotype	Genetic Test	Results	Source/Evidence Level
Coumadin Jantoven TreatGx ReviewGx	Normal metabolizer	CYP2C9	*1/*1	CPIC A[25]; FDA 1[47]
	Increased response	VKORC1 rs9923231	A/G	CPIC A[25]; FDA 1[47]
	Reduced response	CYP4F2 rs2108622	C/T	CPIC A[25]; FDA 1[47]
	Typical response	CYP2C rs12777823	G/G	CPIC A[25]

Drug-Gene Interactions:

2 CPIC – Strong Recommendation for Non-African ancestry/Moderate Recommendation for African ancestry: Calculate initial dose based on validated published pharmacogenetic algorithms, using results for VKORC1-1639G>A and CYP2C9 *2 and *3.
It is important to note that these algorithms do not include CYP4F2, CYP2C9*5, *6, *8 or *11, or rs12777823, and incorporation of these should be added when results are available.

The International Warfarin Pharmacogenetics Consortium (IWPC) dosing algorithm is available online at: https://files.cpicpgx.org/data/guideline/publication/warfarin/2011/IWPC_dose_calculator.xls

Another option http://warfarindosing.org/ contains Gage as the primary algorithm and IWPC as the secondary algorithm, and can adjust for CYP4F2, CYP2C9*5, and *6.

The two algorithms provide very similar dose recommendations. Most algorithms are developed for target INR 2-3.

The IWPC algorithm is available within the TreatGx software (see Atrial Fibrillation – Anticoagulation), accounting for all factors from the IWPC calculation (height, weight, age, VKORC1, CYP2C9*2 and *3, ethnicity/race, drug-drug interactions) along with additional optional adjustments for CYP2C9 *5, *6, *8, *11, CYP4F2 rs2108622, CYP2C rs12777823, smoking, and target INR other than 2-3.

An alternative is to use the FDA-approved Product Monograph, which provides expected maintenance dose ranges based on VKORC1 and CYP2C9 results.

CPIC – Optional Recommendation: For loading dose, a pharmacogenetics-based warfarin initiation dose algorithm could be considered. See the EU-PACT trial for pharmacogenetics-based warfarin initiation (loading) dose algorithm. https://www.nejm.org/doi/full/10.1056/NEJMoa1311386

2 CPIC – Optional Recommendation for Non-African ancestry: Carriers of CYP4F2 rs2108622 T allele: Increase dose by 5-10%.

Ziprasidone	Phenotype	Genetic Test	Results	Source/Evidence Level
Geodon Zeldox TreatGx ReviewGx	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Zuclopenthixol	Phenotype	Genetic Test	Results	Source/Evidence Level
Clopixol TreatG ReviewG	Intermediate metabolizer	CYP2D6	*2.019/*4.001	DPWG[16]
	Increased likelihood of drug toxicity, increased prolactin level, and/or increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	A/G	Clinical studies[2,6,50,51]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[34,35,41]
	Decreased likelihood of weight gain	MC4R rs489693	C/C	PharmGKB 3[48,49]

Drug-Gene Interactions:

1 3 Clinical Studies from PharmGKB Variant Annotations – HTR2C rs1414334 Allele G is associated with decreased likelihood of metabolic syndrome and/or obesity as compared to allele C. *Based on antipsychotic class and not the individual medication, effects may vary.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 C/C genotype may have decreased but not absent likelihood of weight gain when treated with antipsychotics as compared to patients with the A/A or A/C genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence weight gain. *PharmGKB clinical annotation is based on antipsychotic class and not the individual medication, effects may vary.

2 DPWG – CYP2D6 Description: The risk of side effects may be elevated. The genetic variation leads to decreased conversion of zuclopenthixol, which causes the plasma concentration to be approximately 1.35-fold higher.
DPWG – CYP2D6 Recommendation: Use 75% of the normal dose.

2 4 Clinical Studies from PharmGKB Variant Annotations – ANKK1/DRD2 rs1800497 Allele A is associated with hyperprolactinemia and/or increased likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele G, and allele G is associated with increased risk of tardive dyskinesia as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Table of Available References

Drug	Genetic Test	Sources
Abacavir	HLA-B*57:01	CPIC[30]; FDA[47]
Abrocitinib	CYP2C19	FDA[37,47]
Acetylsalicylic acid	PTGS1 rs10306114	PharmGKB[48,49]
Alfentanil	OPRM1 rs1799971	PharmGKB[48,49]
Allopurinol	HLA-B*58:01	CPIC[42]; FDA[47]
Alprazolam	CYP2C9	Clinical studies[21]
Amitriptyline	CYP2D6	CPIC[23]; FDA[47]
Amitriptyline	CYP2C19	CPIC[23]
Amoxapine	CYP2D6	FDA[47]
Amphetamine	CYP2D6	FDA[47]
Aripiprazole	CYP2D6	FDA[47]; Product monograph (actionable)[43]
Aripiprazole	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Aripiprazole	HTR2C rs1414334	Clinical studies[34,35,41]
Aripiprazole	MC4R rs489693	PharmGKB[48,49]
Aripiprazole lauroxil	CYP2D6	FDA[47]; Product monograph (actionable)[3]
Aripiprazole lauroxil	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Aripiprazole lauroxil	HTR2C rs1414334	Clinical studies[34,35,41]
Aripiprazole lauroxil	MC4R rs489693	PharmGKB[48,49]
Asenapine	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Asenapine	HTR2C rs1414334	Clinical studies[34,35,41]
Asenapine	MC4R rs489693	PharmGKB[48,49]
Atazanavir	UGT1A1	CPIC[17]
Atomoxetine	CYP2D6 (Activity Score)	CPIC[10]; FDA[47]
Atorvastatin	SLCO1B1	CPIC[12]; FDA[47]
Avatrombopag	CYP2C9	FDA[47]
Avatrombopag	Factor II rs1799963	Product monograph (actionable)[1]
Avatrombopag	Factor V rs6025	Product monograph (actionable)[1]
Azathioprine	TPMT	CPIC[40,45]; FDA[47]
Azathioprine	NUDT15	CPIC[40,45]; FDA[47]
Brexpiprazole	CYP2D6	FDA[47]; Product monograph (actionable)[4]
Brexpiprazole	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Brexpiprazole	HTR2C rs1414334	Clinical studies[34,35,41]
Brexpiprazole	MC4R rs489693	PharmGKB[48,49]
Brivaracetam	CYP2C19	FDA[47]
Bromazepam	CYP2C9	Clinical studies[21]
Bupropion	ANKK1/DRD2 rs1800497	PharmGKB[48,49]
Capecitabine	DPYD	CPIC[5]; FDA[47]
Carbamazepine	HLA-A*31:01	CPIC[39]; FDA[47]
Carbamazepine	HLA-B*15:02	CPIC[39]; FDA[47]
Cariprazine	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Cariprazine	HTR2C rs1414334	Clinical studies[34,35,41]
Cariprazine	MC4R rs489693	PharmGKB[48,49]
Carisoprodol	CYP2C19	FDA[47]

Drug	Genetic Test	Sources
Carvedilol	CYP2D6	FDA[47]
Celecoxib	CYP2C9 (Star Alleles)	CPIC[46]; FDA[47]
Cevimeline	CYP2D6	FDA[47]
Chlordiazepoxide	CYP2C9	Clinical studies[21]
Chlorpromazine	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Chlorpromazine	HTR2C rs1414334	Clinical studies[34,35,41]
Chlorpromazine	MC4R rs489693	PharmGKB[48,49]
Citalopram	CYP2C19	CPIC[9]; FDA[47]
Clobazam	CYP2C19	FDA[47]; Product monograph (actionable)[29]
Clobazam	CYP2C9	Clinical Studies[21]
Clomipramine	CYP2D6	CPIC[23]; FDA[47]
Clomipramine	CYP2C19	CPIC[23]
Clonazepam	CYP2C9	Clinical studies[21]
Clopidogrel	CYP2C19	CPIC[27]; FDA[47]
Clorazepate	CYP2C9	Clinical studies[21]
Clozapine	CYP2D6	FDA[47]
Clozapine	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Clozapine	HTR2C rs1414334	PharmGKB[48,49]
Clozapine	MC4R rs489693	PharmGKB[48,49]
Codeine	CYP2D6	CPIC[13]; FDA[47]
Cyclosporine	CYP3A5	PharmGKB[48,49]
Dapsone	G6PD	CPIC[18]
Darifenacin	CYP2D6	FDA[47]
Desflurane	CACNA1S rs1800559	CPIC[20]
Desflurane	CACNA1S rs772226819	CPIC[20]
Desflurane	RYR1 rs111888148	CPIC[20]
Desflurane	RYR1 rs112563513	CPIC[20]
Desflurane	RYR1 rs118192122	CPIC[20]
Desflurane	RYR1 rs118192124	CPIC[20]
Desflurane	RYR1 rs118192161	CPIC[20]
Desflurane	RYR1 rs118192167	CPIC[20]
Desflurane	RYR1 rs118192168	CPIC[20]
Desflurane	RYR1 rs118192170	CPIC[20]
Desflurane	RYR1 rs118192172	CPIC[20]
Desflurane	RYR1 rs118192175	CPIC[20]
Desflurane	RYR1 rs118192176	CPIC[20]
Desflurane	RYR1 rs121918593	CPIC[20]
Desflurane	RYR1 rs121918594	CPIC[20]
Desflurane	RYR1 rs121918595	CPIC[20]
Desflurane	RYR1 rs144336148	CPIC[20]
Desflurane	RYR1 rs193922747	CPIC[20]
Desflurane	RYR1 rs193922748	CPIC[20]
Desflurane	RYR1 rs193922753	CPIC[20]
Desflurane	RYR1 rs193922770	CPIC[20]
Desflurane	RYR1 rs193922802	CPIC[20]
Desflurane	RYR1 rs193922803	CPIC[20]

Drug	Genetic Test	Sources
Desflurane	RYR1 rs193922807	CPIC[20]
Desflurane	RYR1 rs193922809	CPIC[20]
Desflurane	RYR1 rs193922816	CPIC[20]
Desflurane	RYR1 rs193922818	CPIC[20]
Desflurane	RYR1 rs193922832	CPIC[20]
Desflurane	RYR1 rs193922843	CPIC[20]
Desflurane	RYR1 rs193922876	CPIC[20]
Desflurane	RYR1 rs193922878	CPIC[20]
Desflurane	RYR1 rs28933396	CPIC[20]
Desflurane	RYR1 rs28933397	CPIC[20]
Desflurane	RYR1 rs63749869	CPIC[20]
Desipramine	CYP2D6	CPIC[23]; FDA[47]
Deutetrabenazine	CYP2D6	FDA[47]
Dexlansoprazole	CYP2C19	CPIC[28]; FDA[47]
Diazepam	CYP2C19	FDA[47]
Diazepam	CYP2C9	Clinical Studies[21]
Donepezil	CYP2D6	FDA[47]
Doxepin	CYP2D6	CPIC[23]; FDA[47]
Doxepin	CYP2C19	CPIC[23]; FDA[47]
Dronabinol	CYP2C9	FDA[47]
Efavirenz	CYP2B6	CPIC[14]; FDA[47]
Elagolix	SLCO1B1	FDA[47]
Eliglustat	CYP2D6	DPWG[16]; FDA[47]
Eltrombopag	Factor V rs6025	Product monograph (actionable)[36]
Erdafitinib	CYP2C9 (Star Alleles)	FDA[47]
Escitalopram	CYP2C19	CPIC[9]; FDA[47]
Esomeprazole	CYP2C19	FDA[47]
Etanercept	TNF-alpha rs1800629	PharmGKB[48,49]
Fentanyl	OPRM1 rs1799971	PharmGKB[48,49]
Fesoterodine	CYP2D6	FDA[47]
Flecainide	CYP2D6	DPWG[16]
Flibanserin	CYP2C19	FDA[47]
Fluorouracil	DPYD	CPIC[5]; FDA[47]
Fluoxetine	CYP2D6	Product monograph (actionable)[11]
Flupentixol	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Flupentixol	HTR2C rs1414334	Clinical studies[34,35,41]
Flupentixol	MC4R rs489693	PharmGKB[48,49]
Fluphenazine	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Fluphenazine	HTR2C rs1414334	Clinical studies[34,35,41]
Fluphenazine	MC4R rs489693	PharmGKB[48,49]
Flurazepam	CYP2C9	Clinical studies[21]
Flurbiprofen	CYP2C9 (Star Alleles)	CPIC[46]; FDA[47]
Fluvastatin	CYP2C9	CPIC[12]
Fluvastatin	SLCO1B1	CPIC[12]
Fluvoxamine	CYP2D6	CPIC[9]; FDA[47]
Fosphenytoin	HLA-B*15:02	CPIC[26]; FDA[47]
Fosphenytoin	CYP2C9	CPIC[26]; FDA[47]

Drug	Genetic Test	Sources
Galantamine	CYP2D6	FDA[47]
Gefitinib	CYP2D6	FDA[47]
Haloperidol	CYP2D6	DPWG[16]
Haloperidol	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Haloperidol	HTR2C rs1414334	Clinical studies[34,35,41]
Haloperidol	MC4R rs489693	PharmGKB[48,49]
Hydrocodone	CYP2D6	CPIC[13]
Ibuprofen	CYP2C9 (Star Alleles)	CPIC[46]; FDA[47]
Iloperidone	CYP2D6	FDA[47]
Iloperidone	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Iloperidone	HTR2C rs1414334	Clinical studies[34,35,41]
Iloperidone	MC4R rs489693	PharmGKB[48,49]
Imipramine	CYP2D6	CPIC[23]; FDA[47]
Imipramine	CYP2C19	CPIC[23]
Irinotecan	UGT1A1	DPWG[16]; FDA[24,38,47]
Isoflurane	CACNA1S rs1800559	CPIC[20]
Isoflurane	CACNA1S rs772226819	CPIC[20]
Isoflurane	RYR1 rs111888148	CPIC[20]
Isoflurane	RYR1 rs112563513	CPIC[20]
Isoflurane	RYR1 rs118192122	CPIC[20]
Isoflurane	RYR1 rs118192124	CPIC[20]
Isoflurane	RYR1 rs118192161	CPIC[20]
Isoflurane	RYR1 rs118192167	CPIC[20]
Isoflurane	RYR1 rs118192168	CPIC[20]
Isoflurane	RYR1 rs118192170	CPIC[20]
Isoflurane	RYR1 rs118192172	CPIC[20]
Isoflurane	RYR1 rs118192175	CPIC[20]
Isoflurane	RYR1 rs118192176	CPIC[20]
Isoflurane	RYR1 rs121918593	CPIC[20]
Isoflurane	RYR1 rs121918594	CPIC[20]
Isoflurane	RYR1 rs121918595	CPIC[20]
Isoflurane	RYR1 rs144336148	CPIC[20]
Isoflurane	RYR1 rs193922747	CPIC[20]
Isoflurane	RYR1 rs193922748	CPIC[20]
Isoflurane	RYR1 rs193922753	CPIC[20]
Isoflurane	RYR1 rs193922770	CPIC[20]
Isoflurane	RYR1 rs193922802	CPIC[20]
Isoflurane	RYR1 rs193922803	CPIC[20]
Isoflurane	RYR1 rs193922807	CPIC[20]
Isoflurane	RYR1 rs193922809	CPIC[20]
Isoflurane	RYR1 rs193922816	CPIC[20]
Isoflurane	RYR1 rs193922818	CPIC[20]
Isoflurane	RYR1 rs193922832	CPIC[20]
Isoflurane	RYR1 rs193922843	CPIC[20]
Isoflurane	RYR1 rs193922876	CPIC[20]
Isoflurane	RYR1 rs193922878	CPIC[20]
Isoflurane	RYR1 rs28933396	CPIC[20]

Drug	Genetic Test	Sources
Isoflurane	RYR1 rs28933397	CPIC[20]
Isoflurane	RYR1 rs63749869	CPIC[20]
Lamotrigine	HLA-B*15:02	DPWG[16]
Lamotrigine	HLA-B*58:01	PharmGKB[48,49]
Lansoprazole	CYP2C19	CPIC[28]; FDA[47]
Lofexidine	CYP2D6	FDA[47]
Lorazepam	CYP2C9	Clinical studies[21]
Lovastatin	SLCO1B1	CPIC[12]
Loxapine	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Loxapine	HTR2C rs1414334	Clinical studies[34,35,41]
Loxapine	MC4R rs489693	PharmGKB[48,49]
Lurasidone	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Lurasidone	HTR2C rs1414334	Clinical studies[34,35,41]
Lurasidone	MC4R rs489693	PharmGKB[48,49]
Lusutrombopag	Factor II rs1799963	Product monograph (actionable)[44]
Lusutrombopag	Factor V rs6025	Product monograph (actionable)[44]
Mavacamten	CYP2C19	FDA[47]
Meclizine	CYP2D6	FDA[47]
Meloxicam	CYP2C9 (Star Alleles)	CPIC[46]; FDA[47]
Mercaptopurine	TPMT	CPIC[40,45]; FDA[47]
Mercaptopurine	NUDT15	CPIC[40,45]; FDA[47]
Methotrexate	MTHFR rs1801133	PharmGKB[48,49]
Methotrimeprazine	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Methotrimeprazine	HTR2C rs1414334	Clinical studies[34,35,41]
Methotrimeprazine	MC4R rs489693	PharmGKB[48,49]
Methoxyflurane	CACNA1S rs1800559	CPIC[20]
Methoxyflurane	CACNA1S rs772226819	CPIC[20]
Methoxyflurane	RYR1 rs111888148	CPIC[20]
Methoxyflurane	RYR1 rs112563513	CPIC[20]
Methoxyflurane	RYR1 rs118192122	CPIC[20]
Methoxyflurane	RYR1 rs118192124	CPIC[20]
Methoxyflurane	RYR1 rs118192161	CPIC[20]
Methoxyflurane	RYR1 rs118192167	CPIC[20]
Methoxyflurane	RYR1 rs118192168	CPIC[20]
Methoxyflurane	RYR1 rs118192170	CPIC[20]
Methoxyflurane	RYR1 rs118192172	CPIC[20]
Methoxyflurane	RYR1 rs118192175	CPIC[20]
Methoxyflurane	RYR1 rs118192176	CPIC[20]
Methoxyflurane	RYR1 rs121918593	CPIC[20]
Methoxyflurane	RYR1 rs121918594	CPIC[20]
Methoxyflurane	RYR1 rs121918595	CPIC[20]
Methoxyflurane	RYR1 rs144336148	CPIC[20]
Methoxyflurane	RYR1 rs193922747	CPIC[20]
Methoxyflurane	RYR1 rs193922748	CPIC[20]
Methoxyflurane	RYR1 rs193922753	CPIC[20]
Methoxyflurane	RYR1 rs193922770	CPIC[20]
Methoxyflurane	RYR1 rs193922802	CPIC[20]

Drug	Genetic Test	Sources
Methoxyflurane	RYR1 rs193922803	CPIC[20]
Methoxyflurane	RYR1 rs193922807	CPIC[20]
Methoxyflurane	RYR1 rs193922809	CPIC[20]
Methoxyflurane	RYR1 rs193922816	CPIC[20]
Methoxyflurane	RYR1 rs193922818	CPIC[20]
Methoxyflurane	RYR1 rs193922832	CPIC[20]
Methoxyflurane	RYR1 rs193922843	CPIC[20]
Methoxyflurane	RYR1 rs193922876	CPIC[20]
Methoxyflurane	RYR1 rs193922878	CPIC[20]
Methoxyflurane	RYR1 rs28933396	CPIC[20]
Methoxyflurane	RYR1 rs28933397	CPIC[20]
Methoxyflurane	RYR1 rs63749869	CPIC[20]
Methylene blue	G6PD	CPIC[18]
Methylphenidate	ADRA2A rs1800544	PharmGKB[48,49]
Methylphenidate	COMT rs4680	PharmGKB[48,49]
Metoclopramide	CYP2D6	FDA[47]
Metoprolol	CYP2D6	CPIC[15]
Mirabegron	CYP2D6	FDA[47]
Molindone	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Molindone	HTR2C rs1414334	Clinical studies[34,35,41]
Molindone	MC4R rs489693	PharmGKB[48,49]
Morphine	OPRM1 rs1799971	PharmGKB[48,49]
Nateglinide	CYP2C9	FDA[47]
Nebivolol	CYP2D6	FDA[47]
Nicotine replacement therapy	ANKK1/DRD2 rs1800497	PharmGKB[48,49]
Nitrazepam	CYP2C9	Clinical studies[21]
Nitrofurantoin	G6PD	CPIC[18]
Nortriptyline	CYP2D6	CPIC[23]; FDA[47]
Olanzapine	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Olanzapine	DRD2 rs1799978	PharmGKB[48,49]
Olanzapine	HTR2C rs1414334	Clinical studies[34,35,41]
Olanzapine	MC4R rs489693	PharmGKB[48,49]
Oliceridine	CYP2D6	FDA[47]
Omeprazole	CYP2C19	CPIC[28]; FDA[47]
Ondansetron	CYP2D6	CPIC[7]
Oral contraceptives	Factor II rs1799963	PharmGKB[48,49]
Oral contraceptives	Factor V rs6025	PharmGKB[48,49]
Oxazepam	CYP2C9	Clinical studies[21]
Oxcarbazepine	HLA-B*15:02	CPIC[39]; FDA[47]
Paliperidone	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Paliperidone	HTR2C rs1414334	Clinical studies[34,35,41]
Paliperidone	MC4R rs489693	PharmGKB[48,49]
Pantoprazole	CYP2C19	CPIC[28]; FDA[47]
Paroxetine	CYP2D6	CPIC[9]; FDA[47]
Pazopanib	HLA-B*57:01	FDA[47]
PEG-interferon alpha	IFNL3 rs12979860	CPIC[33]
Pegloticase	G6PD	CPIC[18]

Drug	Genetic Test	Sources
Perphenazine	CYP2D6	FDA[47]
Perphenazine	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Perphenazine	HTR2C rs1414334	Clinical studies[34,35,41]
Perphenazine	MC4R rs489693	PharmGKB[48,49]
Phenytoin	HLA-B*15:02	CPIC[26]; FDA[47]
Phenytoin	CYP2C9	CPIC[26]; FDA[47]
Pimozide	CYP2D6	FDA[47]
Pimozide	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Pimozide	HTR2C rs1414334	Clinical studies[34,35,41]
Pimozide	MC4R rs489693	PharmGKB[48,49]
Piroxicam	CYP2C9 (Star Alleles)	CPIC[46]; FDA[47]
Pitavastatin	SLCO1B1	CPIC[12]
Pitolisant	CYP2D6	FDA[47]; Product monograph (actionable)[22]
Pravastatin	SLCO1B1	CPIC[12]
Primaquine	G6PD	CPIC[18]
Prochlorperazine	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Prochlorperazine	HTR2C rs1414334	Clinical studies[34,35,41]
Prochlorperazine	MC4R rs489693	PharmGKB[48,49]
Promethazine	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Promethazine	HTR2C rs1414334	Clinical studies[34,35,41]
Promethazine	MC4R rs489693	PharmGKB[48,49]
Propafenone	CYP2D6	DPWG[16]; FDA[47]
Propranolol	CYP2D6	FDA[47]
Protriptyline	CYP2D6	FDA[47]
Quetiapine	CYP3A4	DPWG[16]
Quetiapine	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Quetiapine	HTR2C rs1414334	Clinical studies[34,35,41]
Quetiapine	MC4R rs489693	PharmGKB[48,49]
Rabeprazole	CYP2C19	FDA[47]
Rasburicase	G6PD	CPIC[18]
Risperidone	CYP2D6	DPWG[16]; FDA[47]
Risperidone	ANKK1/DRD2 rs1800497	PharmGKB[48,49]
Risperidone	DRD2 rs1799978	PharmGKB[48,49]
Risperidone	HTR2C rs1414334	PharmGKB[48,49]
Risperidone	MC4R rs489693	PharmGKB[48,49]
Rosuvastatin	SLCO1B1	CPIC[12]; FDA[47]
Rosuvastatin	ABCG2 rs2231142	CPIC[12]
Salmeterol	ADRB2 rs1042713	PharmGKB[48,49]
Sertraline	CYP2B6	CPIC[9]
Sertraline	CYP2C19	CPIC[9]
Sevoflurane	CACNA1S rs1800559	CPIC[20]
Sevoflurane	CACNA1S rs772226819	CPIC[20]
Sevoflurane	RYR1 rs111888148	CPIC[20]
Sevoflurane	RYR1 rs112563513	CPIC[20]
Sevoflurane	RYR1 rs118192122	CPIC[20]
Sevoflurane	RYR1 rs118192124	CPIC[20]

Drug	Genetic Test	Sources
Sevoflurane	RYR1 rs118192161	CPIC[20]
Sevoflurane	RYR1 rs118192167	CPIC[20]
Sevoflurane	RYR1 rs118192168	CPIC[20]
Sevoflurane	RYR1 rs118192170	CPIC[20]
Sevoflurane	RYR1 rs118192172	CPIC[20]
Sevoflurane	RYR1 rs118192175	CPIC[20]
Sevoflurane	RYR1 rs118192176	CPIC[20]
Sevoflurane	RYR1 rs121918593	CPIC[20]
Sevoflurane	RYR1 rs121918594	CPIC[20]
Sevoflurane	RYR1 rs121918595	CPIC[20]
Sevoflurane	RYR1 rs144336148	CPIC[20]
Sevoflurane	RYR1 rs193922747	CPIC[20]
Sevoflurane	RYR1 rs193922748	CPIC[20]
Sevoflurane	RYR1 rs193922753	CPIC[20]
Sevoflurane	RYR1 rs193922770	CPIC[20]
Sevoflurane	RYR1 rs193922802	CPIC[20]
Sevoflurane	RYR1 rs193922803	CPIC[20]
Sevoflurane	RYR1 rs193922807	CPIC[20]
Sevoflurane	RYR1 rs193922809	CPIC[20]
Sevoflurane	RYR1 rs193922816	CPIC[20]
Sevoflurane	RYR1 rs193922818	CPIC[20]
Sevoflurane	RYR1 rs193922832	CPIC[20]
Sevoflurane	RYR1 rs193922843	CPIC[20]
Sevoflurane	RYR1 rs193922876	CPIC[20]
Sevoflurane	RYR1 rs193922878	CPIC[20]
Sevoflurane	RYR1 rs28933396	CPIC[20]
Sevoflurane	RYR1 rs28933397	CPIC[20]
Sevoflurane	RYR1 rs63749869	CPIC[20]
Sildenafil	GNB3 rs5443	PharmGKB[48,49]
Simvastatin	SLCO1B1	CPIC[12]; FDA[47]
Siponimod	CYP2C9 (Star Alleles)	FDA[47]
Succinylcholine	CACNA1S rs1800559	CPIC[20]
Succinylcholine	CACNA1S rs772226819	CPIC[20]
Succinylcholine	RYR1 rs111888148	CPIC[20]
Succinylcholine	RYR1 rs112563513	CPIC[20]
Succinylcholine	RYR1 rs118192122	CPIC[20]
Succinylcholine	RYR1 rs118192124	CPIC[20]
Succinylcholine	RYR1 rs118192161	CPIC[20]
Succinylcholine	RYR1 rs118192167	CPIC[20]
Succinylcholine	RYR1 rs118192168	CPIC[20]
Succinylcholine	RYR1 rs118192170	CPIC[20]
Succinylcholine	RYR1 rs118192172	CPIC[20]
Succinylcholine	RYR1 rs118192175	CPIC[20]
Succinylcholine	RYR1 rs118192176	CPIC[20]
Succinylcholine	RYR1 rs121918593	CPIC[20]
Succinylcholine	RYR1 rs121918594	CPIC[20]
Succinylcholine	RYR1 rs121918595	CPIC[20]

Drug	Genetic Test	Sources
Succinylcholine	RYR1 rs144336148	CPIC[20]
Succinylcholine	RYR1 rs193922747	CPIC[20]
Succinylcholine	RYR1 rs193922748	CPIC[20]
Succinylcholine	RYR1 rs193922753	CPIC[20]
Succinylcholine	RYR1 rs193922770	CPIC[20]
Succinylcholine	RYR1 rs193922802	CPIC[20]
Succinylcholine	RYR1 rs193922803	CPIC[20]
Succinylcholine	RYR1 rs193922807	CPIC[20]
Succinylcholine	RYR1 rs193922809	CPIC[20]
Succinylcholine	RYR1 rs193922816	CPIC[20]
Succinylcholine	RYR1 rs193922818	CPIC[20]
Succinylcholine	RYR1 rs193922832	CPIC[20]
Succinylcholine	RYR1 rs193922843	CPIC[20]
Succinylcholine	RYR1 rs193922876	CPIC[20]
Succinylcholine	RYR1 rs193922878	CPIC[20]
Succinylcholine	RYR1 rs28933396	CPIC[20]
Succinylcholine	RYR1 rs28933397	CPIC[20]
Succinylcholine	RYR1 rs63749869	CPIC[20]
Tacrolimus	CYP3A5	CPIC[8]; FDA[47]
Tacrolimus	CYP3A4	PharmGKB[48,49]
Tafenoquine	G6PD	CPIC[18]
Tamoxifen	CYP2D6 (Activity Score)	CPIC[19]; FDA[47]
Tamsulosin	CYP2D6	FDA[47]
Temazepam	CYP2C9	Clinical studies[21]
Tenoxicam	CYP2C9 (Star Alleles)	CPIC[46]
Tetrabenazine	CYP2D6	FDA[47]
Thioguanine	TPMT	CPIC[40,45]; FDA[47]
Thioguanine	NUDT15	CPIC[40,45]; FDA[47]
Thioridazine	CYP2D6	FDA[47]
Thioridazine	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Thioridazine	HTR2C rs1414334	Clinical studies[34,35,41]
Thioridazine	MC4R rs489693	PharmGKB[48,49]
Thiothixene	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Thiothixene	HTR2C rs1414334	Clinical studies[34,35,41]
Thiothixene	MC4R rs489693	PharmGKB[48,49]
Tolterodine	CYP2D6	FDA[47]
Tramadol	CYP2D6	CPIC[13]; FDA[47]
Triazolam	CYP2C9	Clinical studies[21]
Trifluoperazine	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Trifluoperazine	HTR2C rs1414334	Clinical studies[34,35,41]
Trifluoperazine	MC4R rs489693	PharmGKB[48,49]
Trimipramine	CYP2D6	CPIC[23]; FDA[47]
Trimipramine	CYP2C19	CPIC[23]
Valbenazine	CYP2D6	FDA[47]
Venlafaxine	CYP2D6	CPIC[9]; FDA[47]
Viloxazine	CYP2D6	FDA[47]
Voriconazole	CYP2C19	CPIC[32]; FDA[47]

Drug	Genetic Test	Sources
Vortioxetine	CYP2D6	CPIC[9]; FDA[47]
Warfarin	CYP2C9	CPIC[25]; FDA[47]
Warfarin	VKORC1 rs9923231	CPIC[25]; FDA[47]
Warfarin	CYP4F2 rs2108622	CPIC[25]; FDA[47]
Warfarin	CYP2C rs12777823	CPIC[25]
Ziprasidone	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Ziprasidone	HTR2C rs1414334	Clinical studies[34,35,41]
Ziprasidone	MC4R rs489693	PharmGKB[48,49]
Zuclopenthixol	CYP2D6	DPWG[16]
Zuclopenthixol	ANKK1/DRD2 rs1800497	Clinical studies[2,6,50,51]
Zuclopenthixol	HTR2C rs1414334	Clinical studies[34,35,41]
Zuclopenthixol	MC4R rs489693	PharmGKB[48,49]

References

https://www.genxys.com/lab-references/snp

Laboratory Report

The **Laboratory Report** contains your genetic results.

Gene	rsID	HGVS	HGVS Reference	Result
ABCG2	rs2231142	c.421C>A	NM_004827.3	G/T
ADBR2	rs1042713	c.46G>A	NM_000024.6	G/G
ADRA2A	rs1800544	g.111076745G>C	NC_000010.11	C/C
ANKK1/DRD2	rs1800497	c.2137G>A	NM_178510.2	A/G (T/C)[1]
CACNA1S	rs1800559	c.3257G>A	NM_000069.3	C/C
CACNA1S	rs772226819	c.520C>T	NM_000069.3	G/G
COMT	rs4680	c.472G>A	NM_000754.4	G/G
CYP2B6	rs3745274	c.516G>A/T	NM_000767.5	T/G
CYP2B6	rs2279343	c.785A>G	NM_000767.5	A/G
CYP2B6	rs28399499	c.983T>C	NM_000767.5	T/T
CYP2B6	rs3211371	c.1459C>T	NM_000767.5	C/C
CYP2B6	rs34223104	g.4926T>C	NG_007929.1	T/T
CYP2B6	rs36056539	c.503C>T	NM_000767.5	C/C
CYP2B6	rs34826503	c.1006C>T	NM_000767.5	C/C
CYP2C18	rs12777823	g.94645745G>A	NC_000010.11	G/G
CYP2C19	rs12248560	c.-806C>T	NM_000769.2	C/T
CYP2C19	rs28399504	c.1A>G	NM_000769.1	A/A
CYP2C19	rs41291556	c.358T>C	NM_000769.1	T/T
CYP2C19	rs4244285	c.681G>A	NM_000769.1	G/G
CYP2C19	rs4986893	c.636G>A	NM_000769.1	G/G
CYP2C19	rs72552267	c.395G>A	NM_000769.1	G/G
CYP2C19	rs17884712	c.431G>A	NM_000769.1	G/G
CYP2C19	rs6413438	c.680C>T	NM_000769.1	C/C
CYP2C19	rs72558186	g.19294T>A	NM_000769.1	T/T
CYP2C19	rs17879685	c.1228C>T	NM_000769.1	C/C
CYP2C19	rs56337013	c.1297C>T	NM_000769.4	C/C
CYP2C19	rs140278421	c.557G>C	NM_000769.4	G/G
CYP2C19	rs192154563	c.1324C>T	NM_000769.4	C/C
CYP2C19	rs113934938	c.1120G>A	NM_000769.4	G/G
CYP2C19	rs118203759	c.1344C>G	NM_000769.4	C/C
CYP2C19	rs138142612	c.986G>A	NM_000769.4	G/G
CYP2C19	rs12769205	c.332-23A>G	NM_000769.4	A/A
CYP2C19	rs118203757	c.1004G>A	NM_000769.4	G/G
CYP2C19	rs17882687	c.55A>C	NM_000769.4	A/A
CYP2C19	rs375781227	c.766G>A	NM_000769.4	G/G
CYP2C19	rs58973490	c.449G>A	NM_000769.4	G/G
CYP2C9	rs1057910	c.1075A>C	NM_000771.3	A/A
CYP2C9	rs1799853	c.430C>T	NM_000771.3	C/C
CYP2C9	rs28371685	c.1003C>T	NM_000771.3	C/C
CYP2C9	rs28371686	c.1080C>G	NM_000771.3	C/C
CYP2C9	rs9332131	c.817delA	NM_000771.3	A/A
CYP2C9	rs56165452	c.1076T>C	NM_000771.3	T/T
CYP2C9	rs7900194	c.449G>A/C/T	NM_000771.4	G/G
CYP2C9	rs72558190	c.485C>A	NM_000771.3	C/C
CYP2C9	rs2256871	c.752A>G	NM_000771.3	A/A
CYP2C9	rs72558193	c.1190A>C	NM_000771.3	A/A
CYP2C9	rs72558187	c.269T>C	NM_000771.4	T/T
CYP2C9	rs9332239	c.1465C>T	NM_000771.4	C/C
CYP2C9	rs57505750	c.980T>C	NM_000771.4	T/T
CYP2C9	rs72558189	c.374G>A	NM_000771.4	G/G

Gene	rsID	HGVS	HGVS Reference	Result
CYP2D6	rs1065852	c.100C>T	NM_000106.5	A/G
CYP2D6	rs16947	c.886C>T	NM_000106.5	A/G
CYP2D6	rs28371706	c.320C>A	NM_000106.5	G/G
CYP2D6	rs28371725	c.985+39G>A	NM_000106.5	C/C
CYP2D6	rs3892097	c.506-1G>A	NM_000106.5	T/C
CYP2D6	rs5030655	c.454delT	NM_000106.5	A/A
CYP2D6	rs5030867	c.971A>C	NM_000106.5	T/T
CYP2D6	rs59421388	c.1012G>A	NM_000106.5	C/C
CYP2D6	rs5030865	c.505G>T,G>A	NM_000106.6	C/C
CYP2D6	rs769258	c.31G>A	NM_000106.6	C/C
CYP2D6	rs1080985	g.3436C>G	NG_008376.4	G/G
CYP2D6	rs1135840	c.1304G>C	NM_001025161.2	G/G
CYP2D6	rs28371710	c.463G>A	NM_000106.5	C/C
CYP2D6	rs267608297	c.782C>T	NM_000106.6	G/G
CYP2D6	rs28371717	c.709G>T	NM_000106.6	C/C
CYP2D6	rs267608289	c.352+7A>G	NM_000106.6	T/T
CYP2D6	rs72549356	c.514TTTCGCCCC[3]	NM_000106.6	-/-
CYP2D6	rs730882251	c.1321C>T	NM_000106.6	G/G
CYP2D6	rs267608319	c.1319G>A	NM_000106.6	C/C
CYP2D6	rs72549347	c.1030C>T	NM_000106.6	G/G
CYP2D6	rs61736512	c.406G>A	NM_000106.6	C/C
CYP2D6	rs267608279	c.864del	NM_000106.6	G/G
CYP2D6	rs72549351	c.815_818del	NM_000106.6	TCAG/TCAG
CYP2D6	rs267608311	c.184C>T	NM_000106.6	G/G
CYP2D6	rs72549354	c.635dup	NM_000106.6	-/-
CYP2D6	rs267608302	c.467A>C	NM_000106.6	T/T
CYP2D6	rs1135822	c.358T>A	NM_000106.6	A/A
CYP2D6	rs148769737	c.800C>A	NM_000106.6	G/G
CYP2D6	rs1058164	c.408G>C	NM_000106.6	G/G
CYP2D6	rs1135823	c.364G>T	NM_000106.6	C/C
CYP2D6	rs72549348	c.1001A>C	NM_000106.6	T/T
CYP3A4	rs35599367	c.522-191G>A	NC_000007.14	G/G
CYP3A4	rs55785340	c.664T>C	NM_017460.6	A/A
CYP3A4	rs4986910	c.1334T>C	NM_017460.6	A/A
CYP3A4	rs12721629	c.1117C>T	NM_017460.6	G/G
CYP3A4	rs4987161	c.566T>C	NM_017460.6	A/A
CYP3A5	rs10264272	c.624G>A	NM_000777.4	C/C
CYP3A5	rs776746	c.219-237G>A	NM_000777.4	C/C
CYP4F2	rs2108622	c.1297G>A	NM_001082.5	T/C
DPYD	rs1801268	c.2983G>T	NM_000110.3	C/C
DPYD	rs1801267	c.2657G>A	NM_000110.4	C/C
DPYD	rs80081766	c.62G>A	NM_000110.4	C/C
DPYD	rs67376798	c.2846A>T	NM_000110.4	T/T
DPYD	rs72549310	c.61C>T	NM_000110.4	G/G
DPYD	rs75017182	c.1129-5923C>G	NM_000110.4	G/G
DPYD	rs78060119	c.1156G>T	NM_000110.4	C/C
DPYD	rs1801158	c.1601G>A	NM_000110.4	C/C
DPYD	rs1801265	c.85T>C	NM_000110.4	A/G
DPYD	rs2297595	c.496A>G	NM_000110.4	T/C
DPYD	rs1801159	c.1627A>G	NM_000110.4	T/T
DPYD	rs3918290	g.97450058C>T	NC_000001.11	C/C
DPYD	rs17376848	c.1896T>C	NM_000110.4	A/A
DPYD	rs1801160	c.2194G>A	NM_000110.4	C/C
DPYD	rs56038477	c.1236G>A	NM_000110.4	C/C

Gene	rsID	HGVS	HGVS Reference	Result
DPYD	rs115232898	c.557A>G	NM_000110.4	T/T
DPYD	rs1801266	c.703C>T	NM_000110.4	G/G
DPYD	rs72549303	c.1898del	NM_000110.4	G/G
DPYD	rs6670886	c.525G>A	NM_000110.4	C/C
DPYD	rs72549306	c.1003G>T	NM_000110.4	C/C
DRD2	rs1799978	g.4651A>G	NG_008841.1	T/T
Factor II	rs1799963	c.*97G>A	NM_000506.4	G/G
Factor V	rs6025	c.1601G>A	NM_000130.4	C/C (G/G)[1]
FKBP5	rs4713916	c.-20+18122T>C	NM_001145775.1	G/G
G6PD	rs1050828	c.292G>A	NM_000402.4	C/C
G6PD	rs2230037	c.1401T>C	NM_000402.4	G/G
G6PD	rs78478128	c.221C>G	NM_000402.4	G/G
G6PD	rs782308266	c.332G>A	NM_000402.4	C/C
G6PD	rs137852337	c.1406G>C	NM_000402.4	C/C
G6PD	rs34193178	c.1138G>C	NM_000402.4	C/C
G6PD	rs137852342	c.1114C>T	NM_000402.4	G/G
G6PD	rs782754619	c.724A>G	NM_000402.4	T/T
G6PD	rs137852346	c.896G>A	NM_000402.4	C/C
G6PD	rs137852326	c.727G>T	NM_000402.4	C/C
G6PD	rs137852335	c.1270G>C	NM_000402.4	C/C
G6PD	rs74575103	c.944G>A	NM_000402.4	C/C
G6PD	rs267606836	c.634C>T	NM_000402.4	G/G
G6PD	rs782090947	c.299A>G	NM_000402.4	T/T
G6PD	rs587776730	c.1047_1070del	NM_000402.4	GTGGGGTCGTCCAGGTACCCTTTG/GTGGGGTCGTCCAGGTACCCTTTG
G6PD	rs137852343	c.607T>C	NM_000402.4	A/A
G6PD	rs782757170	c.793C>T	NM_000402.4	G/G
G6PD	rs398123544	c.1127A>T	NM_000402.4	T/T
G6PD	rs782487723	c.563G>A	NM_000402.4	C/C
G6PD	rs371489738	c.1222G>A	NM_000402.4	C/C
G6PD	rs137852347	c.1054T>C	NM_000402.4	A/A
G6PD	rs137852344	c.1490C>G	NM_000402.4	G/G
G6PD	rs5030872	c.632A>T	NM_000402.4	T/T
G6PD	rs78365220	c.473T>C	NM_000402.4	A/A
G6PD	rs782098548	c.1381G>A	NM_000402.4	C/C
G6PD	rs72554665	c.1466G>A	NM_000402.4	C/C
G6PD	rs1050829	c.466A>G	NM_000402.4	T/T
G6PD	rs137852338	c.192CAT[2]	NM_000402.4	ATG/ATG
G6PD	rs5030868	c.653C>T	NM_000402.4	G/G
G6PD	rs5030869	c.1093G>A	NM_000402.4	C/C
G6PD	rs5030870	c.427G>A	NM_000402.4	C/C
G6PD	rs76723693	c.1058T>C	NM_000402.4	A/A
G6PD	rs137852349	c.298T>C	NM_000402.4	A/A
G6PD	rs137852327	c.961G>A	NM_000402.4	C/C
G6PD	rs137852341	c.482G>T	NM_000402.4	C/C
G6PD	rs137852340	c.185A>G	NM_000402.4	T/T
G6PD	rs72554664	c.1478G>A	NM_000402.4	C/C
G6PD	rs138687036	c.331C>T	NM_000402.4	G/G
G6PD	rs137852313	c.556G>A	NM_000402.4	C/C
G6PD	rs398123546	c.1450C>T	NM_000402.4	G/G
G6PD	rs137852339	c.1039G>A	NM_000402.4	C/C
G6PD	rs137852331	c.583A>G	NM_000402.4	T/T
G6PD	rs76645461	c.233T>C	NM_000402.4	A/A
G6PD	rs137852319	c.738T>G	NM_000402.4	A/A
G6PD	rs137852348	c.1532C>G	NM_000402.4	G/G

Gene	rsID	HGVS	HGVS Reference	Result
G6PD	rs137852320	c.1246A>G	NM_000402.4	T/T
G6PD	rs137852316	c.1268G>A	NM_000402.4	C/C
G6PD	rs137852317	c.1429G>A	NM_000402.4	C/C
G6PD	rs137852329	c.1179C>A	NM_000402.4	G/G
G6PD	rs137852314	c.577G>A	NM_000402.4	C/C
G6PD	rs137852333	c.1147C>T	NM_000402.4	G/G
G6PD	rs137852334	c.1249C>T	NM_000402.4	G/G
G6PD	rs137852323	c.1318G>T	NM_000402.4	C/C
G6PD	rs137852321	c.1250G>A	NM_000402.4	C/C
G6PD	rs137852322	c.1243T>C	NM_000402.4	A/A
G6PD	rs137852315	c.262G>A	NM_000402.4	C/C
G6PD	rs137852345	c.1172C>T	NM_000402.4	G/G
G6PD	rs137852336	c.1319G>A	NM_000402.4	C/C
G6PD	rs137852330	c.682C>T	NM_000402.4	G/G
G6PD	rs137852325	c.1282G>A	NM_000402.4	C/C
G6PD	rs137852324	c.1451G>A	NM_000402.4	C/C
G6PD	rs267606835	c.407C>G	NM_000402.4	G/G
GNB3	rs5443	g.6845711C>T	NC_000012.12	C/C
GRIK4	rs1954787	c.83-10039T>C	NM_014619.2	T/T
HCP5	rs2395029	c.*568T>G	NM_006674.3	T/T
HLAA	rs1061235	c.*66A>T	NM_002116.8	A/A
HLAB	rs144012689	c.1012+104A>T	NM_005514.8	T/T
HTR2A	rs7997012	c.614-2211T>C	NM_000621.5	A/A
HTR2C	rs1414334	c.551-3008C>G	NM_001256760.3	G/G
IFNL4	rs12979860	c.151-152G>A	NM_001276254.2	T/T
MTHFR	rs1801133	c.665C>T	NM_005957.5	A/G (C/T)[1]
MC4R	rs489693	g.57882787C>A	NC_000018.9	C/C
NUDT15	rs766023281	c.101G>C	NM_018283.1	G/G
NUDT15	rs746071566	g.48611919_48611937=	NC_000013.10	-/-
NUDT15	rs147390019	c.416G>A	NM_018283.1	G/G
NUDT15	rs186364861	c.52G>A	NM_018283.1	G/G
NUDT15	rs116855232	c.415C>T	NM_018283.1	C/C
OPRM1	rs1799971	c.118A>G	NM_000914.5	A/A
PTGS1	rs10306114	g.111076745A>G	NC_000009.12	A/A
RYR1	rs118192124	c.7354C>T	NM_000540.3	C/C
RYR1	rs118192122	c.7361G>A	NM_000540.3	G/G
RYR1	rs28933397	c.7372C>T	NM_000540.3	C/C
RYR1	rs118192176	c.6502G>A	NM_000540.3	G/G
RYR1	rs121918594	c.7373G>A	NM_000540.3	G/G
RYR1	rs193922772	c.1841G>T	NM_000540.3	G/G
RYR1	rs193922843	c.11969G>T	NM_000540.3	G/G
RYR1	rs112563513	c.7007G>A	NM_000540.3	G/G
RYR1	rs193922748	c.130C>T	NM_000540.3	C/C
RYR1	rs193922809	c.7282G>A	NM_000540.3	G/G
RYR1	rs193922878	c.14512C>G	NM_000540.3	C/C
RYR1	rs193922807	c.7124G>C	NM_000540.3	G/G
RYR1	rs193922747	c.103T>C	NM_000540.3	T/T
RYR1	rs193922876	c.14497C>T	NM_000540.3	C/C
RYR1	rs193922816	c.7360C>T	NM_000540.3	C/C
RYR1	rs193922753	c.488G>T	NM_000540.3	G/G
RYR1	rs111888148	c.1589G>A	NM_000540.3	G/G
RYR1	rs193922770	c.1654C>T	NM_000540.3	C/C
RYR1	rs144336148	c.1598G>A	NM_000540.3	G/G
RYR1	rs118192172	c.1840C>T	NM_000540.3	C/C

Gene	rsID	HGVS	HGVS Reference	Result
RYR1	rs193922802	c.7048G>A	NM_000540.3	G/G
RYR1	rs63749869	c.14582G>A	NM_000540.3	G/G
RYR1	rs118192167	c.14387A>G	NM_000540.3	A/A
RYR1	rs118192168	c.14545G>A	NM_000540.3	G/G
RYR1	rs118192170	c.14693T>C	NM_000540.3	T/T
RYR1	rs193922818	c.7523G>A	NM_000540.3	G/G
RYR1	rs118192161	c.487C>A	NM_000540.3	C/C
RYR1	rs121918593	c.7300G>A	NM_000540.3	G/G
RYR1	rs193922803	c.7063C>T	NM_000540.3	C/C
RYR1	rs193922832	c.9310G>A	NM_000540.3	G/G
RYR1	rs121918595	c.14477C>T	NM_000540.3	C/C
RYR1	rs118192175	c.6487C>T	NM_000540.3	C/C
RYR1	rs28933396	c.7304G>T	NM_000540.3	G/G
SLCO1B1	rs4149056	c.521T>C	NM_006446.4	T/T
SLCO1B1	rs11045819	c.463C>T	NM_006446.5	C/C
SLCO1B1	rs72559747	c.1007C>G	NM_006446.5	C/C
SLCO1B1	rs2306283	c.388A>C/G/T	NM_006446.5	A/A
TNF	rs1800629	g.31575254G>A	NC_000006.12	G/G
TPMT	rs1142345	c.719A>G/C	NM_000367.3	T/T
TPMT	rs1800460	c.460G>A	NM_000367.3	C/C
TPMT	rs1800462	c.238G>C	NM_000367.3	C/C
TPMT	rs72552738	c.395G>A	NM_000367.3	C/C
TPMT	rs9333569	c.1A>G	NM_000367.2	T/T
TPMT	rs9333570	g.26254G>A	NG_012137.3	C/C
TPMT	rs74423290	c.500C>G	NM_000367.3	G/G
TPMT	rs267607275	c.2T>C	NM_000367.3	A/A
TPMT	rs56161402	c.644G>A	NM_000367.3	C/C
TPMT	rs1800584	g.29363G>A	NG_012137.3	C/C
UGT1A1	rs35350960	g.233760973C>A/T	NC_000002.12	C/C
UGT1A1	rs4148323	g.233760498G>A	NC_000002.12	G/G
VKORC1	rs9923231	g.31096368C>T	NC_000016.10	A/G (T/C)[1]

1: Pharmacogenetic testing may occasionally lead to unusual genotypes. In these situations, pharmacogenetic laboratories will sometimes report on alternative genotypes. If this is done, then both genotypes appear in the result table; a genotype in () is the alternative genotype chosen by the lab.

HLA

Gene	Allele	Result
HLA-A	*31:01	Positive
HLA-B	*15:02	Negative
HLA-B	*58:01	Positive
HLA-B	*57:01	Negative

Copy Number Variation

Gene	Reference	Result (Copy/Copies)
CYP2D6	NG_008376.3 exon 9	2
CYP2D6_3pFlank	NG_008376.3 CYP2D6_3pFlank	2
CYP2D6_5pFlank	NG_008376.3 CYP2D6_5pFlank	2

Phenotype Table

Gene	Genotype Result	Phenotype Result OR Genotype Explanation
CYP2B6	*1/*6	Intermediate Metabolizer
CYP2D6	*2.019/*4.001	Intermediate Metabolizer
CYP2C9	*1/*1	Normal Metabolizer
CYP2C19	*1/*17	Rapid Metabolizer
CYP3A4	*1/*1	Normal Metabolizer
CYP3A5	*3/*3	Poor Metabolizer

Gene	Genotype Result	Phenotype Result OR Genotype Explanation
DPYD	*1/*1	Normal Metabolizer
G6PD	B (reference)/B (reference)	Normal
NUDT15	*1/*1	Normal Metabolizer
SLCO1B1	*1/*1	Normal Function
TPMT	*1/*1	Normal Metabolizer
UGT1A1	*1/*1	Normal Metabolizer

Limitations

The annotations and interpretations provided in this report are based on scientific literature and do not take into account drug-drug interactions, medical conditions or other clinical factors that may affect medication response. Gene-drug interactions are ranked according to guidelines, level of evidence and clinical utility. GenXys reports and TreatGx Clinical Decision Support are regularly updated. Current predicted phenotype and allele functionality may change in the future depending on new evidence. Phenotype annotations for CYP2C9 are based on total activity scores as defined by CPIC[79]. Genetic test results and interpretation may be inaccurate for individuals who have undergone or are receiving non-autologous blood transfusion, tissue, or organ transplant therapies.

The report includes alleles of proteins involved in the metabolism of many medications. In rare cases, a variant that is not covered may be typed as *1 or other variants. In the case of pseudogenes and mutations in the untranslated regions of genes, incorrect allele typing may occur despite proper SNP detection. Preferential amplification of one allele over another present in the sample may also lead to incorrect genotyping.

Liability Disclaimer

This test was developed and its performance characteristics determined by GenXys Health Care Systems. It has not been cleared or approved by the US Food and Drug Administration. The report is not a diagnostic test, and TreatGx is not a prescribing system. You should discuss your pharmacogenetic information with a physician or other health care provider before you act upon the pharmacogenetic information resulting from this report. The medication brand names are not an exhaustive list and do not include combination therapies. Not all medications in this report are included in the TreatGx or ReviewGx software or other GenXys derivative works.

Laboratory Director

Dr Danny Meyersfeld, Laboratory Director, PhD

05/May/2025 (UTC)

Date of Signature