DNALYSIS Biotechnology

PATIENT INFORMATION

NAME: Jane Doe
DOB: 28/Feb/1972
SEX AT BIRTH: Female

SPECIMEN DETAILS

BARCODE: DNA012345ZA
SAMPLE ID: TESTSAMPLE
TYPE: DBS
COLLECTED: 27/Aug/2026

ORDERED BY

Private Practitioner
GENERATED: 27/Aug/2026

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

No current medications.

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.

✓ **Indicates current medications**

	1 Mild or no drug-gene interaction: no PGx-based action; standard precautions apply	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
Addiction	Buprenorphine Bupropion Lofexidine Methadone			Morphine	Nicotine replacement therapy		
Analgesia (Pain)/ Anesthesiology/ Anti-inflammatory	Amitriptyline Buprenorphine Carisoprodol Celecoxib Codeine Desflurane Desipramine Elagolix Flurbiprofen Halothane Hydrocodone Ibuprofen Imipramine Isoflurane Lamotrigine Meloxicam Methadone Methoxyflurane Nortriptyline Oliceridine Oxcarbazepine Oxycodone Piroxicam Sevoflurane Succinylcholine Tenoxicam Tramadol			Alfentanil Fentanyl Morphine	Fentanyl		Carbamazepine

	1 Mild or no drug-gene interaction: no PGx-based action; standard precautions apply	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						
Cardiovascular Disease	Carvedilol Flecainide Lovastatin Mavacamten Metoprolol Nebivolol Pitavastatin Pravastatin Propafenone Propranolol Rosuvastatin Simvastatin	Fluvastatin		Fluvastatin Hydralazine	Atorvastatin	Fluvastatin Hydralazine Procainamide	
Dermatology	Abrocitinib Dapsone Deuruxolitinib Doxepin Fluorouracil		Tacrolimus	Cyclosporine		Methotrexate	
Endocrinology/ Metabolism/ Bone	Ascorbic acid Eliglustat Glimepiride Glipizide Glyburide Nateglinide						
Gastroenterology	Azathioprine Esomeprazole Meclizine Mercaptopurine Metoclopramide Ondansetron Rabeprazole Seladelpar		Dexlansoprazole Lansoprazole Omeprazole Pantoprazole	Dronabinol	Dexlansoprazole Lansoprazole Omeprazole Pantoprazole	Chlorpromazine Dronabinol Methotrexate Perphenazine Prochlorperazine Promethazine Sulfasalazine	
Gynecology	Elagolix Flibanserin Oral contraceptives						
Hematology/ Coagulation							

	1 Mild or no drug-gene interaction: no PGx-based action; standard precautions apply	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 Avatrombopag Clopidogrel Eltrombopag Lusutrombopag Methylene blue		Warfarin	Warfarin	Warfarin	Warfarin	
Immunomodulators	Abrocitinib Azathioprine Deuruxolitinib Etanercept Hydroxychloroquine Mercaptopurine PEG-interferon alpha Thioguanine		Tacrolimus	Amifampridine Cyclosporine		Amifampridine Methotrexate Sulfasalazine	
Infectious Disease (non viral)	Chloroquine Dapsone Hydroxychloroquine Nitrofurantoin Primaquine Quinine Tafenoquine Voriconazole					Isoniazid Sulfamethoxazole	
Infectious Disease (viral)	Atazanavir Dolutegravir Nevirapine PEG-interferon alpha			Efavirenz		Efavirenz	Abacavir
Neurology	Aducanumab Amitriptyline Brivaracetam Deutetrabenazine Dextromethorphan/ Quinidine Donanemab Donepezil Fosphenytoin Galantamine Lamotrigine			Siponimod		Clobazam Clonazepam Clorazepate Diazepam Lorazepam	Carbamazepine

	1 Mild or no drug-gene interaction: no PGx-based action; standard precautions apply	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
	Lecanemab Metoprolol Oxcarbazepine Phenytoin Pitolisant Propranolol Tetrabenazine Valbenazine Venlafaxine						
Oncology	Capecitabine Dabrafenib Erdafitinib Fluorouracil Flutamide Gefitinib Irinotecan Mercaptopurine PEG-interferon alpha Rasburicase Tamoxifen Thioguanine		Allopurinol		Allopurinol	Daunorubicin Doxorubicin Methotrexate Pazopanib	
Psychiatry	Amitriptyline Amoxapine Amphetamine Bupropion Citalopram Clomipramine Desipramine Dextromethorphan/ Bupropion Doxepin Escitalopram Fluoxetine Fluvoxamine Imipramine Lamotrigine Mirtazapine Nortriptyline Paroxetine Protriptyline Trimipramine		Atomoxetine	Sertraline	Atomoxetine Methylphenidate Olanzapine Risperidone	Alprazolam Aripiprazole Aripiprazole lauroxil Asenapine Brexpiprazole Bromazepam Cariprazine Chlordiazepoxide Chlorpromazine Clonazepam Clorazepate Clozapine Diazepam Flupentixol Fluphenazine Flurazepam Haloperidol Iloperidone Lorazepam	Carbamazepine

	1 Mild or no drug-gene interaction: no PGx-based action; standard precautions apply	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 Viloxazine Vortioxetine					Loxapine Lurasidone Methotrimeprazine Molindone Nitrazepam Olanzapine Oxazepam Paliperidone Perphenazine Pimozide Prochlorperazine Quetiapine Temazepam Thioridazine Thiothixene Triazolam Trifluoperazine Ziprasidone Zuclopenthixol	
Pulmonary	Salmeterol				Sildenafil		
Rheumatology	Azathioprine Celecoxib Cevimeline Etanercept Flurbiprofen Hydroxychloroquine Ibuprofen Meloxicam Pegloticase Piroxicam Tenoxicam		Allopurinol	Cyclosporine	Allopurinol	Methotrexate	
Urologic	Darifenacin Fesoterodine Mirabegron Tamsulosin Tolterodine				Sildenafil		

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 drug-gene interaction: no PGx-based action; standard precautions apply

2 Moderate drug-gene interaction

3 Serious drug-gene interaction: avoid/select alternative

Addiction

1
Buprenorphine
Bupropion
Lofexidine
Methadone

2
Morphine
Nicotine replacement therapy

Analgesia (Pain)/ Anesthesiology/ Anti-inflammatory

1
Amitriptyline
Buprenorphine
Carisoprodol
Celecoxib
Codeine
Desflurane
Desipramine
Elagolix
Flurbiprofen
Halothane
Hydrocodone
Ibuprofen
Imipramine
Isoflurane
Lamotrigine
Meloxicam
Methadone

...Analgesia (Pain)/ Anesthesiology/ Anti-inflammatory

1
Methoxyflurane
Nortriptyline
Oliceridine
Oxcarbazepine
Oxycodone
Piroxicam
Sevoflurane
Succinylcholine
Tenoxicam
Tramadol
Venlafaxine

2
Alfentanil
Fentanyl
Morphine

3
Carbamazepine

Cardiovascular Disease

1
Carvedilol
Flecainide
Lovastatin
Mavacamten
Metoprolol
Nebivolol
Pitavastatin
Pravastatin

...Cardiovascular Disease

1
Propafenone
Propranolol
Rosuvastatin
Simvastatin

2
Atorvastatin
Fluvastatin
Hydralazine
Procainamide

Dermatology

1
Abrocitinib
Dapsone
Deuruxolitinib
Doxepin
Fluorouracil

2
Cyclosporine
Methotrexate
Tacrolimus

Endocrinology/ Metabolism/ Bone

1
Ascorbic acid
Eliglustat
Glimepiride
Glipizide
Glyburide

...Endocrinology/ Metabolism/ Bone

1
Nateglinide

Gastroenterology

1
Azathioprine
Esomeprazole
Meclizine
Mercaptopurine
Metoclopramide
Ondansetron
Rabeprazole
Seladelpar

2
Chlorpromazine
Dexlansoprazole
Dronabinol
Lansoprazole
Methotrexate
Omeprazole
Pantoprazole
Perphenazine
Prochlorperazine
Promethazine
Sulfasalazine

Gynecology

1
Elagolix
Flibanserin

...Gynecology

1
Oral contraceptives

Hematology/ Coagulation

1
Acetylsalicylic acid
Avatrombopag
Clopidogrel
Eltrombopag
Lusutrombopag
Methylene blue

2
Warfarin

Immunomodulators

1
Abrocitinib
Azathioprine
Deuruxolitinib
Etanercept
Hydroxychloroquine
Mercaptopurine
PEG-interferon alpha
Thioguanine

2
Amifampridine
Cyclosporine
Methotrexate
Sulfasalazine
Tacrolimus

Infectious Disease (non viral)

1
Chloroquine
Dapsone
Hydroxychloroquine
Nitrofurantoin

...Infectious Disease (non viral)

1
Primaquine
Quinine
Tafenoquine
Voriconazole

2
Isoniazid
Sulfamethoxazole

Infectious Disease (viral)

1
Atazanavir
Dolutegravir
Nevirapine
PEG-interferon alpha

2
Efavirenz

3
Abacavir

Neurology

1
Aducanumab
Amitriptyline
Brivaracetam
Deutetrabenazine
Dextromethorphan/ Quinidine
Donanemab
Donepezil
Fosphenytoin
Galantamine
Lamotrigine
Lecanemab
Metoprolol
Oxcarbazepine
Phenytoin

...Neurology

1
Pitolisant
Propranolol
Tetrabenazine
Valbenazine
Venlafaxine

2
Clobazam
Clonazepam
Clorazepate
Diazepam
Lorazepam
Siponimod

3
Carbamazepine

Oncology

1
Capecitabine
Dabrafenib
Erdafitinib
Fluorouracil
Flutamide
Gefitinib
Irinotecan
Mercaptopurine
PEG-interferon alpha
Rasburicase
Tamoxifen
Thioguanine

2
Allopurinol
Daunorubicin
Doxorubicin
Methotrexate
Pazopanib

Psychiatry

1
Amitriptyline
Amoxapine
Amphetamine
Bupropion
Citalopram
Clomipramine
Desipramine
Dextromethorphan/ Bupropion
Doxepin
Escitalopram
Fluoxetine
Fluvoxamine
Imipramine
Lamotrigine
Mirtazapine
Nortriptyline
Paroxetine
Protriptyline
Trimipramine
Venlafaxine
Viloxazine
Vortioxetine

2
Alprazolam
Aripiprazole
Aripiprazole lauroxil
Asenapine
Atomoxetine
Brexpiprazole
Bromazepam
Cariprazine
Chlordiazepoxide
Chlorpromazine

...Psychiatry

2

Clonazepam
Clorazepate
Clozapine
Diazepam
Flupentixol
Fluphenazine
Flurazepam
Haloperidol
Iloperidone
Lorazepam
Loxapine
Lurasidone
Methotrimeprazine
Methylphenidate
Molindone
Nitrazepam
Olanzapine
Oxazepam
Paliperidone
Perphenazine
Pimozide
Prochlorperazine
Quetiapine
Risperidone
Sertraline
Temazepam
Thioridazine
Thiothixene
Triazolam
Trifluoperazine
Ziprasidone
Zuclopenthixol

3

Carbamazepine

Pulmonary

1

Salmeterol

2

Sildenafil

Rheumatology

1

Azathioprine
Celecoxib
Cevimeline
Etanercept
Flurbiprofen
Hydroxychloroquine
Ibuprofen
Meloxicam
Pegloticase
Piroxicam
Tenoxicam

2

Allopurinol
Cyclosporine
Methotrexate

Urologic

1

Darifenacin
Fesoterodine
Mirabegron
Tamsulosin
Tolterodine

2

Sildenafil

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 →

Codeine	Phenotype	Genetic Test	Results	Source/Evidence Level
	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	Increased risk of adverse drug reactions	HLA-B*57:01	Positive	CPIC A[35]; FDA 1[52]

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

3 CPIC – Implication: Significantly increased risk of abacavir hypersensitivity.

3 CPIC – Strong Recommendation: Abacavir is not recommended.

Abrocitinib	Phenotype	Genetic Test	Results	Source/Evidence Level
Cibinqo	Normal metabolizer	CYP2C19	*1/*1	FDA 1[52]; Product monograph (actionable)[43]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Decreased, but not absent, risk for non-response	PTGS1 rs10306114	A/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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.

Aducanumab	Phenotype	Genetic Test	Results	Source/Evidence Level
Aduhelm	E3 Homozygous	ApoE	E3/E3	Product monograph (actionable)[9]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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

Alfentanil	Phenotype	Genetic Test	Results	Source/Evidence Level
Alfenta	Increased analgesic response	OPRM1 rs1799971	A/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require a reduced dose

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	Reduced risk of adverse drug reactions	HLA-B*58:01	Negative	CPIC A[48]; FDA 2[52]
	Decreased function	ABCG2 rs2231142	G/T	DPWG[18]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require an increased dose, may reduce efficacy

1 CPIC – HLA-B*58:01 Implication: Low or reduced risk of allopurinol-induced severe cutaneous adverse reaction (SCAR).
CPIC – HLA-B*58:01 Strong Recommendation: Use allopurinol per standard dosing guidelines.

2 DPWG – ABCG2 rs2231142 Description: The effectiveness of allopurinol is reduced, meaning that a higher dose is required. The gene variation reduces the excretion of uric acid by kidneys and intestines, meaning that a stronger inhibition of the uric acid production by allopurinol is required to achieve the desired uric acid concentration.

2 DPWG – ABCG2 rs2231142 Recommendation: Use 1.25 times the standard dose. This equates to a dose titration schedule of 100, 200, 400 and 500 mg/day instead of the usual schedule of 100, 200, 300 and 400 mg/day.

Alprazolam	Phenotype	Genetic Test	Results	Source/Evidence Level
Xanax	Intermediate metabolizer	CYP2C9	*1/*2	Clinical studies[25]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*2 or *3 allele was associated with an increased fall risk among those taking benzodiazepines (HR = 1.35; 95% CI: 1.06-1.72). Heterozygous allele carriers had a fall risk of HR = 1.30; 95% CI: 1.05-1.61, and homozygous allele carriers HR = 1.91; 95% CI: 1.23-2.96. 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.

Amifampridine	Phenotype	Genetic Test	Results	Source/Evidence Level
Firdapse	Poor metabolizer	NAT2	*6/*6	FDA 1[52]; Product monograph (actionable)[71]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require a reduced dose, may increase adverse events

2 FDA PGx Table Section 1 – NAT2 Therapeutic Management Recommendations for Amifampridine phosphate: Results in higher systemic concentrations. Use lowest recommended starting dosage (15 mg/day) and monitor for adverse reactions.

2 FDA Drug Label – NAT2 Actionable PGx for Amifampridine phosphate: The recommended starting dosage of Amifampridine phosphate in known NAT2 poor metabolizers is the lowest recommended initial daily dosage (i.e., 15 mg daily for pediatric patients weighing 45 kg or more and for adults, and 5 mg daily for pediatric patients weighing less than 45 kg) taken orally in divided doses. Exposure is increased in patients who are NAT2 poor metabolizers. Therefore, initiate Amifampridine phosphate in patients who are known NAT2 poor metabolizers at the lowest recommended initial daily dosage and monitor for adverse reactions. Consider dosage modification for patients who are known NAT2 poor metabolizers as needed based on clinical effect and tolerability.

Amitriptyline	Phenotype	Genetic Test	Results	Source/Evidence Level
Elavil Levate	Normal metabolizer	CYP2D6	*1.011/*41	CPIC A[27]; FDA 3[52]
	Normal metabolizer	CYP2C19	*1/*1	CPIC A[27]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – CYP2D6 Implication: Normal metabolism of TCAs.

1 CPIC – CYP2C19 Implication: Normal metabolism of tertiary amines.

1 CPIC – Strong Recommendation: Initiate therapy with recommended starting dose. Patients may receive an initial low dose of TCAs, 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.

Amoxapine	Phenotype	Genetic Test	Results	Source/Evidence Level
	Normal metabolizer	CYP2D6	*1.011/*41	FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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

Amphetamine	Phenotype	Genetic Test	Results	Source/Evidence Level
Adderall Adzenys Dyanavel Evekeo Mydayis	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Aripiprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Abilify	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]; Product monograph (actionable)[49]
	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Decreased likelihood of weight gain compared to A/A	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 A/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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) 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	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]; Product monograph (actionable)[3]
	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Decreased likelihood of weight gain compared to A/A	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 A/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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Ascorbic acid	Phenotype	Genetic Test	Results	Source/Evidence Level
Ascor Vitamin C	Normal	G6PD	B (reference)/B (reference)	Product monograph (actionable)[57]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 FDA Drug Label: No recommended changes or information for this G6PD phenotype in the FDA Drug Label.

Asenapine	Phenotype	Genetic Test	Results	Source/Evidence Level
Saphris	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Atazanavir	Phenotype	Genetic Test	Results	Source/Evidence Level
Reyataz	Normal/Extensive metabolizer	UGT1A1	*1/*1	CPIC A[20]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Normal metabolizer	CYP2D6 (Activity Score)	*1.011/*41	CPIC A[12]; FDA 1[52]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require an increased dose, may reduce efficacy

2 CPIC – Implication: Normal metabolizers of atomoxetine have a lower likelihood of response as compared to poor metabolizers. This is associated with increased discontinuation due to lack of efficacy 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[14]; FDA 3[52]
	Decreased response	ApoE rs7412	C/C	PharmGKB 2B[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may reduce efficacy

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

1 CPIC – SLCO1B1 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.

2 PharmGKB – ApoE Clinical Annotation (Level 2B Efficacy): Patients with Coronary Disease and Hyperlipidemias and the ApoE rs7412 C/C genotype may have decreased response to atorvastatin as compared to patients with the C/T or T/T genotype. Other genetic and clinical factors may also influence response to atorvastatin treatment.

Avatrombopag	Phenotype	Genetic Test	Results	Source/Evidence Level
Doptelet	Intermediate metabolizer	CYP2C9	*1/*2	FDA 3[52]
	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:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 FDA PGx Table Section 3 – CYP2C9 Potential Impact on Pharmacokinetic Properties Only: Results in higher systemic concentrations.

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[34]; FDA 1[52]
	Normal metabolizer	NUDT15	*1/*1	CPIC A[34]; FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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

1 CPIC – Strong Recommendation: Initiate therapy with standard starting dose (e.g., 2 mg/kg/day for autoimmune diseases). During therapy, adjust doses of azathioprine based on disease-specific guidelines. It usually takes at least 2 weeks to reach steady state after each dose adjustment.
Standard starting doses may vary by patient characteristics and/or treatment regimens and according to local clinical practice.

Brexpiprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Rexulti	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]; Product monograph (actionable)[4]
	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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.

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

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Brivaracetam	Phenotype	Genetic Test	Results	Source/Evidence Level
Briviact Brivlera	Normal metabolizer	CYP2C19	*1/*1	FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2C19 alleles do not indicate changes from recommended dose

Bromazepam	Phenotype	Genetic Test	Results	Source/Evidence Level
	Intermediate metabolizer	CYP2C9	*1/*2	Clinical studies[25]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*2 or *3 allele was associated with an increased fall risk among those taking benzodiazepines (HR = 1.35; 95% CI: 1.06-1.72). Heterozygous allele carriers had a fall risk of HR = 1.30; 95% CI: 1.05-1.61, and homozygous allele carriers HR = 1.91; 95% CI: 1.23-2.96. 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.

Buprenorphine	Phenotype	Genetic Test	Results	Source/Evidence Level
Belbuca Brixadi Butrans Sublocade Suboxone Zubsolv	Possible decreased dose requirement to prevent withdrawal symptoms, and increased analgesic response, compared to C/C or C/T	CYP3A4 rs2740574	T/T	PharmGKB 3[53,54]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 PharmGKB – Clinical Annotation (Level 3 Dosage): Patients with opioid-related disorders and the CYP3A4 rs2740574 T/T genotype may require a decreased dose of buprenorphine to prevent withdrawal symptoms as compared to patients with the C/T or C/C genotype. Other genetic and clinical factors may also influence dosage of buprenorphine in patients with opioid-related disorders.

1 PharmGKB – Clinical Annotation (Level 3 Efficacy): Patients with the CYP3A4 rs2740574 T/T genotype who are taking buprenorphine for pain may have an increased analgesic response as compared to patients with the C/C or C/T genotypes. Other genetic and clinical factors may also influence analgesic response to buprenorphine.

Bupropion	Phenotype	Genetic Test	Results	Source/Evidence Level
Wellbutrin Zyban	More likely to quit smoking	ANKK1/DRD2 rs1800497	G/G	PharmGKB 3[53,54]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 PharmGKB – Clinical Annotation (Level 3 Efficacy): Patients with the ANKK1 rs1800497 G/G genotype who are treated with bupropion may be more likely to quit smoking as compared to patients with the A/A or A/G genotypes, 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[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: Normal DPD activity and "normal" risk for fluoropyrimidine toxicity.

1 CPIC – Strong Recommendation: Based on genotype, there is no indication to change dose or therapy. Use label-recommended dosage and administration.

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[45]; FDA 2[52]
	Reduced risk of adverse drug reactions	HLA-B*15:02	Negative	CPIC A[45]; FDA 1[52]

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

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 risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Carisoprodol	Phenotype	Genetic Test	Results	Source/Evidence Level
	Normal metabolizer	CYP2C19	*1/*1	FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2C19 alleles do not indicate changes from recommended dose

Carvedilol	Phenotype	Genetic Test	Results	Source/Evidence Level
Coreg	Normal metabolizer	CYP2D6	*1.011/*41	FDA 2[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Celecoxib	Phenotype	Genetic Test	Results	Source/Evidence Level
Celebrex	Intermediate metabolizer (AS 1.5)	CYP2C9	*1/*2	CPIC A[51]; FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: Mildly reduced metabolism.

1 CPIC – Moderate Recommendation: Initiate therapy with recommended starting dose. In accordance with the prescribing information, use the lowest effective dosage for shortest duration consistent with individual patient treatment goals.
IMs might have a higher than normal risk of adverse events especially in individuals with other factors affecting clearance of these drugs such as hepatic impairment or advanced age.

Cevimeline	Phenotype	Genetic Test	Results	Source/Evidence Level
Evoxac	Normal metabolizer	CYP2D6	*1.011/*41	FDA 2[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Chlordiazepoxide	Phenotype	Genetic Test	Results	Source/Evidence Level
Librium	Intermediate metabolizer	CYP2C9	*1/*2	Clinical studies[25]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*2 or *3 allele was associated with an increased fall risk among those taking benzodiazepines (HR = 1.35; 95% CI: 1.06-1.72). Heterozygous allele carriers had a fall risk of HR = 1.30; 95% CI: 1.05-1.61, and homozygous allele carriers HR = 1.91; 95% CI: 1.23-2.96. 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.

Chloroquine	Phenotype	Genetic Test	Results	Source/Evidence Level
	Normal	G6PD	B (reference)/B (reference)	Product monograph (actionable)[58]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 FDA Drug Label: No recommended changes or information for this G6PD phenotype in the FDA Drug Label.

Chlorpromazine	Phenotype	Genetic Test	Results	Source/Evidence Level
	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Citalopram	Phenotype	Genetic Test	Results	Source/Evidence Level
Celexa	Normal metabolizer	CYP2C19	*1/*1	CPIC A[11]; FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 Normal CYP2C19 metabolism

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

Clobazam	Phenotype	Genetic Test	Results	Source/Evidence Level
Onfi Sympazan	Normal metabolizer	CYP2C19	*1/*1	FDA 1[52]; Product monograph (actionable)[33]
	Intermediate metabolizer	CYP2C9	*1/*2	Clinical Studies[25]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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

2 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*2 or *3 allele was associated with an increased fall risk among those taking benzodiazepines (HR = 1.35; 95% CI: 1.06-1.72). Heterozygous allele carriers had a fall risk of HR = 1.30; 95% CI: 1.05-1.61, and homozygous allele carriers HR = 1.91; 95% CI: 1.23-2.96. 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	Normal metabolizer	CYP2D6	*1.011/*41	CPIC B[27]; FDA 3[52]
	Normal metabolizer	CYP2C19	*1/*1	CPIC B[27]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – CYP2D6 Implication: Normal metabolism of TCAs.

1 CPIC – CYP2C19 Implication: Normal metabolism of tertiary amines.

1 CPIC – Optional Recommendation: Initiate therapy with recommended starting dose. Patients may receive an initial low dose of TCAs, 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.

Clonazepam	Phenotype	Genetic Test	Results	Source/Evidence Level
Klonopin Rivotril	Intermediate metabolizer	CYP2C9	*1/*2	Clinical studies[25]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*2 or *3 allele was associated with an increased fall risk among those taking benzodiazepines (HR = 1.35; 95% CI: 1.06-1.72). Heterozygous allele carriers had a fall risk of HR = 1.30; 95% CI: 1.05-1.61, and homozygous allele carriers HR = 1.91; 95% CI: 1.23-2.96. 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	Normal metabolizer	CYP2C19	*1/*1	CPIC A[31]; FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: Normal clopidogrel active metabolite formation; normal on-treatment platelet reactivity.

1 CPIC – Strong Recommendation: If considering clopidogrel, use at standard dose (75 mg/day).

Clorazepate	Phenotype	Genetic Test	Results	Source/Evidence Level
Gen-Xene Tranxene	Intermediate metabolizer	CYP2C9	*1/*2	Clinical studies[25]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*2 or *3 allele was associated with an increased fall risk among those taking benzodiazepines (HR = 1.35; 95% CI: 1.06-1.72). Heterozygous allele carriers had a fall risk of HR = 1.30; 95% CI: 1.05-1.61, and homozygous allele carriers HR = 1.91; 95% CI: 1.23-2.96. 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	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]
	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased risk of developing metabolic syndrome	HTR2C rs1414334	G/G	PharmGKB 3[53,54]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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.

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

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Codeine	Phenotype	Genetic Test	Results	Source/Evidence Level
Codeine Contin Tylenol with Codeine No. 2/3/4	Normal metabolizer	CYP2D6	*1.011/*41	CPIC A[15]; FDA 1[52]; FDA 2[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: Expected morphine formation.

1 CPIC – Strong Recommendation: Use codeine label recommended age-specific or weight-specific dosing.

Cyclosporine	Phenotype	Genetic Test	Results	Source/Evidence Level
Neoral Sandimmune	Poor metabolizer	CYP3A5	*3/*3	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require a reduced dose

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).

Dabrafenib	Phenotype	Genetic Test	Results	Source/Evidence Level
Tafinlar	Normal	G6PD	B (reference)/B (reference)	Product monograph (actionable)[59]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 FDA Drug Label: No recommended changes or information for this G6PD phenotype in the FDA Drug Label.

Dapsone	Phenotype	Genetic Test	Results	Source/Evidence Level
Aczone	Normal phenotype	G6PD	B (reference)/B (reference)	CPIC A[21]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Normal metabolizer	CYP2D6	*1.011/*41	FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Daunorubicin	Phenotype	Genetic Test	Results	Source/Evidence Level
Cerubidine Vyxeos	G/G	SLC28A3 rs7853758	G/G	PharmGKB 2B[60]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 ClinPGx – SLC28A3 rs7853758 Summary Annotation (Level 2B Toxicity): Pediatric patients with the SLC28A3 rs7853758 G/G genotype and Neoplasms may have increased likelihood of cardiotoxicity when treated with anthracyclines as compared to pediatric patients with the A/A or A/G genotype. Other genetic and clinical factors may also influence development of cardiotoxicity.

Desflurane	Phenotype	Genetic Test	Results	Source/Evidence Level
Suprane	Uncertain Malignant Hyperthermia Susceptibility	CACNA1S	Reference/Reference	CPIC A[24]
	Uncertain Malignant Hyperthermia Susceptibility	RYR1	Reference/Reference	CPIC A[24]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: These results do not eliminate the chance that this patient is susceptible to MH (malignant hyperthermia). The genetic cause of about half of all MH survivors, with MH susceptibility confirmed by contracture test, remains unknown.

1 CPIC – Strong Recommendation: Clinical findings, family history, further genetic testing, and other laboratory data should guide use of halogenated volatile anesthetics or depolarizing muscle relaxants.

Desipramine	Phenotype	Genetic Test	Results	Source/Evidence Level
Norpramin	Normal metabolizer	CYP2D6	*1.011/*41	CPIC B[27]; FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – CYP2D6 Implication: Normal metabolism of TCAs.

1 CPIC – Strong Recommendation: Initiate therapy with 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.

Deuruxolitinib	Phenotype	Genetic Test	Results	Source/Evidence Level
Leqselvi	Intermediate metabolizer	CYP2C9	*1/*2	Product monograph (actionable)[61]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 FDA Drug Label – CYP2C9 Testing Recommended: The pharmacokinetics of deuruxolitinib were not evaluated in individuals who are intermediate metabolizers in the FDA Drug Label.

Deutetrabenazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Austedo	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Dexlansoprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Dexilant	Normal metabolizer	CYP2C19	*1/*1	CPIC B[32]; FDA 3[52]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require an increased dose, may reduce efficacy

2 CPIC – Implication: Normal PPI metabolism; may be at increased risk of therapeutic failure compared with CYP2C19 IMs and PMs.

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.

Dextromethorphan/ Bupropion	Phenotype	Genetic Test	Results	Source/Evidence Level
Auvelity	Normal metabolizer	CYP2D6	*1.011/*41	Product monograph (actionable)[7]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 FDA Product Monograph: no recommended changes or information for this CYP2D6 phenotype in the FDA Product Monograph.

Dextromethorphan/ Quinidine	Phenotype	Genetic Test	Results	Source/Evidence Level
Nuedexta	Normal metabolizer	CYP2D6	*1.011/*41	Product monograph (actionable)[6]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 FDA Product Monograph: no recommended changes or information for this CYP2D6 phenotype in the FDA Product Monograph.

Diazepam	Phenotype	Genetic Test	Results	Source/Evidence Level
Diastat Valium	Normal metabolizer	CYP2C19	*1/*1	FDA 3[52]
	Intermediate metabolizer	CYP2C9	*1/*2	Clinical Studies[25]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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

2 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*2 or *3 allele was associated with an increased fall risk among those taking benzodiazepines (HR = 1.35; 95% CI: 1.06-1.72). Heterozygous allele carriers had a fall risk of HR = 1.30; 95% CI: 1.05-1.61, and homozygous allele carriers HR = 1.91; 95% CI: 1.23-2.96. 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.

Dolutegravir	Phenotype	Genetic Test	Results	Source/Evidence Level
Tivicay	Normal/Extensive metabolizer	UGT1A1	*1/*1	FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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

Donanemab	Phenotype	Genetic Test	Results	Source/Evidence Level
Kisunla	E3/E3	ApoE	E3/E3	Product monograph (actionable)[62]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 FDA Drug Label: No recommended changes or information for this APOE phenotype in the FDA Drug Label.

Donepezil	Phenotype	Genetic Test	Results	Source/Evidence Level
Aricept	Normal metabolizer	CYP2D6	*1.011/*41	FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Doxepin	Phenotype	Genetic Test	Results	Source/Evidence Level
Silenor Sinequan	Normal metabolizer	CYP2D6	*1.011/*41	CPIC B[27]; FDA 3[52]
	Normal metabolizer	CYP2C19	*1/*1	CPIC B[27]; FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – CYP2D6 Implication: Normal metabolism of TCAs.

1 CPIC – CYP2C19 Implication: Normal metabolism of tertiary amines.

1 CPIC – Optional Recommendation: Initiate therapy with recommended starting dose. Patients may receive an initial low dose of TCAs, 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.

Doxorubicin	Phenotype	Genetic Test	Results	Source/Evidence Level
Doxil Caelyx	G/G	SLC28A3 rs7853758	G/G	PharmGKB 2B[60]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 ClinPGx – SLC28A3 rs7853758 Summary Annotation (Level 2B Toxicity): Pediatric patients with the SLC28A3 rs7853758 G/G genotype and Neoplasms may have increased likelihood of cardiotoxicity when treated with anthracyclines as compared to pediatric patients with the A/A or A/G genotype. Other genetic and clinical factors may also influence development of cardiotoxicity.

Dronabinol	Phenotype	Genetic Test	Results	Source/Evidence Level
Marinol Syndros	Intermediate metabolizer	CYP2C9	*1/*2	FDA 1[52]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require a reduced dose, may increase adverse events

2 CYP2C9 intermediate metabolizer: reduced metabolism of Dronabinol 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

Efavirenz	Phenotype	Genetic Test	Results	Source/Evidence Level
Sustiva	Intermediate metabolizer	CYP2B6	*1/*6	CPIC A[16]; FDA 2[52]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require a reduced dose, may increase adverse events

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[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 SLCO1B1 alleles indicate a typical response to Elagolix

Eliglustat	Phenotype	Genetic Test	Results	Source/Evidence Level
Cerdelga	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]; Product monograph (actionable)[22]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

1 Multiple drug-drug interactions may further affect the safety of Eliglustat, 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)[42]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Intermediate metabolizer	CYP2C9	*1/*2	FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Normal metabolizer	CYP2C19	*1/*1	CPIC A[11]; FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 Normal CYP2C19 metabolism

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

Esomeprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Nexium	Normal metabolizer	CYP2C19	*1/*1	FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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[53,54]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Decreased analgesic response	OPRM1 rs1799971	A/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require a reduced dose, may reduce efficacy

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	Normal metabolizer	CYP2D6	*1.011/*41	FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Flecainide	Phenotype	Genetic Test	Results	Source/Evidence Level
Tambocor	Normal metabolizer	CYP2D6	*1.011/*41	DPWG[18]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Flibanserin	Phenotype	Genetic Test	Results	Source/Evidence Level
Addyi	Normal metabolizer	CYP2C19	*1/*1	FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2C19 alleles do not indicate changes from recommended dose

Fluorouracil	Phenotype	Genetic Test	Results	Source/Evidence Level
Carac Efudex Fluoroplex Tolak	Normal metabolizer	DPYD	*1/*1	CPIC A[5]; FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: Normal DPD activity and "normal" risk for fluoropyrimidine toxicity.

1 CPIC – Strong Recommendation: Based on genotype, there is no indication to change dose or therapy. Use label-recommended dosage and administration.

Fluoxetine	Phenotype	Genetic Test	Results	Source/Evidence Level
Prozac	Normal metabolizer	CYP2D6	*1.011/*41	Product monograph (actionable)[13]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Fluphenazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Modecate	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Flurazepam	Phenotype	Genetic Test	Results	Source/Evidence Level
	Intermediate metabolizer	CYP2C9	*1/*2	Clinical studies[25]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*2 or *3 allele was associated with an increased fall risk among those taking benzodiazepines (HR = 1.35; 95% CI: 1.06-1.72). Heterozygous allele carriers had a fall risk of HR = 1.30; 95% CI: 1.05-1.61, and homozygous allele carriers HR = 1.91; 95% CI: 1.23-2.96. 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	Intermediate metabolizer (AS 1.5)	CYP2C9	*1/*2	CPIC A[51]; FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: Mildly reduced metabolism.

1 CPIC – Moderate Recommendation: Initiate therapy with recommended starting dose. In accordance with the prescribing information, use the lowest effective dosage for shortest duration consistent with individual patient treatment goals.
IMs might have a higher than normal risk of adverse events especially in individuals with other factors affecting clearance of these drugs such as hepatic impairment or advanced age.

Flutamide	Phenotype	Genetic Test	Results	Source/Evidence Level
	Normal	G6PD	B (reference)/B (reference)	Product monograph (actionable)[63]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 FDA Drug Label: No recommended changes or information for this G6PD phenotype in the FDA Drug Label.

Fluvastatin	Phenotype	Genetic Test	Results	Source/Evidence Level
Lescol	Intermediate metabolizer	CYP2C9	*1/*2	CPIC A[14]
	Normal function	SLCO1B1	*1/*1	CPIC A[14]

Drug-Gene Interactions:

Moderate drug-gene interaction: consider alternative medications, may require a reduced dose, may increase adverse events

2 CPIC – CYP2C9 Implication: Increased fluvastatin exposure as compared with normal metabolizer, which may translate to increased myopathy risk.

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

2 CPIC – Moderate Recommendation: Prescribe ≤40 mg per day as a starting dose and adjust doses of fluvastatin based on disease-specific guidelines. If dose >40 mg needed for desired efficacy, consider an alternative statin or combination therapy (i.e., fluvastatin plus non-statin guideline-directed medical therapy). The potential for drug-drug interactions and dose limits based on renal and hepatic function should be evaluated prior to initiating a statin. The effects of drug-drug interactions may be more pronounced, resulting in a higher risk of myopathy.

Fluvoxamine	Phenotype	Genetic Test	Results	Source/Evidence Level
Luvox	Normal metabolizer	CYP2D6	*1.011/*41	CPIC B[11]; FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 Normal CYP2D6 metabolism

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

Fosphenytoin	Phenotype	Genetic Test	Results	Source/Evidence Level
Cerebyx	Reduced risk of adverse drug reactions	HLA-B*15:02	Negative	CPIC A[30]; FDA 1[52]
	Intermediate metabolizer (AS 1.5)	CYP2C9	*1/*2	CPIC A[30]; FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

CPIC – CYP2C9 Implication: Slightly reduced Fosphenytoin metabolism; however, this does not appear to translate into increased side effects.

CPIC – Moderate 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	Normal metabolizer	CYP2D6	*1.011/*41	FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

CYP2D6 alleles do not indicate changes from recommended dose

Gefitinib	Phenotype	Genetic Test	Results	Source/Evidence Level
Iressa	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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

Glimepiride	Phenotype	Genetic Test	Results	Source/Evidence Level
	Normal	G6PD	B (reference)/B (reference)	Product monograph (actionable)[64]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

FDA Drug Label: No recommended changes or information for this G6PD phenotype in the FDA Drug Label.

Glipizide	Phenotype	Genetic Test	Results	Source/Evidence Level
Glucotrol XL	Normal	G6PD	B (reference)/B (reference)	Product monograph (actionable)[65]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

FDA Drug Label: No recommended changes or information for this G6PD phenotype in the FDA Drug Label.

Glyburide	Phenotype	Genetic Test	Results	Source/Evidence Level
Diabeta	Normal	G6PD	B (reference)/B (reference)	Product monograph (actionable)[66]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

FDA Drug Label: No recommended changes or information for this G6PD phenotype in the FDA Drug Label.

Haloperidol	Phenotype	Genetic Test	Results	Source/Evidence Level
Haldol	Normal metabolizer	CYP2D6	*1.011/*41	DPWG[18]
	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Decreased likelihood of side effects e.g. weight gain and hypertriglyceridemia compared to A/A	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

1 DPWG: no recommendation for this CYP2D6 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 A/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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Halothane	Phenotype	Genetic Test	Results	Source/Evidence Level
	Uncertain Malignant Hyperthermia Susceptibility	CACNA1S	Reference/Reference	CPIC A[24]
	Uncertain Malignant Hyperthermia Susceptibility	RYR1	Reference/Reference	CPIC A[24]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: These results do not eliminate the chance that this patient is susceptible to MH (malignant hyperthermia). The genetic cause of about half of all MH survivors, with MH susceptibility confirmed by contracture test, remains unknown.

1 CPIC – Strong Recommendation: Clinical findings, family history, further genetic testing, and other laboratory data should guide use of halogenated volatile anesthetics or depolarizing muscle relaxants.

Hydralazine	Phenotype	Genetic Test	Results	Source/Evidence Level
	Poor metabolizer	NAT2	*6/*6	CPIC A[67]; FDA 3[52]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require a reduced dose, may increase adverse events

2 CPIC – NAT2 Implication: Predicted to have increased hydralazine plasma concentrations compared to NAT2 rapid and intermediate metabolizers, which may lead to both increased efficacy and adverse effects, including drug-induced systemic lupus erythematosus.

2 CPIC – NAT2 Moderate Recommendation: In adults with resistant hypertension, initiate therapy at a total daily dose of 40 to 75 mg. Carefully titrate dose upward to clinical effect or guideline-recommended dose; use caution with total daily hydralazine doses of 200 mg or more.

1 FDA PGx Table Section 3 – NAT2 Potential Impact on Pharmacokinetic Properties Only: Results in higher systemic concentrations.
Note: the FDA Table refers to the gene "Nonspecific (NAT)" which ClinPGx interprets as NAT2.

Hydrocodone	Phenotype	Genetic Test	Results	Source/Evidence Level
Anexsia Hycodan Hysingla Zohydro	Normal metabolizer	CYP2D6	*1.011/*41	CPIC B[15]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

CPIC – Implication: Normal hydromorphone formation.

CPIC – Strong Recommendation: Use hydrocodone label recommended age-specific or weight-specific dosing.

Hydroxychloroquine	Phenotype	Genetic Test	Results	Source/Evidence Level
Plaquenil Sovuna	Normal	G6PD	B (reference)/B (reference)	Product monograph (actionable)[68]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

FDA Drug Label: No recommended changes or information for this G6PD phenotype in the FDA Drug Label.

Ibuprofen	Phenotype	Genetic Test	Results	Source/Evidence Level
Advil Caldolor Duexis Motrin IB NeoProfen	Intermediate metabolizer (AS 1.5)	CYP2C9	*1/*2	CPIC A[51]; FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

CPIC – Implication: Mildly reduced metabolism.

CPIC – Moderate Recommendation: Initiate therapy with lowest recommended starting dose. Titrate dose upward to clinical effect or maximum recommended dose with caution. In accordance with the prescribing information, use the lowest effective dosage for shortest duration consistent with individual patient treatment goals. Carefully monitor adverse events such as blood pressure and kidney function during course of therapy. IMs might have a higher than normal risk of adverse events especially in individuals with other factors affecting clearance of these drugs such as hepatic impairment or advanced age. Further caution should be taken with ibuprofen use in individuals carrying the CYP2C9*2 allele as it is in linkage disequilibrium with CYP2C8*3 and ibuprofen is also metabolized by CYP2C8.

Iloperidone	Phenotype	Genetic Test	Results	Source/Evidence Level
Fanapt	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]
	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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.

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

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Imipramine	Phenotype	Genetic Test	Results	Source/Evidence Level
Tofranil	Normal metabolizer	CYP2D6	*1.011/*41	CPIC B[27]; FDA 3[52]
	Normal metabolizer	CYP2C19	*1/*1	CPIC B[27]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – CYP2D6 Implication: Normal metabolism of TCAs.

1 CPIC – CYP2C19 Implication: Normal metabolism of tertiary amines.

1 CPIC – Optional Recommendation: Initiate therapy with recommended starting dose. Patients may receive an initial low dose of TCAs, 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.

Irinotecan	Phenotype	Genetic Test	Results	Source/Evidence Level
Camptosar Onivyde	Normal/Extensive metabolizer	UGT1A1	*1/*1	DPWG[18]; FDA 1[52]; Product monograph (actionable)[28,44]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Uncertain Malignant Hyperthermia Susceptibility	CACNA1S	Reference/Reference	CPIC A[24]
	Uncertain Malignant Hyperthermia Susceptibility	RYR1	Reference/Reference	CPIC A[24]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: These results do not eliminate the chance that this patient is susceptible to MH (malignant hyperthermia). The genetic cause of about half of all MH survivors, with MH susceptibility confirmed by contracture test, remains unknown.

1 CPIC – Strong Recommendation: Clinical findings, family history, further genetic testing, and other laboratory data should guide use of halogenated volatile anesthetics or depolarizing muscle relaxants.

Isoniazid	Phenotype	Genetic Test	Results	Source/Evidence Level
	Poor metabolizer	NAT2	*6/*6	FDA 2[52]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 FDA PGx Table Section 2 – NAT2 Potential Impact on Safety or Response: May result in higher systemic concentrations and adverse reaction risk.
Note: the FDA Table refers to the gene "Nonspecific (NAT)" which ClinPGx interprets as NAT2.

Lamotrigine	Phenotype	Genetic Test	Results	Source/Evidence Level
Lamictal	Reduced risk of adverse drug reactions	HLA-B*15:02	Negative	DPWG[18]
	Reduced risk of adverse drug reactions	HLA-B*58:01	Negative	PharmGKB 3[53,54]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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

1 PharmGKB: No recommended changes or information for this HLA-B *58:01 phenotype in PharmGKB.

Lansoprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Prevacid	Normal metabolizer	CYP2C19	*1/*1	CPIC A[32]; FDA 3[52]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require an increased dose, may reduce efficacy

2 CPIC – Implication: Normal PPI metabolism; may be at increased risk of therapeutic failure compared with CYP2C19 IMs and PMs.

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.

Lecanemab	Phenotype	Genetic Test	Results	Source/Evidence Level
Leqembi	E3 Homozygous	ApoE	E3/E3	Product monograph (actionable)[19]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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

Lofexidine	Phenotype	Genetic Test	Results	Source/Evidence Level
Lucemyra	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Lorazepam	Phenotype	Genetic Test	Results	Source/Evidence Level
Ativan	A/C	UGT2B15 rs1902023	A/C	PharmGKB 3[60]
	Intermediate metabolizer	CYP2C9	*1/*2	Clinical studies[25]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

1 ClinPGx – UGT2B15 rs1902023 Summary Annotation (Level 3 Metabolism/PK): Subjects with the UGT2B15 rs1902023 A/C genotype may have decreased clearance of lorazepam as compared to subjects with the C/C genotype, or increased clearance as compared to subjects with the A/A genotype. Other genetic and clinical factors may also influence the metabolism of lorazepam.

2 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*2 or *3 allele was associated with an increased fall risk among those taking benzodiazepines (HR = 1.35; 95% CI: 1.06-1.72). Heterozygous allele carriers had a fall risk of HR = 1.30; 95% CI: 1.05-1.61, and homozygous allele carriers HR = 1.91; 95% CI: 1.23-2.96. 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[14]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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

1 CPIC – SLCO1B1 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	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Lurasidone	Phenotype	Genetic Test	Results	Source/Evidence Level
Latuda	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Lusutrombopag	Phenotype	Genetic Test	Results	Source/Evidence Level
Mupleta	Normal Factor II	Factor II rs1799963	G/G	Product monograph (actionable)[50]
	Normal Factor V Leiden	Factor V rs6025	C/C	Product monograph (actionable)[50]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Normal metabolizer	CYP2C19	*1/*1	FDA 2[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Meloxicam	Phenotype	Genetic Test	Results	Source/Evidence Level
Anjeso Mobic Qmiiz ODT Vivlodex	Intermediate metabolizer (AS 1.5)	CYP2C9	*1/*2	CPIC A[51]; FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: Mildly reduced metabolism.

1 CPIC – Moderate Recommendation: Initiate therapy with recommended starting dose. In accordance with the prescribing information, use the lowest effective dosage for shortest duration consistent with individual patient treatment goals.
IMs might have a higher than normal risk of adverse events especially in individuals with other factors affecting clearance of these drugs such as hepatic impairment or advanced age.

Mercaptopurine	Phenotype	Genetic Test	Results	Source/Evidence Level
Purinethol Purixan	Normal metabolizer	TPMT	*1/*1	CPIC A[34]; FDA 1[52]
	Normal metabolizer	NUDT15	*1/*1	CPIC A[34]; FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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

1 CPIC – Strong Recommendation: Initiate therapy with standard starting dose of mecaptopurine (e.g., 75 mg/m2/day for malignancy or 1.5 mg/kg/day for nonmalignancy). During therapy, adjust the doses of myelosuppressive agents, as per standard clinical practice. It usually takes at least 2 weeks of stable dosing to reach steady state after each dose adjustment.
Standard starting doses may vary by patient characteristics and/or treatment regimens and according to local clinical practice.

Methadone	Phenotype	Genetic Test	Results	Source/Evidence Level
Methadose	Intermediate metabolizer	CYP2B6	*1/*6	PharmGKB 2A[47,53,54]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – CYP2B6 Level C Implication, as adapted by PharmGKB (Level 2A Metabolism/PK Clinical Annotation): Increased S-methadone plasma concentrations; unknown clinical implications. No difference in steady-state R-methadone plasma concentrations.
PharmGKB: Other genetic and clinical factors may also affect methadone metabolism. This annotation only covers the pharmacokinetic relationship between CYP2B6 and methadone and does not include evidence about clinical outcomes.

1 CPIC – CYP2B6 Moderate Recommendation: Standard dosing, titration, and monitoring of methadone. Clinical guidelines for ECG monitoring in the context of methadone therapy, including risk assessment of other clinical factors, should be followed. None of the pharmacogenetic information in this guideline should be interpreted to influence the use of ECG monitoring guidelines.

Methotrexate	Phenotype	Genetic Test	Results	Source/Evidence Level
Metoject Otrexup Rasuvo Trexall Xatmep	Increased risk of toxicity	MTHFR rs1801133	A/A	PharmGKB 2A[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 PharmGKB – Clinical Annotation (Level 2A Toxicity): Patients with the MTHFR rs1801133 A/A genotype and cancer or arthritis who are treated with methotrexate may have an increased risk of toxicity as compared to patients with the A/G or G/G 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	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Methoxyflurane	Phenotype	Genetic Test	Results	Source/Evidence Level
Penthrox	Uncertain Malignant Hyperthermia Susceptibility	CACNA1S	Reference/Reference	CPIC A[24]
	Uncertain Malignant Hyperthermia Susceptibility	RYR1	Reference/Reference	CPIC A[24]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: These results do not eliminate the chance that this patient is susceptible to MH (malignant hyperthermia). The genetic cause of about half of all MH survivors, with MH susceptibility confirmed by contracture test, remains unknown.

1 CPIC – Strong Recommendation: Clinical findings, family history, further genetic testing, and other laboratory data should guide use of halogenated volatile anesthetics or depolarizing muscle relaxants.

Methylene blue	Phenotype	Genetic Test	Results	Source/Evidence Level
Provayblue	Normal phenotype	G6PD	B (reference)/B (reference)	CPIC A[21]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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

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

Methylphenidate	Phenotype	Genetic Test	Results	Source/Evidence Level
Aptensio Concerta Cotempla Daytrana Jornay Metadate Methylin Quillichew Quillivant Relexxiii Ritalin	Poorer response	ADRA2A rs1800544	C/C	PharmGKB 4[53,54]
	No significant association to response	COMT rs4680	A/A	PharmGKB 4[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may reduce efficacy

1 PharmGKB – Clinical Annotation (Level 4 Efficacy): The current evidence base suggests that there is no significant association between the COMT rs4680 A/A 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	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Metoprolol	Phenotype	Genetic Test	Results	Source/Evidence Level
Kapspargo Sprinkle Lopressor Toprol-XL	Normal metabolizer	CYP2D6	*1.011/*41	CPIC B[17]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: Normal metabolism of metoprolol.

1 CPIC – Strong Recommendation: Initiate standard dosing.

Mirabegron	Phenotype	Genetic Test	Results	Source/Evidence Level
Myrbetriq	Normal metabolizer	CYP2D6	*1.011/*41	FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Mirtazapine	Phenotype	Genetic Test	Results	Source/Evidence Level
Remeron	Normal metabolizer	CYP2D6	*1.011/*41	DPWG[18]; PharmGKB 2A[53,54]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 DPWG: no recommendation for this CYP2D6 phenotype.

Molindone	Phenotype	Genetic Test	Results	Source/Evidence Level
Moban	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Morphine	Phenotype	Genetic Test	Results	Source/Evidence Level
Kadian M-Eslon Morphabond ER MS Contin MS-IR Statex	Increased analgesic response	OPRM1 rs1799971	A/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require a reduced dose

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
	Intermediate metabolizer	CYP2C9	*1/*2	FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Normal metabolizer	CYP2D6	*1.011/*41	FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Nevirapine	Phenotype	Genetic Test	Results	Source/Evidence Level
Viramune	Decreased risk for SJS/TEN	CYP2B6 rs28399499	T/T	PharmGKB 2A[53,54]
	Decreased clearance and increased exposure compared to G/G	CYP2B6 rs3745274	G/T	PharmGKB 2A[53,54]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 PharmGKB – CYP2B6 Clinical Annotation (Level 2A Toxicity): Patients with the CYP2B6 rs28399499 T/T genotype and HIV may have a decreased risk for Stevens-Johnson Syndrome/toxic epidermal necrolysis (SJS/TEN) when treated with nevirapine as compared to patients with the C/C or C/T genotype. Other genetic and clinical factors may also influence risk for developing SJS/TEN when receiving nevirapine.

1 PharmGKB – CYP2B6 Clinical Annotation (Level 2A Metabolism/PK): Patients with the CYP2B6 rs3745274 G/T genotype and HIV infection may have decreased clearance of and increased exposure to nevirapine as compared to patients with the G/G genotype. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence clearance of nevirapine and exposure to drug. This annotation only covers the pharmacokinetic relationship between CYP2B6 rs3745274 and nevirapine and does not include evidence about clinical outcomes.

Nicotine replacement therapy	Phenotype	Genetic Test	Results	Source/Evidence Level
Nicorette Nicotrol Habitrol Nicoderm Thrive	Decreased likelihood of smoking cessation	ANKK1/DRD2 rs1800497	G/G	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may reduce efficacy

2 PharmGKB – Clinical Annotation (Level 3 Efficacy): Patients with the ANKK1 rs1800497 G/G genotype may have a decreased likelihood of smoking cessation when treated with nicotine replacement therapy as compared to patients with the A/G and A/A 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	Intermediate metabolizer	CYP2C9	*1/*2	Clinical studies[25]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*2 or *3 allele was associated with an increased fall risk among those taking benzodiazepines (HR = 1.35; 95% CI: 1.06-1.72). Heterozygous allele carriers had a fall risk of HR = 1.30; 95% CI: 1.05-1.61, and homozygous allele carriers HR = 1.91; 95% CI: 1.23-2.96. 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	Normal phenotype	G6PD	B (reference)/B (reference)	CPIC A[21]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Normal metabolizer	CYP2D6	*1.011/*41	CPIC A[27]; FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – CYP2D6 Implication: Normal metabolism of TCAs.

1 CPIC – Strong Recommendation: Initiate therapy with 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.

Olanzapine	Phenotype	Genetic Test	Results	Source/Evidence Level
Zyprexa	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	May have increased time until response	DRD2 rs1799978	T/T	PharmGKB 3[53,54]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may reduce efficacy, may increase adverse events

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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) 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.

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Oliceridine	Phenotype	Genetic Test	Results	Source/Evidence Level
Olinvyk	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Normal metabolizer	CYP2C19	*1/*1	CPIC A[32]; FDA 3[52]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require an increased dose, may reduce efficacy

2 CPIC – Implication: Normal PPI metabolism; may be at increased risk of therapeutic failure compared with CYP2C19 IMs and PMs.

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	Normal metabolizer	CYP2D6	*1.011/*41	CPIC A[37]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: Normal metabolism.

1 CPIC – Strong Recommendation: Initiate therapy with recommended starting dose.
Drug-drug interactions, the involvement of other metabolizing enzymes, and patient characteristics (e.g., age, renal and hepatic function) should be considered when selecting alternative therapy, as emerging evidence may further clarify the role of enzymes beyond CYP2D6 in the metabolism of these agents.

Oral contraceptives	Phenotype	Genetic Test	Results	Source/Evidence Level
	Decreased risk for DVT	Factor II rs1799963	G/G	PharmGKB 2B[53,54]
	Decreased risk of thrombosis (normal Factor V)	Factor V rs6025	C/C	PharmGKB 2B[53,54]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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
	Intermediate metabolizer	CYP2C9	*1/*2	Clinical studies[25]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*2 or *3 allele was associated with an increased fall risk among those taking benzodiazepines (HR = 1.35; 95% CI: 1.06-1.72). Heterozygous allele carriers had a fall risk of HR = 1.30; 95% CI: 1.05-1.61, and homozygous allele carriers HR = 1.91; 95% CI: 1.23-2.96. 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[45]; FDA 2[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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.

Oxycodone	Phenotype	Genetic Test	Results	Source/Evidence Level
OxyContin Roxicodone Roxybond Xtampza ER	Normal metabolizer	CYP2D6	*1.011/*41	PharmGKB 2A[15,53,54]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – CYP2D6 Level C Implication, as adapted by PharmGKB (Level 2A Metabolism/PK Clinical Annotation): Expected oxymorphone formation.
PharmGKB: This annotation only covers the pharmacokinetic relationship between CYP2D6 and oxycodone and does not include evidence about clinical outcomes. This drug-variant pair has been assigned a "no recommendation" by CPIC and DPWG, as it was determined to be not clinically actionable. Other genetic and clinical factors may also affect oxycodone metabolism.

1 CPIC – CYP2D6 Strong Recommendation: Use oxycodone label recommended age- or weight-specific dosing.

Paliperidone	Phenotype	Genetic Test	Results	Source/Evidence Level
Invega	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Decreased likelihood of weight gain compared to A/A	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

1 PharmGKB – Clinical Annotation (Level 3 Efficacy): Patients with the MC4R rs489693 A/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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) 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	Normal metabolizer	CYP2C19	*1/*1	CPIC A[32]; FDA 1[52]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require an increased dose, may reduce efficacy

2 CPIC – Implication: Normal PPI metabolism; may be at increased risk of therapeutic failure compared with CYP2C19 IMs and PMs.

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	Normal metabolizer	CYP2D6	*1.011/*41	CPIC A[11]; FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 Normal metabolism of paroxetine to less active compounds. Paroxetine-associated phenoconversion of normal metabolizers to intermediate or poor metabolizers due to CYP2D6 autoinhibition may occur and is dose-dependent and greater at steady state concentrations.

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

Pazopanib	Phenotype	Genetic Test	Results	Source/Evidence Level
Votrient	Increased risk of adverse drug reactions	HLA-B*57:01	Positive	FDA 2[52]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 FDA PGx Table Section 2 – HLA-B *57:01 Potential Impact on Safety or Response: May result in higher adverse reaction risk (liver enzyme elevations). Monitor liver function tests regardless of genotype.

PEG-interferon alpha	Phenotype	Genetic Test	Results	Source/Evidence Level
Alferon N Injection Intron A Pegasys Pegintron Sylatron	C/C	IFNL3 rs12979860	C/C	PharmGKB 1B[60]; PharmGKB 2A[60]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 ClinPGx – IFNL3 rs12979860 Summary Annotation (Level 1B Efficacy): Patients with the IFNL3 rs12979860 C/C genotype and hepatitis C infection may have increased response (typically assayed as sustained virological response, SVR) when administered peg interferon alpha 2a or 2b in combination with ribavirin as compared to patients with the C/T or T/T genotypes. However, conflicting evidence has been reported. Other genetic and clinical factors may also influence response to peginterferon alpha and ribavirin.

1 ClinPGx – IFNL3 rs12979860 Summary Annotation (Level 2A Efficacy): Patients with the IFNL3 rs12979860 C/C genotype and hepatitis C infection may have increased response to triple therapy (boceprevir, peginterferon alfa-2a/2b and ribavirin) as compared to patients with the C/T or T/T genotype. Other genetic and clinical factors may also influence response to boceprevir-peginterferon based therapy.

1 ClinPGx – IFNL3 rs12979860 Summary Annotation (Level 2A Efficacy): Patients with the IFNL3 rs12979860 C/C genotype and hepatitis C infection may have higher response rates (SVR) to triple therapy (telaprevir, peginterferon alfa-2a/b and ribavirin) as compared to patients with the C/T or T/T genotype. However, conflicting evidence has been reported. The impact of IL28B genotype may be dampened in patients with prior PegIFN/RBV treatment failure. Other genetic and clinical factors may also influence response to HCV triple therapy.

Pegloticase	Phenotype	Genetic Test	Results	Source/Evidence Level
Krystexxa	Normal phenotype	G6PD	B (reference)/B (reference)	CPIC A[21]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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
	Normal metabolizer	CYP2D6	*1.011/*41	FDA 2[52]
	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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.

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

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Phenytoin	Phenotype	Genetic Test	Results	Source/Evidence Level
Dilantin Tremytoine Phenytek	Reduced risk of adverse drug reactions	HLA-B*15:02	Negative	CPIC A[30]; FDA 1[52]
	Intermediate metabolizer (AS 1.5)	CYP2C9	*1/*2	CPIC A[30]; FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – CYP2C9 Implication: Slightly reduced Phenytoin metabolism; however, this does not appear to translate into increased side effects.

1 CPIC – Moderate 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	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]
	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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.

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

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Piroxicam	Phenotype	Genetic Test	Results	Source/Evidence Level
Feldene	Intermediate metabolizer (AS 1.5)	CYP2C9	*1/*2	CPIC A[51]; FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: Mildly reduced metabolism.

1 CPIC – Moderate Recommendation: Initiate therapy with recommended starting dose. In accordance with the prescribing information, use the lowest effective dosage for shortest duration consistent with individual patient treatment goals.
IMs might have a higher than normal risk of adverse events especially in individuals with other factors affecting clearance of these drugs such as hepatic impairment or advanced age.

Pitavastatin	Phenotype	Genetic Test	Results	Source/Evidence Level
Livalo Zypitamag	Normal function	SLCO1B1	*1/*1	CPIC A[14]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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

1 CPIC – SLCO1B1 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	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]; Product monograph (actionable)[26]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Normal function	SLCO1B1	*1/*1	CPIC A[14]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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

1 CPIC – SLCO1B1 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	Normal phenotype	G6PD	B (reference)/B (reference)	CPIC A[21]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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

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

Procainamide	Phenotype	Genetic Test	Results	Source/Evidence Level
	Poor metabolizer	NAT2	*6/*6	FDA 2[52]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 FDA PGx Table Section 2 – NAT2 Potential Impact on Safety or Response: Alters systemic parent drug and metabolite concentrations. May result in higher adverse reaction risk.
Note: the FDA Table refers to the gene "Nonspecific (NAT)" which ClinPGx interprets as NAT2.

Prochlorperazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Compro	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.
2. PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Promethazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Phenadoz Promethegan	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.
2. PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Propafenone	Phenotype	Genetic Test	Results	Source/Evidence Level
Rythmol	Normal metabolizer	CYP2D6	*1.011/*41	DPWG[18]; FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1. CYP2D6 alleles do not indicate changes from recommended dose

Propranolol	Phenotype	Genetic Test	Results	Source/Evidence Level
Inderal Innopran	Normal metabolizer	CYP2D6	*1.011/*41	FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Protriptyline	Phenotype	Genetic Test	Results	Source/Evidence Level
Vivactil	Normal metabolizer	CYP2D6	*1.011/*41	FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Quetiapine	Phenotype	Genetic Test	Results	Source/Evidence Level
Seroquel	Normal metabolizer	CYP3A4	*1/*1	DPWG[18]
	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Decreased likelihood of side effects e.g. weight gain and hypertriglyceridemia compared to A/A	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 A/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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

Quinine	Phenotype	Genetic Test	Results	Source/Evidence Level
	Normal	G6PD	B (reference)/B (reference)	Product monograph (actionable)[69]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 FDA Drug Label: No recommended changes or information for this G6PD phenotype in the FDA Drug Label.

Rabeprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Aciphex Pariet	Normal metabolizer	CYP2C19	*1/*1	FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Normal phenotype	G6PD	B (reference)/B (reference)	CPIC A[21]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Normal metabolizer	CYP2D6	*1.011/*41	DPWG[18]; FDA 3[52]
	Decreased prolactin compared to A/A	ANKK1/DRD2 rs1800497	G/G	PharmGKB 3[53,54]
	May have increased time until response	DRD2 rs1799978	T/T	PharmGKB 3[53,54]
	Decreased risk of developing metabolic syndrome	HTR2C rs1414334	G/G	PharmGKB 3[53,54]
	Decreased likelihood of side effects e.g. weight gain and hypertriglyceridemia compared to A/A	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may reduce efficacy

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

1 DPWG: no recommendation for this CYP2D6 phenotype.

1 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the ANKK1/DRD2 rs1800497 G/G genotype and schizophrenia may have decreased prolactin when treated with risperidone as compared to patients with the A/A genotype. Other genetic and clinical factors may also influence risperidone related hyperprolactinemia.

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 A/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 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[14]; FDA 3[52]
	Decreased function	ABCG2 rs2231142	G/T	CPIC A[14]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Increased response compared to A/A	ADRB2 rs1042713	A/G	PharmGKB 2A[53,54]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1. PharmGKB – Clinical Annotation (Level 2A Efficacy): Patients with the ADRB2 rs1042713 A/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 a response to salmeterol.

Seladelpar	Phenotype	Genetic Test	Results	Source/Evidence Level
Livdelzi	Intermediate metabolizer	CYP2C9	*1/*2	Product monograph (actionable)[70]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1. FDA Drug Label: No recommended changes or information for this CYP2C9 phenotype in the FDA Drug Label.

Sertraline	Phenotype	Genetic Test	Results	Source/Evidence Level
Zoloft	Intermediate metabolizer	CYP2B6	*1/*6	CPIC B[11]
	Normal metabolizer	CYP2C19	*1/*1	CPIC A[11]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require a reduced dose

2. Reduced metabolism of sertraline to less active compounds when compared with CYP2B6 normal metabolizers.
2. Normal CYP2C19 metabolism
2. Initiate therapy with recommended starting dose. Consider a slower titration schedule and lower maintenance dose (per CPIC moderate recommendation).

Sevoflurane	Phenotype	Genetic Test	Results	Source/Evidence Level
Sojourn Ultane	Uncertain Malignant Hyperthermia Susceptibility	CACNA1S	Reference/Reference	CPIC A[24]
	Uncertain Malignant Hyperthermia Susceptibility	RYR1	Reference/Reference	CPIC A[24]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: These results do not eliminate the chance that this patient is susceptible to MH (malignant hyperthermia). The genetic cause of about half of all MH survivors, with MH susceptibility confirmed by contracture test, remains unknown.

1 CPIC – Strong Recommendation: Clinical findings, family history, further genetic testing, and other laboratory data should guide use of halogenated volatile anesthetics or depolarizing muscle relaxants.

Sildenafil	Phenotype	Genetic Test	Results	Source/Evidence Level
Viagra Revatio	Less likely to have positive erectile response compared to T/T	GNB3 rs5443	C/T	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may reduce efficacy

2 PharmGKB – Clinical Annotation (Level 3 Efficacy): Patients with the GNB3 rs5443 C/T 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[14]; FDA 2[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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

1 CPIC – SLCO1B1 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	Intermediate metabolizer	CYP2C9	*1/*2	FDA 1[52]; Product monograph (actionable)[41]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require a reduced dose

2 FDA PGx Table Section 1 – CYP2C9 Therapeutic Management Recommendations: Results in higher systemic concentrations. May need to adjust dosage depending on genotype (Refer to FDA labeling for specific dosing recommendations).

1 FDA Drug Label – CYP2C9 Testing Required: After treatment titration, the recommended maintenance dosage is 2 mg taken orally once daily starting on Day 6 for patient with CYP2C9 *1/*1, *1/*2, or *2/*2 genotypes (standard dosing).
Drug-Drug Interactions: strong CYP2C9 inhibitors or dual moderate CYP2C9/strong CYP3A4 inducers are not recommended with siponimod. Monitor for adverse reactions during concomitant use of siponimod with moderate CYP2C9 inhibitors or moderate CYP2C9/CYP3A4 dual inhibitors.

Succinylcholine	Phenotype	Genetic Test	Results	Source/Evidence Level
Anectine Quelicin	Uncertain Malignant Hyperthermia Susceptibility	CACNA1S	Reference/Reference	CPIC A[24]
	Uncertain Malignant Hyperthermia Susceptibility	RYR1	Reference/Reference	CPIC A[24]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: These results do not eliminate the chance that this patient is susceptible to MH (malignant hyperthermia). The genetic cause of about half of all MH survivors, with MH susceptibility confirmed by contracture test, remains unknown.

1 CPIC – Strong Recommendation: Clinical findings, family history, further genetic testing, and other laboratory data should guide use of halogenated volatile anesthetics or depolarizing muscle relaxants.

Sulfamethoxazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Bactrim Septra Sulfatrim	Poor metabolizer	NAT2	*6/*6	FDA 2[52]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 FDA PGx Table Section 2 – NAT2 Potential Impact on Safety or Response for Sulfamethoxazole/Trimethoprim: May result in higher adverse reaction risk.
Note: the FDA Table refers to the gene "Nonspecific (NAT)" which ClinPGx interprets as NAT2.

Sulfasalazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Azulfidine	Poor metabolizer	NAT2	*6/*6	FDA 2[52]
	Normal	G6PD	B (reference)/B (reference)	Product monograph (actionable)[72]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 FDA PGx Table Section 2 – NAT2 Potential Impact on Safety or Response: Results in higher systemic metabolite concentrations and higher adverse reaction risk.
Note: the FDA Table refers to the gene "Nonspecific (NAT)" which ClinPGx interprets as NAT2.

1 FDA Drug Label: No recommended changes or information for this G6PD phenotype in the FDA Drug Label.

Tacrolimus	Phenotype	Genetic Test	Results	Source/Evidence Level
Advagraf Astagraf XL Envarsus XR Prograf Protopic	Poor metabolizer	CYP3A5	*3/*3	CPIC A[10]; FDA 1[52]
	Normal metabolizer	CYP3A4	*1/*1	PharmGKB 2A[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require an increased dose

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	Normal phenotype	G6PD	B (reference)/B (reference)	CPIC A[21]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Normal metabolizer	CYP2D6 (Activity Score)	*1.011/*41	CPIC A[23]; FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: Therapeutic endoxifen concentrations.

1 CPIC – Strong Recommendation: Avoid moderate and strong CYP2D6 inhibitors. Initiate therapy with recommended standard of care dosing (tamoxifen 20 mg/day).

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

Tamsulosin	Phenotype	Genetic Test	Results	Source/Evidence Level
Flomax	Normal metabolizer	CYP2D6	*1.011/*41	FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Temazepam	Phenotype	Genetic Test	Results	Source/Evidence Level
Restoril	Intermediate metabolizer	CYP2C9	*1/*2	Clinical studies[25]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*2 or *3 allele was associated with an increased fall risk among those taking benzodiazepines (HR = 1.35; 95% CI: 1.06-1.72). Heterozygous allele carriers had a fall risk of HR = 1.30; 95% CI: 1.05-1.61, and homozygous allele carriers HR = 1.91; 95% CI: 1.23-2.96. 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	Intermediate metabolizer (AS 1.5)	CYP2C9	*1/*2	CPIC A[51]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: Mildly reduced metabolism.

1 CPIC – Moderate Recommendation: Initiate therapy with recommended starting dose. In accordance with the prescribing information, use the lowest effective dosage for shortest duration consistent with individual patient treatment goals.
IMs might have a higher than normal risk of adverse events especially in individuals with other factors affecting clearance of these drugs such as hepatic impairment or advanced age.

Tetrabenazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Austedo Nitoman Xenazine	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Thioguanine	Phenotype	Genetic Test	Results	Source/Evidence Level
Lanvis	Normal metabolizer	TPMT	*1/*1	CPIC A[34]; FDA 1[52]
	Normal metabolizer	NUDT15	*1/*1	CPIC A[34]; FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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

1 CPIC – Strong Recommendation: Initiate therapy with standard starting dose of thioguanine (e.g., 40 mg/m2/day for malignancy). During therapy, adjust the doses of myelosuppressive agents, as per standard clinical practice. It usually takes at least 2 weeks of stable dosing to reach steady state after each dose adjustment.
Standard starting doses may vary by patient characteristics and/or treatment regimens and according to local clinical practice.

Thioridazine	Phenotype	Genetic Test	Results	Source/Evidence Level
	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]
	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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.

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

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Thiothixene	Phenotype	Genetic Test	Results	Source/Evidence Level
	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Tolterodine	Phenotype	Genetic Test	Results	Source/Evidence Level
Detrol	Normal metabolizer	CYP2D6	*1.011/*41	FDA 2[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Tramadol	Phenotype	Genetic Test	Results	Source/Evidence Level
Conzip Durela Ralivia Ultram Zytram XL	Normal metabolizer	CYP2D6	*1.011/*41	CPIC A[15]; FDA 1[52]; FDA 2[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – Implication: Expected O-desmethyltramadol (active metabolite) formation.

1 CPIC – Strong Recommendation: Use tramadol label recommended age specific or weight-specific dosing.

Triazolam	Phenotype	Genetic Test	Results	Source/Evidence Level
Halcion	Intermediate metabolizer	CYP2C9	*1/*2	Clinical studies[25]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 Meta-Analysis: 11,485 participants ≥55 years – CYP2C9*2 or *3 allele was associated with an increased fall risk among those taking benzodiazepines (HR = 1.35; 95% CI: 1.06-1.72). Heterozygous allele carriers had a fall risk of HR = 1.30; 95% CI: 1.05-1.61, and homozygous allele carriers HR = 1.91; 95% CI: 1.23-2.96. 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
	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Trimipramine	Phenotype	Genetic Test	Results	Source/Evidence Level
Surmontil	Normal metabolizer	CYP2D6	*1.011/*41	CPIC B[27]; FDA 3[52]
	Normal metabolizer	CYP2C19	*1/*1	CPIC B[27]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CPIC – CYP2D6 Implication: Normal metabolism of TCAs.

1 CPIC – CYP2C19 Implication: Normal metabolism of tertiary amines.

1 CPIC – Optional Recommendation: Initiate therapy with recommended starting dose. Patients may receive an initial low dose of TCAs, 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.

Valbenazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Ingrezza	Normal metabolizer	CYP2D6	*1.011/*41	FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 CYP2D6 alleles do not indicate changes from recommended dose

Venlafaxine	Phenotype	Genetic Test	Results	Source/Evidence Level
Effexor XR	Normal metabolizer	CYP2D6	*1.011/*41	CPIC B[11]; FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

1 Normal CYP2D6 metabolism

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

Viloxazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Qelbree	Normal metabolizer	CYP2D6	*1.011/*41	FDA 3[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

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	Normal metabolizer	CYP2C19	*1/*1	CPIC A[38]; FDA 2[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

CPIC – Implication: Normal voriconazole metabolism.

CPIC – Strong Recommendation: Initiate therapy with recommended standard of care dosing
Notes (Adult & Pediatrics): 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.

Vortioxetine	Phenotype	Genetic Test	Results	Source/Evidence Level
Trintellix	Normal metabolizer	CYP2D6	*1.011/*41	CPIC A[11]; FDA 1[52]

Drug-Gene Interactions:

Mild or no drug-gene interaction: no PGx-based action; standard precautions apply

Normal CYP2D6 metabolism

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

Warfarin	Phenotype	Genetic Test	Results	Source/Evidence Level
Coumadin Jantoven	Intermediate metabolizer	CYP2C9	*1/*2	CPIC A[29]; FDA 1[52]
	Low warfarin sensitivity (no change in PGx-related dose requirement)	VKORC1 rs9923231	G/G	CPIC A[29]; FDA 1[52]
	Reduced response to warfarin (increased PGx-related dose requirement for patients with non-African ancestry)	CYP4F2 rs2108622	C/T	CPIC A[29]; FDA 1[52]
	Typical response to warfarin (no change in PGx-related dose requirement)	CYP2C rs12777823	G/G	CPIC A[29]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require a reduced dose, may require an increased dose, may reduce efficacy, may increase adverse events

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 calculated dose by 5-10%.

Ziprasidone	Phenotype	Genetic Test	Results	Source/Evidence Level
Geodon Zeldox	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 G is associated with increased risk of tardive dyskinesia, but decreased hyperprolactinemia and/or likelihood of drug toxicity (e.g. parkinsonian side effects, sedation, metabolic, cognitive and sexual side effects) as compared to allele A. *Based on antipsychotic class and not the individual medication, effects may vary.

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Zuclopenthixol	Phenotype	Genetic Test	Results	Source/Evidence Level
Clopixol	Normal metabolizer	CYP2D6	*1.011/*41	DPWG[18]
	Increased risk of tardive dyskinesia	ANKK1/DRD2 rs1800497	G/G	Clinical studies[2,8,55,56]
	Decreased likelihood of metabolic syndrome and/or obesity	HTR2C rs1414334	G/G	Clinical studies[39,40,46]
	Increased likelihood of weight gain compared to C/C	MC4R rs489693	C/A	PharmGKB 3[53,54]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

1 DPWG: no recommendation for this CYP2D6 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.

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

2 PharmGKB – Clinical Annotation (Level 3 Toxicity): Patients with the MC4R rs489693 A/C genotype may have increased likelihood of weight gain when treated with antipsychotics as compared to patients with the C/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.

Table of Available References

Drug	Genetic Test	Sources
Abacavir	HLA-B*57:01	CPIC[35]; FDA[52]
Abrocitinib	CYP2C19	FDA[52]; Product monograph (actionable)[43]
Acetylsalicylic acid	PTGS1 rs10306114	PharmGKB[53,54]
Aducanumab	ApoE	Product monograph (actionable)[9]
Alfentanil	OPRM1 rs1799971	PharmGKB[53,54]
Allopurinol	HLA-B*58:01	CPIC[48]; FDA[52]
Allopurinol	ABCG2 rs2231142	DPWG[18]
Alprazolam	CYP2C9	Clinical studies[25]
Amifampridine	NAT2	FDA 1[52]; Product monograph (actionable)[71]
Amitriptyline	CYP2D6	CPIC[27]; FDA[52]
Amitriptyline	CYP2C19	CPIC[27]
Amoxapine	CYP2D6	FDA[52]
Amphetamine	CYP2D6	FDA[52]
Aripiprazole	CYP2D6	FDA[52]; Product monograph (actionable)[49]
Aripiprazole	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Aripiprazole	HTR2C rs1414334	Clinical studies[39,40,46]
Aripiprazole	MC4R rs489693	PharmGKB[53,54]
Aripiprazole lauroxil	CYP2D6	FDA[52]; Product monograph (actionable)[3]
Aripiprazole lauroxil	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Aripiprazole lauroxil	HTR2C rs1414334	Clinical studies[39,40,46]
Aripiprazole lauroxil	MC4R rs489693	PharmGKB[53,54]
Ascorbic acid	G6PD	Product monograph (actionable)[57]
Asenapine	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Asenapine	HTR2C rs1414334	Clinical studies[39,40,46]
Asenapine	MC4R rs489693	PharmGKB[53,54]
Atazanavir	UGT1A1	CPIC[20]
Atomoxetine	CYP2D6 (Activity Score)	CPIC[12]; FDA[52]
Atorvastatin	SLCO1B1	CPIC[14]; FDA[52]
Atorvastatin	ApoE rs7412	PharmGKB[53,54]
Avatrombopag	CYP2C9	FDA[52]
Avatrombopag	Factor II rs1799963	Product monograph (actionable)[1]
Avatrombopag	Factor V rs6025	Product monograph (actionable)[1]
Azathioprine	TPMT	CPIC[34]; FDA[52]
Azathioprine	NUDT15	CPIC[34]; FDA[52]
Brexpiprazole	CYP2D6	FDA[52]; Product monograph (actionable)[4]
Brexpiprazole	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Brexpiprazole	HTR2C rs1414334	Clinical studies[39,40,46]
Brexpiprazole	MC4R rs489693	PharmGKB[53,54]
Brivaracetam	CYP2C19	FDA[52]
Bromazepam	CYP2C9	Clinical studies[25]
Buprenorphine	CYP3A4 rs2740574	PharmGKB[53,54]
Bupropion	ANKK1/DRD2 rs1800497	PharmGKB[53,54]

Drug	Genetic Test	Sources
Capecitabine	DPYD	CPIC[5]; FDA[52]
Carbamazepine	HLA-A*31:01	CPIC[45]; FDA[52]
Carbamazepine	HLA-B*15:02	CPIC[45]; FDA[52]
Cariprazine	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Cariprazine	HTR2C rs1414334	Clinical studies[39,40,46]
Cariprazine	MC4R rs489693	PharmGKB[53,54]
Carisoprodol	CYP2C19	FDA[52]
Carvedilol	CYP2D6	FDA[52]
Celecoxib	CYP2C9	CPIC[51]; FDA[52]
Cevimeline	CYP2D6	FDA[52]
Chlordiazepoxide	CYP2C9	Clinical studies[25]
Chloroquine	G6PD	Product monograph (actionable)[58]
Chlorpromazine	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Chlorpromazine	HTR2C rs1414334	Clinical studies[39,40,46]
Chlorpromazine	MC4R rs489693	PharmGKB[53,54]
Citalopram	CYP2C19	CPIC[11]; FDA[52]
Clobazam	CYP2C19	FDA[52]; Product monograph (actionable)[33]
Clobazam	CYP2C9	Clinical Studies[25]
Clomipramine	CYP2D6	CPIC[27]; FDA[52]
Clomipramine	CYP2C19	CPIC[27]
Clonazepam	CYP2C9	Clinical studies[25]
Clopidogrel	CYP2C19	CPIC[31]; FDA[52]
Clorazepate	CYP2C9	Clinical studies[25]
Clozapine	CYP2D6	FDA[52]
Clozapine	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Clozapine	HTR2C rs1414334	PharmGKB[53,54]
Clozapine	MC4R rs489693	PharmGKB[53,54]
Codeine	CYP2D6	CPIC[15]; FDA[52]
Cyclosporine	CYP3A5	PharmGKB[53,54]
Dabrafenib	G6PD	Product monograph (actionable)[59]
Dapsone	G6PD	CPIC[21]
Darifenacin	CYP2D6	FDA[52]
Daunorubicin	SLC28A3	PharmGKB 2B[60]
Desflurane	CACNA1S	CPIC[24]
Desflurane	RYR1	CPIC[24]
Desipramine	CYP2D6	CPIC[27]; FDA[52]
Deuruxolitinib	CYP2C9	Product monograph (actionable)[61]
Deutetrabenazine	CYP2D6	FDA[52]
Dexlansoprazole	CYP2C19	CPIC[32]; FDA[52]
Dextromethorphan/ Bupropion	CYP2D6	Product monograph (actionable)[7]
Dextromethorphan/ Quinidine	CYP2D6	Product monograph (actionable)[6]
Diazepam	CYP2C19	FDA[52]
Diazepam	CYP2C9	Clinical Studies[25]
Dolutegravir	UGT1A1	FDA 3[52]
Donanemab	ApoE	Product monograph (actionable)[62]
Donepezil	CYP2D6	FDA[52]
Doxepin	CYP2D6	CPIC[27]; FDA[52]
Doxepin	CYP2C19	CPIC[27]; FDA[52]

Drug	Genetic Test	Sources
Doxorubicin	SLC28A3	PharmGKB 2B[60]
Dronabinol	CYP2C9	FDA[52]
Efavirenz	CYP2B6	CPIC[16]; FDA[52]
Elagolix	SLCO1B1	FDA[52]
Eliglustat	CYP2D6	FDA[52]; Product monograph (actionable)[22]
Eltrombopag	Factor V rs6025	Product monograph (actionable)[42]
Erdafitinib	CYP2C9	FDA[52]
Escitalopram	CYP2C19	CPIC[11]; FDA[52]
Esomeprazole	CYP2C19	FDA[52]
Etanercept	TNF-alpha rs1800629	PharmGKB[53,54]
Fentanyl	OPRM1 rs1799971	PharmGKB[53,54]
Fesoterodine	CYP2D6	FDA[52]
Flecainide	CYP2D6	DPWG[18]
Flibanserin	CYP2C19	FDA[52]
Fluorouracil	DPYD	CPIC[5]; FDA[52]
Fluoxetine	CYP2D6	Product monograph (actionable)[13]
Flupentixol	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Flupentixol	HTR2C rs1414334	Clinical studies[39,40,46]
Flupentixol	MC4R rs489693	PharmGKB[53,54]
Fluphenazine	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Fluphenazine	HTR2C rs1414334	Clinical studies[39,40,46]
Fluphenazine	MC4R rs489693	PharmGKB[53,54]
Flurazepam	CYP2C9	Clinical studies[25]
Flurbiprofen	CYP2C9	CPIC[51]; FDA[52]
Flutamide	G6PD	Product monograph (actionable)[63]
Fluvastatin	CYP2C9	CPIC[14]
Fluvastatin	SLCO1B1	CPIC[14]
Fluvoxamine	CYP2D6	CPIC[11]; FDA[52]
Fosphenytoin	HLA-B*15:02	CPIC[30]; FDA[52]
Fosphenytoin	CYP2C9	CPIC[30]; FDA[52]
Galantamine	CYP2D6	FDA[52]
Gefitinib	CYP2D6	FDA[52]
Glimepiride	G6PD	Product monograph (actionable)[64]
Glipizide	G6PD	Product monograph (actionable)[65]
Glyburide	G6PD	Product monograph (actionable)[66]
Haloperidol	CYP2D6	DPWG[18]
Haloperidol	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Haloperidol	HTR2C rs1414334	Clinical studies[39,40,46]
Haloperidol	MC4R rs489693	PharmGKB[53,54]
Halothane	CACNA1S	CPIC[24]
Halothane	RYR1	CPIC[24]
Hydralazine	NAT2	CPIC A[67]; FDA 3[52]
Hydrocodone	CYP2D6	CPIC[15]
Hydroxychloroquine	G6PD	Product monograph (actionable)[68]
Ibuprofen	CYP2C9	CPIC[51]; FDA[52]
Iloperidone	CYP2D6	FDA[52]
Iloperidone	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Iloperidone	HTR2C rs1414334	Clinical studies[39,40,46]

Drug	Genetic Test	Sources
Iloperidone	MC4R rs489693	PharmGKB[53,54]
Imipramine	CYP2D6	CPIC[27]; FDA[52]
Imipramine	CYP2C19	CPIC[27]
Irinotecan	UGT1A1	DPWG[18]; FDA[52]; Product monograph (actionable)[28,44]
Isoflurane	CACNA1S	CPIC[24]
Isoflurane	RYR1	CPIC[24]
Isoniazid	NAT2	FDA 2[52]
Lamotrigine	HLA-B*15:02	DPWG[18]
Lamotrigine	HLA-B*58:01	PharmGKB[53,54]
Lansoprazole	CYP2C19	CPIC[32]; FDA[52]
Lecanemab	ApoE	Product monograph (actionable)[19]
Lofexidine	CYP2D6	FDA[52]
Lorazepam	CYP2C9	Clinical studies[25]
Lorazepam	UGT2B15	PharmGKB 3[60]
Lovastatin	SLCO1B1	CPIC[14]
Loxapine	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Loxapine	HTR2C rs1414334	Clinical studies[39,40,46]
Loxapine	MC4R rs489693	PharmGKB[53,54]
Lurasidone	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Lurasidone	HTR2C rs1414334	Clinical studies[39,40,46]
Lurasidone	MC4R rs489693	PharmGKB[53,54]
Lusutrombopag	Factor II rs1799963	Product monograph (actionable)[50]
Lusutrombopag	Factor V rs6025	Product monograph (actionable)[50]
Mavacamten	CYP2C19	FDA[52]
Meclizine	CYP2D6	FDA[52]
Meloxicam	CYP2C9	CPIC[51]; FDA[52]
Mercaptopurine	TPMT	CPIC[34]; FDA[52]
Mercaptopurine	NUDT15	CPIC[34]; FDA[52]
Methadone	CYP2B6	CPIC[47]; PharmGKB[53,54]
Methotrexate	MTHFR rs1801133	PharmGKB[53,54]
Methotrimeprazine	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Methotrimeprazine	HTR2C rs1414334	Clinical studies[39,40,46]
Methotrimeprazine	MC4R rs489693	PharmGKB[53,54]
Methoxyflurane	CACNA1S	CPIC[24]
Methoxyflurane	RYR1	CPIC[24]
Methylene blue	G6PD	CPIC[21]
Methylphenidate	ADRA2A rs1800544	PharmGKB[53,54]
Methylphenidate	COMT rs4680	PharmGKB[53,54]
Metoclopramide	CYP2D6	FDA[52]
Metoprolol	CYP2D6	CPIC[17]
Mirabegron	CYP2D6	FDA[52]
Mirtazapine	CYP2D6	DPWG[18]; PharmGKB 2A[53,54]
Molindone	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Molindone	HTR2C rs1414334	Clinical studies[39,40,46]
Molindone	MC4R rs489693	PharmGKB[53,54]
Morphine	OPRM1 rs1799971	PharmGKB[53,54]
Nateglinide	CYP2C9	FDA[52]
Nebivolol	CYP2D6	FDA[52]

Drug	Genetic Test	Sources
Nevirapine	CYP2B6 rs28399499	PharmGKB[53,54]
Nevirapine	CYP2B6 rs3745274	PharmGKB[53,54]
Nicotine replacement therapy	ANKK1/DRD2 rs1800497	PharmGKB[53,54]
Nitrazepam	CYP2C9	Clinical studies[25]
Nitrofurantoin	G6PD	CPIC[21]
Nortriptyline	CYP2D6	CPIC[27]; FDA[52]
Olanzapine	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Olanzapine	DRD2 rs1799978	PharmGKB[53,54]
Olanzapine	HTR2C rs1414334	Clinical studies[39,40,46]
Olanzapine	MC4R rs489693	PharmGKB[53,54]
Oliceridine	CYP2D6	FDA[52]
Omeprazole	CYP2C19	CPIC[32]; FDA[52]
Ondansetron	CYP2D6	CPIC[37]
Oral contraceptives	Factor II rs1799963	PharmGKB[53,54]
Oral contraceptives	Factor V rs6025	PharmGKB[53,54]
Oxazepam	CYP2C9	Clinical studies[25]
Oxcarbazepine	HLA-B*15:02	CPIC[45]; FDA[52]
Oxycodone	CYP2D6	CPIC[15]; PharmGKB[53,54]
Paliperidone	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Paliperidone	HTR2C rs1414334	Clinical studies[39,40,46]
Paliperidone	MC4R rs489693	PharmGKB[53,54]
Pantoprazole	CYP2C19	CPIC[32]; FDA[52]
Paroxetine	CYP2D6	CPIC[11]; FDA[52]
Pazopanib	HLA-B*57:01	FDA[52]
PEG-interferon alpha	IFNL3	PharmGKB 1B[60]; PharmGKB 2A[60]
Pegloticase	G6PD	CPIC[21]
Perphenazine	CYP2D6	FDA[52]
Perphenazine	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Perphenazine	HTR2C rs1414334	Clinical studies[39,40,46]
Perphenazine	MC4R rs489693	PharmGKB[53,54]
Phenytoin	HLA-B*15:02	CPIC[30]; FDA[52]
Phenytoin	CYP2C9	CPIC[30]; FDA[52]
Pimozide	CYP2D6	FDA[52]
Pimozide	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Pimozide	HTR2C rs1414334	Clinical studies[39,40,46]
Pimozide	MC4R rs489693	PharmGKB[53,54]
Piroxicam	CYP2C9	CPIC[51]; FDA[52]
Pitavastatin	SLCO1B1	CPIC[14]
Pitolisant	CYP2D6	FDA[52]; Product monograph (actionable)[26]
Pravastatin	SLCO1B1	CPIC[14]
Primaquine	G6PD	CPIC[21]
Procainamide	NAT2	FDA 2[52]
Prochlorperazine	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Prochlorperazine	HTR2C rs1414334	Clinical studies[39,40,46]
Prochlorperazine	MC4R rs489693	PharmGKB[53,54]
Promethazine	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Promethazine	HTR2C rs1414334	Clinical studies[39,40,46]
Promethazine	MC4R rs489693	PharmGKB[53,54]

Drug	Genetic Test	Sources
Propafenone	CYP2D6	DPWG[18]; FDA[52]
Propranolol	CYP2D6	FDA[52]
Protriptyline	CYP2D6	FDA[52]
Quetiapine	CYP3A4	DPWG[18]
Quetiapine	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Quetiapine	HTR2C rs1414334	Clinical studies[39,40,46]
Quetiapine	MC4R rs489693	PharmGKB[53,54]
Quinine	G6PD	Product monograph (actionable)[69]
Rabeprazole	CYP2C19	FDA[52]
Rasburicase	G6PD	CPIC[21]
Risperidone	CYP2D6	DPWG[18]; FDA[52]
Risperidone	ANKK1/DRD2 rs1800497	PharmGKB[53,54]
Risperidone	DRD2 rs1799978	PharmGKB[53,54]
Risperidone	HTR2C rs1414334	PharmGKB[53,54]
Risperidone	MC4R rs489693	PharmGKB[53,54]
Rosuvastatin	SLCO1B1	CPIC[14]; FDA[52]
Rosuvastatin	ABCG2 rs2231142	CPIC[14]
Salmeterol	ADRB2 rs1042713	PharmGKB[53,54]
Seladelpar	CYP2C9	Product monograph (actionable)[70]
Sertraline	CYP2B6	CPIC[11]
Sertraline	CYP2C19	CPIC[11]
Sevoflurane	CACNA1S	CPIC[24]
Sevoflurane	RYR1	CPIC[24]
Sildenafil	GNB3 rs5443	PharmGKB[53,54]
Simvastatin	SLCO1B1	CPIC[14]; FDA[52]
Siponimod	CYP2C9	FDA[52]; Product monograph (actionable)[41]
Succinylcholine	CACNA1S	CPIC[24]
Succinylcholine	RYR1	CPIC[24]
Sulfamethoxazole	NAT2	FDA 2[52]
Sulfasalazine	NAT2	FDA 2[52]
Sulfasalazine	G6PD	Product monograph (actionable)[72]
Tacrolimus	CYP3A5	CPIC[10]; FDA[52]
Tacrolimus	CYP3A4	PharmGKB[53,54]
Tafenoquine	G6PD	CPIC[21]
Tamoxifen	CYP2D6 (Activity Score)	CPIC[23]; FDA[52]
Tamsulosin	CYP2D6	FDA[52]
Temazepam	CYP2C9	Clinical studies[25]
Tenoxicam	CYP2C9	CPIC[51]
Tetrabenazine	CYP2D6	FDA[52]
Thioguanine	TPMT	CPIC[34]; FDA[52]
Thioguanine	NUDT15	CPIC[34]; FDA[52]
Thioridazine	CYP2D6	FDA[52]
Thioridazine	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Thioridazine	HTR2C rs1414334	Clinical studies[39,40,46]
Thioridazine	MC4R rs489693	PharmGKB[53,54]
Thiothixene	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Thiothixene	HTR2C rs1414334	Clinical studies[39,40,46]
Thiothixene	MC4R rs489693	PharmGKB[53,54]

Drug	Genetic Test	Sources
Tolterodine	CYP2D6	FDA[52]
Tramadol	CYP2D6	CPIC[15]; FDA[52]
Triazolam	CYP2C9	Clinical studies[25]
Trifluoperazine	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Trifluoperazine	HTR2C rs1414334	Clinical studies[39,40,46]
Trifluoperazine	MC4R rs489693	PharmGKB[53,54]
Trimipramine	CYP2D6	CPIC[27]; FDA[52]
Trimipramine	CYP2C19	CPIC[27]
Valbenazine	CYP2D6	FDA[52]
Venlafaxine	CYP2D6	CPIC[11]; FDA[52]
Viloxazine	CYP2D6	FDA[52]
Voriconazole	CYP2C19	CPIC[38]; FDA[52]
Vortioxetine	CYP2D6	CPIC[11]; FDA[52]
Warfarin	CYP2C9	CPIC[29]; FDA[52]
Warfarin	VKORC1 rs9923231	CPIC[29]; FDA[52]
Warfarin	CYP4F2 rs2108622	CPIC[29]; FDA[52]
Warfarin	CYP2C rs12777823	CPIC[29]
Ziprasidone	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Ziprasidone	HTR2C rs1414334	Clinical studies[39,40,46]
Ziprasidone	MC4R rs489693	PharmGKB[53,54]
Zuclopenthixol	CYP2D6	DPWG[18]
Zuclopenthixol	ANKK1/DRD2 rs1800497	Clinical studies[2,8,55,56]
Zuclopenthixol	HTR2C rs1414334	Clinical studies[39,40,46]
Zuclopenthixol	MC4R rs489693	PharmGKB[53,54]

References

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

Laboratory Report

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

Gene	rsID	Allele Information	Allele Information Reference	Result
ABCG2	rs2231142	c.421C>A	NM_004827.3	G/T
ADRB2	rs1042713	c.46G>A	NM_000024.6	A/G
ADRA2A	rs1800544	g.111076745G>C	NC_000010.11	C/C
ANKK1/DRD2	rs1800497	c.2137G>A	NM_178510.2	G/G (C/C)[1]
APOE	rs429358	c.388T>C	NM_000041.4	T/T
APOE	rs7412	c.526C>T	NM_000041.4	C/C
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	A/A
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/C
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/T
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

Gene	rsID	Allele Information	Allele Information Reference	Result
CYP2C9	rs57505750	c.980T>C	NM_000771.4	T/T
CYP2C9	rs72558189	c.374G>A	NM_000771.4	G/G
CYP2D6	rs1065852	c.100C>T	NM_000106.5	G/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	T/C
CYP2D6	rs3892097	c.506-1G>A	NM_000106.5	C/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/C
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	C/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
CYP3A4	rs2740574	g.99784473C>T	NC_000007.14	T/T (A/A)[1]
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/A
DPYD	rs2297595	c.496A>G	NM_000110.4	T/T
DPYD	rs1801159	c.1627A>G	NM_000110.4	T/T
DPYD	rs3918290	g.97450058C>T	NC_000001.11	C/C

Gene	rsID	Allele Information	Allele Information Reference	Result
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
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	A/A
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

Gene	rsID	Allele Information	Allele Information Reference	Result
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
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	T/C
GRIK4	rs1954787	c.83-10039T>C	NM_014619.2	T/C
HCP5	rs2395029	c.*568T>G	NM_006674.3	T/G
HLAA	rs1061235	c.*66A>T	NM_002116.8	A/T
HLAB	rs144012689	c.1012+104A>T	NM_005514.8	T/T
HTR2A	rs7997012	c.614-2211T>C	NM_000621.5	A/G
HTR2C	rs1414334	c.551-3008C>G	NM_001256760.3	G/G
IFNL3	rs12979860	c.151-152G>A	NM_001276254.2	C/C
NAT2	rs72466456	g.18400032T>C	NC_000008.11	T/T
NAT2	rs45477599	g.18400073T>A	NC_000008.11	T/T
NAT2	rs72554615	g.18400114T>C	NC_000008.11	T/T
NAT2	rs72466457	g.18400155G>T	NC_000008.11	G/G
NAT2	rs1805158	g.18400193C>T	NC_000008.11	C/C
NAT2	rs1801279	g.18400194G>A	NC_000008.11	G/G
NAT2	rs72466458	g.18400206G>A	NC_000008.11	G/G
NAT2	rs72466459	g.18400231C>T	NC_000008.11	C/C
NAT2	rs1041983	g.18400285C>T	NC_000008.11	T/T
NAT2	rs1801280	g.18400344T>C	NC_000008.11	T/T
NAT2	rs45532639	g.18400348C>T	NC_000008.11	C/C
NAT2	rs4986996	g.18400367G>A	NC_000008.11	G/G
NAT2	rs12720065	g.18400406C>G	NC_000008.11	C/C
NAT2	rs72466460	g.18400461C>T	NC_000008.11	C/C
NAT2	rs139351995	g.18400475A>C	NC_000008.11	A/A
NAT2	rs1799929	g.18400484C>T	NC_000008.11	C/C
NAT2	rs72554617	g.18400502G>A	NC_000008.11	G/G
NAT2	rs369500066	g.18400521A>G	NC_000008.11	A/A
NAT2	rs79050330	g.18400581C>T	NC_000008.11	C/C
NAT2	rs1799930	g.18400593G>A	NC_000008.11	A/A
NAT2	rs72466461	g.18400603A>G	NC_000008.11	A/A
NAT2	rs45618543	g.18400612G>T	NC_000008.11	G/G
NAT2	rs45607939	g.18400616A>T	NC_000008.11	A/A
NAT2	rs138707146	g.18400641C>T	NC_000008.11	C/C
NAT2	rs45518335	g.18400686C>T	NC_000008.11	C/C
NAT2	rs56011192	g.18400762C>T	NC_000008.11	C/C

Gene	rsID	Allele Information	Allele Information Reference	Result
NAT2	rs55700793	g.18400769A>G	NC_000008.11	A/A
NAT2	rs1208	g.18400806G>A	NC_000008.11	A/A
NAT2	rs56393504	g.18400841G>A	NC_000008.11	G/G
NAT2	rs56054745	g.18400848A>G	NC_000008.11	A/A
NAT2	rs1799931	g.18400860G>A	NC_000008.11	G/G
MTHFR	rs1801133	c.665C>T	NM_005957.5	A/A (T/T)[1]
MC4R	rs489693	g.57882787C>A	NC_000018.9	A/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
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>T	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
SLC28A3	rs7853758	g.84286011G>A	NC_000009.12	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

Gene	rsID	Allele Information	Allele Information Reference	Result
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
UGT2B15	rs1902023	g.68670366A>C	NC_000004.12	A/C (T/G)[1]
VKORC1	rs9923231	g.31096368C>T	NC_000016.10	G/G (C/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.

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, PhD, Laboratory Director

27/Aug/2026

Date of Signature

Summary of Genetic and Phenotype Data

HLA

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

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	Activity Score	Phenotype Result OR Genotype Explanation
ABCG2 (rs2231142)	G/T	N/A	Decreased Function
APOE	E3/E3	N/A	E3 Homozygous
CACNA1S	Reference/Reference	N/A	Uncertain Malignant Hyperthermia Susceptibility with Potent Volatile Anesthetic Agents and Succinylcholine
CYP2C (rs12777823)	G/G	N/A	Typical response to warfarin (no change in PGx-related dose requirement)
CYP2B6	*1/*6	N/A	Intermediate Metabolizer
CYP2D6	*1.011/*41	1.25	Normal Metabolizer
CYP2C9	*1/*2	1.5	Intermediate Metabolizer
CYP2C19	*1/*1	N/A	Normal Metabolizer
CYP3A4	*1/*1	N/A	Normal Metabolizer
CYP3A5	*3/*3	N/A	Poor Metabolizer
CYP4F2 (rs2108622)	C/T	N/A	Reduced response to warfarin (increased PGx-related dose requirement for patients with non-African ancestry)
DPYD	*1/*1	2	Normal Metabolizer
G6PD	B (reference)/B (reference)	N/A	Normal
NAT2	*6/*6	N/A	Poor Metabolizer
NUDT15	*1/*1	N/A	Normal Metabolizer
RYR1	Reference/Reference	N/A	Uncertain Malignant Hyperthermia Susceptibility with Potent Volatile Anesthetic Agents and Succinylcholine
SLCO1B1	*1/*1	N/A	Normal Function
TPMT	*1/*1	N/A	Normal Metabolizer
UGT1A1	*1/*1	N/A	Normal Metabolizer
VKORC1 (rs9923231)	G/G	N/A	Low warfarin sensitivity (no change in PGx-related dose requirement)