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). Please refer to the Methods, Limitations, and Liability Disclaimer at the end of this report.

Medication Summary

The Medication Summary is a list of medications with evidence for the use of pharmacogenetic information, organized by their therapeutic area. Medications are further organized based on drug-gene interactions. Health care providers should consider the information contained in the Medication Report before making any clinical or therapeutic decisions.

1 Mild or no known interaction
2 Moderate gene-drug interaction
3 Serious drug-gene interaction: evaluate and consider alternative medications

Analgesia

1
Carisoprodol
Hydrocodone

2
Alfentanil
Celecoxib
Fentanyl
Flurbiprofen
Ibuprofen
Meloxicam
Morphine
Oliceridine
Piroxicam
Tenoxicam
Venlafaxine

3
Amitriptyline
Codeine
Desipramine
Imipramine
Nortriptyline
Tramadol

Autoimmune

1
Tacrolimus

2
Cyclosporine

...Autoimmune

2
Methotrexate
Siponimod

Cancer

1
Erdafitinib

2
Gefitinib
Methotrexate

3
Tamoxifen

Cardiovascular

1
Atorvastatin
Clopidogrel
Lovastatin
Mavacamten
Nebivolol
Pitavastatin
Pravastatin
Propranolol
Rosuvastatin
Simvastatin

2
Carvedilol
Flecainide
Fluvastatin
Propafenone
Warfarin

...Cardiovascular

3
Metoprolol

Endocrinology

1
Nateglinide

Gastroenterology

1
Esomeprazole
Ondansetron
Rabeprazole

2
Dexlansoprazole
Dronabinol
Lansoprazole
Meclizine
Methotrexate
Metoclopramide
Omeprazole
Pantoprazole

Infection

1
Efavirenz
Voriconazole

Mental Health

1
Amoxapine
Citalopram
Diazepam
Escitalopram

...Mental Health

1
Methylphenidate
Nicotine replacement therapy
Protriptyline
Quetiapine
Sertraline
Viloxazine

2
Amphetamine
Aripiprazole
Aripiprazole lauroxil
Atomoxetine
Brexpiprazole
Bupropion
Clozapine
Fluvoxamine
Haloperidol
Iloperidone
Lofexidine
Paroxetine
Perphenazine
Pimozide
Risperidone
Venlafaxine
Vortioxetine
Zuclopenthixol

3
Amitriptyline
Clomipramine

...Mental Health

3

Desipramine
Doxepin
Imipramine
Nortriptyline
Thioridazine
Trimipramine

Neurology

1

Brivaracetam
Clobazam
Diazepam
Donepezil
Galantamine
Propranolol

2

Deutetrabenazine
Fosphenytoin
Phenytoin
Pitolisant
Tetrabenazine
Valbenazine
Venlafaxine

3

Amitriptyline
Metoprolol

Rheumatology

2

Celecoxib
Flurbiprofen
Ibuprofen
Meloxicam
Methotrexate
Piroxicam
Tenoxicam

Urology

1

Darifenacin
Fesoterodine
Mirabegron
Tamsulosin

2

Tolterodine

Other

1

Abrocitinib
Avatrombopag
Elagolix
Eltrombopag
Flibanserin
Lusutrombopag
Oral contraceptives

2

Cevimeline

3

Eliglustat

Overview

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

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

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

TreatGx

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

ReviewGx

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

Components of the Medication Report

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

Example:

Generic Name → Codeine

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

Potential Kidney or Liver Interaction → TreatGx ReviewGx

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

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

3 Avoid Codeine use

Source/Evidence 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.

Abrocitinib	Phenotype	Genetic Test	Results	Source/Evidence
Cibinqo ReviewGx	Normal metabolizer	CYP2C19	*1/*1	FDA 1[34]; Product monograph (actionable)[27]
	Implication: FDA PGx Table: no information for this phenotype.			

Alfentanil	Phenotype	Genetic Test	Results	Source/Evidence
Alfenta ReviewGx	Increased analgesic response	OPRM1 rs1799971	A/A	PharmGKB 3
	Implication: 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.			

Amitriptyline	Phenotype	Genetic Test	Results	Source/Evidence
Elavil Levate TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	CPIC A[16]; FDA 3[34]
	Normal metabolizer	CYP2C19	*1/*1	CPIC A[16]
	Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Amitriptyline to less active compounds Higher plasma concentrations of active drug may increase the risk of adverse drug reactions			
	3 Avoid Amitriptyline use. If use is warranted, consider a reduction of recommended starting dose (per CPIC strong recommendation). Refer to TreatGx for alternatives and specific dosing recommendations.			

Amoxapine	Phenotype	Genetic Test	Results	Source/Evidence
ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 3[34]
	Implication: FDA PGx Table Section 3 – Potential Impact on Pharmacokinetic Properties Only: May alter systemic concentrations.			

Amphetamine	Phenotype	Genetic Test	Results	Source/Evidence
Adzenys TreatG ReviewG	Poor metabolizer	CYP2D6	*4/*4	FDA 1[34]

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Amphetamine to less active compounds

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

2 Consider a lower starting dose or use an alternative drug not predominantly metabolized by CYP2D6

2 This drug has an FDA therapeutic recommendation, refer to drug monograph or FDA labelling for dosing recommendations

Aripiprazole	Phenotype	Genetic Test	Results	Source/Evidence
Abilify Aristada TreatG ReviewG	Poor metabolizer	CYP2D6	*4/*4	DPWG[10]; FDA 1[34]

Implication: 2 FDA PGx Table Section 1 – CYP2D6 Therapeutic Management Recommendations: Results in higher systemic concentrations and higher adverse reaction risk. Dosage adjustment is recommended. Refer to FDA labeling for specific dosing recommendations.

Aripiprazole lauroxil	Phenotype	Genetic Test	Results	Source/Evidence
Aristada TreatG ReviewG	Poor metabolizer	CYP2D6	*4/*4	FDA 1[34]

Implication: 2 FDA PGx Table Section 1 – CYP2D6 Therapeutic Management Recommendations: Results in higher systemic concentrations. Dosage adjustment is recommended. Refer to FDA labeling for specific dosing recommendations.

Atomoxetine	Phenotype	Genetic Test	Results	Source/Evidence
Strattera TreatG ReviewG	Poor metabolizer	CYP2D6 (Activity Score)	*4/*4	CPIC A[6]; FDA 1[34]

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Atomoxetine to less active compounds

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

2 Strong CPIC recommendation: Increase the daily dose only if symptoms fail to improve after 14 days and previous dose is well tolerated, consider obtaining a plasma concentration 2-4 h after dosing. If response is inadequate and concentration is <200 ng/ml, consider a proportional dose increase to achieve a concentration to approach 400 ng/ml.

Atorvastatin	Phenotype	Genetic Test	Results	Source/Evidence
Lipitor TreatG ReviewG	Normal function	SLCO1B1	*1/*1	CPIC A[7]; FDA 3[34]

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

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

Avatrombopag	Phenotype	Genetic Test	Results	Source/Evidence
Doptelet ReviewGx	Intermediate metabolizer	CYP2C9	*1/*3	FDA 3[34]
	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]

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

Product monograph: no change in risk stated for normal Factor II (i.e. Prothrombin 20210A mutation absent).

Product monograph: no change in risk stated for normal Factor V.

Brexpiprazole	Phenotype	Genetic Test	Results	Source/Evidence
Rexulti	Poor metabolizer	CYP2D6	*4/*4	DPWG[10]; FDA 1[34]

Implication: 2 FDA PGx Table Section 1 – CYP2D6 Therapeutic Management Recommendations: Results in higher systemic concentrations. Dosage adjustment is recommended. Refer to FDA labeling for specific dosing recommendations.

TreatGx
ReviewGx

Brivaracetam	Phenotype	Genetic Test	Results	Source/Evidence
Briviact Brivlera	Normal metabolizer	CYP2C19	*1/*1	FDA 1[34]

Implication: CYP2C19 alleles do not indicate changes from recommended dose

ReviewGx

Bupropion	Phenotype	Genetic Test	Results	Source/Evidence
Wellbutrin Zyban	Less likely to quit smoking compared to G/G	ANKK1/DRD2 rs1800497	A/G	PharmGKB 3

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

TreatGx
ReviewGx

Carisoprodol	Phenotype	Genetic Test	Results	Source/Evidence
ReviewGx	Normal metabolizer	CYP2C19	*1/*1	FDA 3[34]

Implication: CYP2C19 alleles do not indicate changes from recommended dose

Carvedilol	Phenotype	Genetic Test	Results	Source/Evidence
Coreg	Poor metabolizer	CYP2D6	*4/*4	FDA 2[34]

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Carvedilol to less active compounds

Higher plasma concentrations of active drug may increase the risk of adverse drug reactions (dizziness)

TreatGx
ReviewGx

2 Data indicate a potential impact on patient safety

Celecoxib	Phenotype	Genetic Test	Results	Source/Evidence
Celebrex	Intermediate metabolizer (AS 1.0)	CYP2C9 (Star Alleles)	*1/*3	CPIC A[33]; FDA 1[34]

TreatGx
ReviewGx

Implication: CYP2C9 intermediate metabolizer with an activity score of 1.0: reduced metabolism of Celecoxib to less active compounds

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

2 Initiate therapy with the lowest recommended dose of Celecoxib

Cevimeline	Phenotype	Genetic Test	Results	Source/Evidence
Evoxac	Poor metabolizer	CYP2D6	*4/*4	FDA 2[34]

ReviewGx

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Cevimeline to less active compounds

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

2 Data indicate a potential impact on patient safety

Citalopram	Phenotype	Genetic Test	Results	Source/Evidence
Celexa	Normal metabolizer	CYP2C19	*1/*1	CPIC A[5]; FDA 1[34]

TreatGx
ReviewGx

Implication: Normal CYP2C19 metabolism

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

Clobazam	Phenotype	Genetic Test	Results	Source/Evidence
Onfi Sympazan	Normal metabolizer	CYP2C19	*1/*1	FDA 1[34]

ReviewGx

Implication: FDA PGx Table: no information for this CYP2C19 phenotype.

Clomipramine	Phenotype	Genetic Test	Results	Source/Evidence
Anafranil	Poor metabolizer	CYP2D6	*4/*4	CPIC B[16]; FDA 3[34]
	Normal metabolizer	CYP2C19	*1/*1	CPIC B[16]

ReviewGx

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Clomipramine to less active compounds
Higher plasma concentrations of active drug may increase the risk of adverse drug reactions

3 Avoid Clomipramine use. If use is warranted, consider a reduction of recommended starting dose (per CPIC optional recommendation). Refer to TreatGx for alternatives and specific dosing recommendations.

Clopidogrel	Phenotype	Genetic Test	Results	Source/Evidence
Plavix	Normal metabolizer	CYP2C19	*1/*1	CPIC A[19]; FDA 1[34]

TreatGx
ReviewGx

Implication: CYP2C19 alleles do not indicate changes from recommended dose

Clozapine	Phenotype	Genetic Test	Results	Source/Evidence
Clozaril Fazaclo ODT Versacloz TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 1[34]

Implication: 2 FDA PGx Table Section 1 – CYP2D6 Therapeutic Management Recommendations: Results in higher systemic concentrations. Dosage reductions may be necessary.

Codeine	Phenotype	Genetic Test	Results	Source/Evidence
Codeine Contin Tylenol with Codeine No. 2/3/4 TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	CPIC A[8]; FDA 1[34]; FDA 2[34]

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Codeine to active metabolite may result in diminished analgesia

3 Avoid Codeine use due to possibility of diminished analgesia. If opioid use is warranted, consider an opioid other than tramadol or codeine (per CPIC strong recommendation). Refer to TreatGx for alternatives and specific dosing recommendations.

Cyclosporine	Phenotype	Genetic Test	Results	Source/Evidence
Neoral Sandimmune ReviewGx	Poor metabolizer	CYP3A5	*3/*3	PharmGKB 3

Implication: 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).

Darifenacin	Phenotype	Genetic Test	Results	Source/Evidence
Enablex TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 3[34]

Implication: CYP2D6 poor metabolizer: reduced metabolism of Darifenacin leads to higher plasma concentrations

There is a potential impact on pharmacokinetic properties. The impact of CYP2D6 variants on the safety of Darifenacin has not been established

Desipramine	Phenotype	Genetic Test	Results	Source/Evidence
Norpramin TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	CPIC B[16]; FDA 3[34]

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Desipramine to less active compounds
Higher plasma concentrations of active drug may increase the risk of adverse drug reactions

3 Avoid Desipramine use due to potential for adverse effects. Consider alternative drug not metabolized by CYP2D6. If use is warranted, consider a reduction of the recommended dose (per CPIC optional recommendation). Refer to TreatGx for alternatives and specific dosing recommendations.

Deutetrabenazine	Phenotype	Genetic Test	Results	Source/Evidence
Austedo	Poor metabolizer	CYP2D6	*4/*4	FDA 1[34]

ReviewGx

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Deutetrabenazine to less active compounds

Higher plasma concentrations of active drug may increase the risk of QT prolongation

2 Consider a reduction of maximum daily dose

2 This drug has an FDA therapeutic recommendation, refer to drug monograph or FDA labelling for dosing recommendations

Dexlansoprazole	Phenotype	Genetic Test	Results	Source/Evidence
Dexilant	Normal metabolizer	CYP2C19	*1/*1	CPIC B[20]; FDA 3[34]

TreatGx
ReviewGx

Implication: 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.

Diazepam	Phenotype	Genetic Test	Results	Source/Evidence
Diastat Valium	Normal metabolizer	CYP2C19	*1/*1	FDA 3[34]

Implication: FDA PGx Table: no information for this CYP2C19 phenotype.

TreatGx
ReviewGx

Donepezil	Phenotype	Genetic Test	Results	Source/Evidence
Aricept	Poor metabolizer	CYP2D6	*4/*4	FDA 3[34]

TreatGx
ReviewGx

Implication: CYP2D6 poor metabolizer: reduced metabolism of Donepezil to less active compounds leads to higher plasma concentrations of active drug

There is a potential impact on pharmacokinetic properties. The impact of CYP2D6 variants on the safety of Donepezil has not been established

Doxepin	Phenotype	Genetic Test	Results	Source/Evidence
Silenor Sinequan	Poor metabolizer	CYP2D6	*4/*4	CPIC B[16]; FDA 3[34]
	Normal metabolizer	CYP2C19	*1/*1	CPIC B[16]; FDA 3[34]

TreatGx
ReviewGx

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Doxepin to less active compounds
Higher plasma concentrations of active drug may increase the risk of adverse drug reactions

3 Avoid Doxepin use. If use is warranted, consider a reduction of recommended starting dose (per CPIC optional recommendation). Refer to TreatGx for alternatives and specific dosing recommendations.

Dronabinol	Phenotype	Genetic Test	Results	Source/Evidence
Marinol Syndros ReviewGx	Intermediate metabolizer	CYP2C9	*1/*3	FDA 1[34]

Implication: CYP2C9 intermediate metabolizer: reduced metabolism of Dronabinol to less active compounds

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
Sustiva ReviewGx	Normal metabolizer	CYP2B6	*1/*1	CPIC A[9]; FDA 2[34]

Implication: CYP2B6 alleles do not indicate changes from recommended dose

Elagolix	Phenotype	Genetic Test	Results	Source/Evidence
Orilissa ReviewGx	Normal function	SLCO1B1	*1/*1	FDA 3[34]

Implication: SLCO1B1 alleles indicate a typical response to Elagolix

Eliglustat	Phenotype	Genetic Test	Results	Source/Evidence
Cerdelga ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 1[34]

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Eliglustat to less active compounds

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

2 Consider reducing eliglustat dose, refer to drug monograph or FDA labelling for dosing recommendations

3 Concurrent use of a mild, moderate or strong CYP3A inhibitor, or use of a strong CYP3A inducer: Avoid Eliglustat use

Eltrombopag	Phenotype	Genetic Test	Results	Source/Evidence
Promacta Revolade ReviewGx	Normal Factor V Leiden	Factor V rs6025	C/C	Product monograph (actionable)[26]

Implication: Product monograph: no change in risk stated for normal Factor V.

Erdafitinib	Phenotype	Genetic Test	Results	Source/Evidence
Balversa ReviewGx	Intermediate metabolizer	CYP2C9 (Star Alleles)	*1/*3	FDA 1[34]

Implication: CYP2C9 alleles do not indicate changes from recommended dose

Escitalopram	Phenotype	Genetic Test	Results	Source/Evidence
Cipralex Lexapro TreatGx ReviewGx	Normal metabolizer	CYP2C19	*1/*1	CPIC A[5]; FDA 3[34]

Implication: Normal CYP2C19 metabolism

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

Esomeprazole	Phenotype	Genetic Test	Results	Source/Evidence
Nexium	Normal metabolizer	CYP2C19	*1/*1	FDA 3[34]
TreatGx ReviewGx	**Implication:** FDA PGx Table: no information for this phenotype.			

Fentanyl	Phenotype	Genetic Test	Results	Source/Evidence
Actiq Duragesic Fentora Sublimaze	Decreased analgesic response	OPRM1 rs1799971	A/A	PharmGKB 3
ReviewGx	**Implication:** 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
Toviaz	Poor metabolizer	CYP2D6	*4/*4	FDA 3[34]
TreatGx ReviewGx	**Implication:** CYP2D6 poor metabolizer: reduced metabolism of Fesoterodine leads to higher plasma concentrations There is a potential impact on pharmacokinetic properties. The impact of CYP2D6 variants on the safety of Fesoterodine has not been established			

Flecainide	Phenotype	Genetic Test	Results	Source/Evidence
Tambocor	Poor metabolizer	CYP2D6	*4/*4	DPWG[10]
TreatGx ReviewGx	**Implication:** CYP2D6 poor metabolizer: greatly reduced metabolism of Flecainide to less active compounds Higher plasma concentrations of active drug may increase the risk of adverse drug reactions 2 Reduce the standard dose by 50%, record electrocardiogram, and monitor plasma concentration			

Flibanserin	Phenotype	Genetic Test	Results	Source/Evidence
Addyi	Normal metabolizer	CYP2C19	*1/*1	FDA 1[34]
ReviewGx	**Implication:** CYP2C19 alleles do not indicate changes from recommended dose			

Flurbiprofen	Phenotype	Genetic Test	Results	Source/Evidence
Ansaid	Intermediate metabolizer (AS 1.0)	CYP2C9 (Star Alleles)	*1/*3	CPIC A[33]; FDA 1[34]

TreatG: ReviewG:

Implication: CYP2C9 intermediate metabolizer with an activity score of 1.0: reduced metabolism of Flurbiprofen to less active compounds

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

2 Initiate therapy with the lowest recommended dose of Flurbiprofen

Fluvastatin	Phenotype	Genetic Test	Results	Source/Evidence
Lescol	Intermediate metabolizer	CYP2C9	*1/*3	CPIC A[7]
	Normal function	SLCO1B1	*1/*1	CPIC A[7]

TreatG: ReviewG:

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

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
Luvox	Poor metabolizer	CYP2D6	*4/*4	CPIC B[5]; FDA 3[34]

TreatG: ReviewG:

Implication: Greatly reduced metabolism of fluvoxamine to less active compounds when compared with CYP2D6 normal metabolizers. Higher plasma concentrations may increase the probability of side effects

2 Consider a 25–50% lower starting dose and slower titration schedule as compared with normal metabolizers or consider a clinically appropriate alternative antidepressant not predominantly metabolized by CYP2D6 (per CPIC optional recommendation).

Fosphenytoin	Phenotype	Genetic Test	Results	Source/Evidence
Cerebyx	Intermediate metabolizer	CYP2C9	*1/*3	CPIC A[18]; FDA 1[34]

ReviewG:

Implication: CYP2C9 intermediate metabolizer with an activity score of 1.0: reduced metabolism of Fosphenytoin to less active compounds

Higher plasma concentrations may increase the risk of cutaneous adverse reactions

2 For first dose, use typical initial dose. Consider a 25% reduction for subsequent doses

Galantamine	Phenotype	Genetic Test	Results	Source/Evidence
Razadyne	Poor metabolizer	CYP2D6	*4/*4	FDA 3[34]

TreatGx ReviewGx

Implication: CYP2D6 poor metabolizer: reduced metabolism of Galantamine to less active compounds leads to higher plasma concentrations of active drug

There is a potential impact on pharmacokinetic properties. The impact of CYP2D6 variants on the safety of Galantamine has not been established

Titrate dose based on tolerability

Gefitinib	Phenotype	Genetic Test	Results	Source/Evidence
Iressa	Poor metabolizer	CYP2D6	*4/*4	FDA 1[34]

ReviewGx

Implication: 2 FDA PGx Table Section 1 – Therapeutic Management Recommendations: Results in higher systemic concentrations and higher adverse reaction risk.
Monitor for adverse reactions.

Haloperidol	Phenotype	Genetic Test	Results	Source/Evidence
Haldol	Poor metabolizer	CYP2D6	*4/*4	DPWG[10]

TreatGx ReviewGx

Implication: 2 DWPG – Description: There are indications for an increased risk of side effects. The CYP2D6 genetic variation leads to decreased conversion of haloperidol, resulting in plasma concentrations that are approximately 1.7-fold higher.
DPWG – CYP2D6 Recommendation: Use 60% of the normal dose.

Hydrocodone	Phenotype	Genetic Test	Results	Source/Evidence
Hysingla Zohydro	Poor metabolizer	CYP2D6	*4/*4	CPIC B[8]

TreatGx ReviewGx

Implication: CYP2D6 poor metabolizer: reduced metabolism of Hydrocodone to active metabolite, but there is insufficient evidence to determine if these effects on pharmacokinetics translate into decreased analgesia or adverse effects.

CYP2D6 alleles do not indicate changes from recommended dose. If no response to Hydrocodone and opioid use is warranted, consider an opioid other than tramadol or codeine (per CPIC optional recommendation). Refer to TreatGx for alternatives and specific dosing recommendations.

Ibuprofen	Phenotype	Genetic Test	Results	Source/Evidence
Advil Caldolor Duexis Motrin IB NeoProfen	Intermediate metabolizer (AS 1.0)	CYP2C9 (Star Alleles)	*1/*3	CPIC A[33]; FDA 3[34]

TreatGx ReviewGx

Implication: CYP2C9 intermediate metabolizer with an activity score of 1.0: reduced metabolism of Ibuprofen to less active compounds

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

2 Initiate therapy with the lowest recommended dose of Ibuprofen

Iloperidone	Phenotype	Genetic Test	Results	Source/Evidence
Fanapt	Poor metabolizer	CYP2D6	*4/*4	FDA 1[34]

TreatGx ReviewGx

Implication: 2 FDA PGx Table Section 1 – CYP2D6 Therapeutic Management Recommendations: Results in higher systemic concentrations and higher adverse reaction risk (QT prolongation). Reduce dosage by 50%.

Imipramine	Phenotype	Genetic Test	Results	Source/Evidence
Tofranil	Poor metabolizer	CYP2D6	*4/*4	CPIC B[16]; FDA 3[34]
TreatGx ReviewGx	Normal metabolizer	CYP2C19	*1/*1	CPIC B[16]

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Imipramine to less active compounds
Higher plasma concentrations of active drug may increase the risk of adverse drug reactions

3 Avoid Imipramine use. If use is warranted, consider a reduction of recommended starting dose (per CPIC optional recommendation). Refer to TreatGx for alternatives and specific dosing recommendations.

Lansoprazole	Phenotype	Genetic Test	Results	Source/Evidence
Prevacid	Normal metabolizer	CYP2C19	*1/*1	CPIC A[20]; FDA 3[34]

Implication: 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.

Lofexidine	Phenotype	Genetic Test	Results	Source/Evidence
Lucemyra	Poor metabolizer	CYP2D6	*4/*4	FDA 1[34]

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Lofexidine to less active compounds

Higher plasma concentrations of active drug may increase the risk of orthostatic hypotension and bradycardia

2 This drug has an FDA therapeutic recommendation, refer to drug monograph or FDA labelling for dosing recommendations

Lovastatin	Phenotype	Genetic Test	Results	Source/Evidence
Altoprev	Normal function	SLCO1B1	*1/*1	CPIC A[7]

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

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

Lusutrombopag	Phenotype	Genetic Test	Results	Source/Evidence
Mupleta ReviewGx	Normal Factor II	Factor II rs1799963	G/G	Product monograph (actionable)[31]
	Normal Factor V Leiden	Factor V rs6025	C/C	Product monograph (actionable)[31]

Implication: Product monograph: no change in risk stated for normal Factor II (i.e. Prothrombin 20210A mutation absent).

Product monograph: no change in risk stated for normal Factor V.

Mavacamten	Phenotype	Genetic Test	Results	Source/Evidence
Camzyos ReviewGx	Normal metabolizer	CYP2C19	*1/*1	FDA 2[34]

Implication: FDA PGx Table: no information for this phenotype.

Meclizine	Phenotype	Genetic Test	Results	Source/Evidence
Antivert	Poor metabolizer	CYP2D6	*4/*4	FDA 1[34]

ReviewGx

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Meclizine to less active compounds

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

2 This drug has an FDA therapeutic recommendation, refer to drug monograph or FDA labelling for dosing recommendations

Meloxicam	Phenotype	Genetic Test	Results	Source/Evidence
Anjeso Mobic Qmiiz ODT Vivlodex	Intermediate metabolizer (AS 1.0)	CYP2C9 (Star Alleles)	*1/*3	CPIC A[33]; FDA 1[34]

TreatGx
ReviewGx

Implication: CYP2C9 intermediate metabolizer with an activity score of 1.0: reduced metabolism of Meloxicam to less active compounds

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

2 Consider a 50% reduction of the recommended dose

Dose titration should not occur until after steady state is reached (7 days after first dose)

Methotrexate	Phenotype	Genetic Test	Results	Source/Evidence
Metoject Otrexup Rasuvo Trexall Xatmep	Increased risk of toxicity compared to G/G or decreased compared to A/A	MTHFR rs1801133	G/A	PharmGKB 2A

TreatGx
ReviewGx

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

Methylphenidate	Phenotype	Genetic Test	Results	Source/Evidence
Aptensio Concerta Cotempla Daytrana Jornay Metadate Methylin Quillichew Quillivant Relexxiii Ritalin	No significant association to response	COMT rs4680	G/A	PharmGKB 4

TreatGx
ReviewGx

Implication: PharmGKB – Clinical Annotation (Level 4 Efficacy): The current evidence base suggests that there is no significant association between the COMT rs4680 A/G genotype and response to methylphenidate. However, conflicting evidence has been reported. This drug-variant pair has been assigned a "no recommendation" by DPWG, as it was determined to be not clinically actionable. Other genetic and clinical factors may also influence response to methylphenidate.

Metoclopramide	Phenotype	Genetic Test	Results	Source/Evidence
Metonia Reglan	Poor metabolizer	CYP2D6	*4/*4	FDA 1[34]

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Metoclopramide to less active compounds

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

TreatG
ReviewG

2 Consider a reduction of the recommended dose

2 This drug has an FDA therapeutic recommendation, refer to drug monograph or FDA labelling for dosing recommendations

Metoprolol	Phenotype	Genetic Test	Results	Source/Evidence
Kapspargo Sprinkle Lopressor Toprol-XL	Poor metabolizer	CYP2D6	*4/*4	DPWG[10]; FDA 3[34]

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Metoprolol to less active compounds

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

TreatG
ReviewG

3 If a gradual reduction in heart rate is desired, or in the event of clinically significant bradycardia, increase the dose in small steps and/or prescribe no more than 25% of the standard dose.

Mirabegron	Phenotype	Genetic Test	Results	Source/Evidence
Myrbetriq	Poor metabolizer	CYP2D6	*4/*4	FDA 3[34]

Implication: CYP2D6 poor metabolizer: reduced metabolism of Mirabegron leads to higher plasma concentrations

There is a potential impact on pharmacokinetic properties. The impact of CYP2D6 variants on the safety of Mirabegron has not been established

TreatG
ReviewG

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

Implication: 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.

TreatG
ReviewG

Nateglinide	Phenotype	Genetic Test	Results	Source/Evidence
	Intermediate metabolizer	CYP2C9	*1/*3	FDA 1[34]

Implication: FDA PGx Table: no information for this phenotype.

ReviewG

Nebivolol	Phenotype	Genetic Test	Results	Source/Evidence
Bystolic	Poor metabolizer	CYP2D6	*4/*4	FDA 3[34]

TreatGx
ReviewGx

Implication: CYP2D6 poor metabolizer: reduced metabolism of Nebivolol leads to higher plasma concentrations

There is a potential impact on pharmacokinetic properties. The impact of CYP2D6 variants on the safety of Nebivolol has not been established

Nicotine replacement therapy	Phenotype	Genetic Test	Results	Source/Evidence
Nicorette Nicotrol Habitrol Nicoderm Thrive	Increased likelihood of smoking cessation compared to G/G	ANKK1/DRD2 rs1800497	A/G	PharmGKB 3

TreatGx
ReviewGx

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

Nortriptyline	Phenotype	Genetic Test	Results	Source/Evidence
Aventyl Pamelor	Poor metabolizer	CYP2D6	*4/*4	CPIC A[16]; FDA 3[34]

TreatGx
ReviewGx

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Nortriptyline to less active compounds
Higher plasma concentrations of active drug may increase the risk of adverse drug reactions

3 Avoid Nortriptyline use due to potential for adverse effects. Consider alternative drug not metabolized by CYP2D6. If use is warranted, consider a reduction of the recommended dose (per CPIC strong recommendation). Refer to TreatGx for alternatives and specific dosing recommendations.

Oliceridine	Phenotype	Genetic Test	Results	Source/Evidence
Olinvyk	Poor metabolizer	CYP2D6	*4/*4	FDA 1[34]

ReviewGx

Implication: 2 FDA PGx Table Section 1 – Therapeutic Management Recommendations: Results in higher systemic concentrations and higher adverse reaction risk (respiratory depression and sedation). May require less frequent dosing.

Omeprazole	Phenotype	Genetic Test	Results	Source/Evidence
Losec Olex Prilosec	Normal metabolizer	CYP2C19	*1/*1	CPIC A[20]; FDA 3[34]

TreatGx
ReviewGx

Implication: 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
Zofran Zuplenz	Poor metabolizer	CYP2D6	*4/*4	CPIC A[3]

ReviewGx

Implication: CYP2D6 alleles do not indicate changes from recommended dose

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

Implication: 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.

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.

Pantoprazole	Phenotype	Genetic Test	Results	Source/Evidence
Pantoloc Protonix Tecta	Normal metabolizer	CYP2C19	*1/*1	CPIC A[20]; FDA 1[34]

TreatGx
ReviewGx

Implication: 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
Brisdelle Paxil Pexeva	Poor metabolizer	CYP2D6	*4/*4	CPIC A[5]; FDA 3[34]

Implication: Greatly reduced metabolism when compared with CYP2D6 normal metabolizers. Higher plasma concentrations may increase the probability of side effects. The impact of paroxetine-associated autoinhibition of CYP2D6 is minimal in poor metabolizers.

TreatGx
ReviewGx

2 Consider a 50% reduction in recommended starting dose, slower titration schedule, and a 50% lower maintenance dose as compared with normal metabolizers (per CPIC moderate recommendation).

Perphenazine	Phenotype	Genetic Test	Results	Source/Evidence
	Poor metabolizer	CYP2D6	*4/*4	FDA 2[34]

TreatGx
ReviewGx

Implication: 2 FDA PGx Table Section 2 – CYP2D6 Potential Impact on Safety or Response: Results in higher systemic concentrations and higher adverse reaction risk.

Phenytoin	Phenotype	Genetic Test	Results	Source/Evidence
Dilantin Tremytoine Phenytek ReviewGx	Intermediate metabolizer	CYP2C9	*1/*3	CPIC A[18]; FDA 1[34]

Implication: CYP2C9 intermediate metabolizer with an activity score of 1.0: reduced metabolism of Phenytoin to less active compounds

Higher plasma concentrations may increase the risk of cutaneous adverse reactions

2 For first dose, use typical initial dose. Consider a 25% reduction for subsequent doses

Pimozide	Phenotype	Genetic Test	Results	Source/Evidence
Orap TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 1[34]

Implication: 2 FDA PGx Table Section 1 – CYP2D6 Therapeutic Management Recommendations: Results in higher systemic concentrations. Dosages should not exceed 0.05 mg/kg in children or 4 mg/day in adults who are poor metabolizers and dosages should not be increased earlier than 14 days.

Piroxicam	Phenotype	Genetic Test	Results	Source/Evidence
Feldene TreatGx ReviewGx	Intermediate metabolizer (AS 1.0)	CYP2C9 (Star Alleles)	*1/*3	CPIC A[33]; FDA 1[34]

Implication: CYP2C9 intermediate metabolizer with an activity score of 1.0: reduced metabolism of Piroxicam to less active compounds

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

2 Consider an alternative drug not predominantly metabolized by CYP2C9

Pitavastatin	Phenotype	Genetic Test	Results	Source/Evidence
Livalo Zypitamag TreatGx ReviewGx	Normal function	SLCO1B1	*1/*1	CPIC A[7]

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

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

Pitolisant	Phenotype	Genetic Test	Results	Source/Evidence
Wakix ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 1[34]; Product monograph (actionable)[15]

Implication: 2 FDA PGx Table Section 1 – Therapeutic Management Recommendations: Results in higher systemic concentrations. Use lowest recommended starting dosage. Refer to FDA labeling for specific dosing recommendations.

2 FDA Product Monograph: In patients known to be poor CYP2D6 metabolizers, initiate pitolisant at 8.9 mg once daily and titrate to a maximum dose of 17.8 mg once daily after 7 days.

Pravastatin	Phenotype	Genetic Test	Results	Source/Evidence
Pravachol	Normal function	SLCO1B1	*1/*1	CPIC A[7]

TreatG
ReviewG

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

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

Propafenone	Phenotype	Genetic Test	Results	Source/Evidence
Rythmol	Poor metabolizer	CYP2D6	*4/*4	DPWG[10]; FDA 1[34]

TreatG
ReviewG

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Propafenone to less active compounds

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

2 Reduce the standard dose by 70%, record electrocardiogram, and monitor plasma concentration

Propranolol	Phenotype	Genetic Test	Results	Source/Evidence
Inderal Innopran	Poor metabolizer	CYP2D6	*4/*4	FDA 3[34]

TreatG
ReviewG

Implication: CYP2D6 poor metabolizer: reduced metabolism of Propranolol leads to higher plasma concentrations

There is a potential impact on pharmacokinetic properties. The impact of CYP2D6 variants on the safety of Propranolol has not been established

Protriptyline	Phenotype	Genetic Test	Results	Source/Evidence
Vivactil	Poor metabolizer	CYP2D6	*4/*4	FDA 3[34]

ReviewG

Implication: CYP2D6 poor metabolizer: reduced metabolism of Protriptyline to less active compounds leads to higher plasma concentrations of active drug

There is a potential impact on pharmacokinetic properties. The impact of CYP2D6 variants on the safety of Protriptyline has not been established

Quetiapine	Phenotype	Genetic Test	Results	Source/Evidence
Seroquel	Normal metabolizer	CYP3A4	*1/*1	DPWG[10]

TreatG
ReviewG

Implication: DPWG: no recommendation for this CYP3A4 phenotype.

Rabeprazole	Phenotype	Genetic Test	Results	Source/Evidence
Aciphex Pariet	Normal metabolizer	CYP2C19	*1/*1	FDA 3[34]

TreatG
ReviewG

Implication: FDA PGx Table: no information for this phenotype.

Risperidone	Phenotype	Genetic Test	Results	Source/Evidence
Perseris Risperdal	Poor metabolizer	CYP2D6	*4/*4	DPWG[10]; FDA 3[34]
	Increased prolactin compared to G/G	ANKK1/DRD2 rs1800497	A/G	PharmGKB 3

TreatG
ReviewG

Implication:

2 DWPG – CYP2D6 Description: The percentage of patients with therapy failure increased from 16% to 26%. The gene variation increases the plasma concentration of risperidone plus the active metabolite and increases the proportion of risperidone in this ratio, which is more effective at crossing the blood-brain barrier.
DWPG – CYP2D6 Recommendation: Use 67% of the normal dose. If problematic side effects originating in the central nervous system occur despite this reduced dose, then reduce the dose further to 50% of the normal dose.

FDA PGx Table Section 3 – CYP2D6 Potential Impact on Pharmacokinetic Properties Only: Alters systemic parent drug and metabolite concentrations.

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

Rosuvastatin	Phenotype	Genetic Test	Results	Source/Evidence
Crestor	Normal function	SLCO1B1	*1/*1	CPIC A[7]; FDA 3[34]

TreatG
ReviewG

Implication:

CPIC – SLCO1B1 Implication: Typical myopathy risk and Rosuvastatin exposure.

CPIC – Strong 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 ancestry should be evaluated prior to initiating a statin.

Sertraline	Phenotype	Genetic Test	Results	Source/Evidence
Zoloft	Normal metabolizer	CYP2B6	*1/*1	CPIC B[5]
	Normal metabolizer	CYP2C19	*1/*1	CPIC A[5]

TreatG
ReviewG

Implication:

Normal CYP2B6 metabolism

Normal CYP2C19 metabolism

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

Simvastatin	Phenotype	Genetic Test	Results	Source/Evidence
Zocor Flolipid	Normal function	SLCO1B1	*1/*1	CPIC A[7]; FDA 2[34]

TreatG
ReviewG

Implication:

CPIC – Implication: Typical myopathy risk and Simvastatin exposure.

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

Siponimod	Phenotype	Genetic Test	Results	Source/Evidence
Mayzent	Intermediate metabolizer	CYP2C9 (Star Alleles)	*1/*3	FDA 1[34]

ReviewGx

Implication: Reduced metabolism of Siponimod to less active compounds

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

2 Consider a reduction of the recommended dose

2 This drug has an FDA therapeutic recommendation, refer to drug monograph or FDA labelling for dosing recommendations

Tacrolimus	Phenotype	Genetic Test	Results	Source/Evidence
Advagraf Astagraf XL Envarsus XR Prograf Protopic	Poor metabolizer	CYP3A5	*3/*3	CPIC A[4]; FDA 1[34]
	Normal metabolizer	CYP3A4	*1/*1	PharmGKB 2A

ReviewGx

Implication: 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.

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.

Tamoxifen	Phenotype	Genetic Test	Results	Source/Evidence
Nolvadex Soltamox	Poor metabolizer	CYP2D6 (Activity Score)	*4/*4	CPIC A[13]; FDA 3[34]

ReviewGx

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Tamoxifen to endoxifen

3 Strong CPIC recommendation for breast cancer therapy: Alternative hormonal therapy recommended.

2 Higher dose tamoxifen (40 mg/day) increases but does not normalize endoxifen concentrations and can be considered if there are contraindications to aromatase inhibitor therapy.

Recommendation for conditions other than breast cancer: There is a potential impact on pharmacokinetic properties. The impact of CYP2D6 variants on the safety of Tamoxifen has not been established (FDA PGx Table)

Tamsulosin	Phenotype	Genetic Test	Results	Source/Evidence
Flomax	Poor metabolizer	CYP2D6	*4/*4	FDA 3[34]

ReviewGx

Implication: CYP2D6 poor metabolizer: reduced metabolism of Tamsulosin to less active compounds leads to higher plasma concentrations of active drug

There is a potential impact on pharmacokinetic properties. The impact of CYP2D6 variants on the safety of Tamsulosin has not been established

Tenoxicam	Phenotype	Genetic Test	Results	Source/Evidence
Mobiflex	Intermediate metabolizer (AS 1.0)	CYP2C9 (Star Alleles)	*1/*3	CPIC A[33]

ReviewGx

Implication: CYP2C9 intermediate metabolizer with an activity score of 1.0: reduced metabolism of Tenoxicam to less active compounds

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

2 Consider an alternative drug not predominantly metabolized by CYP2C9

Tetrabenazine	Phenotype	Genetic Test	Results	Source/Evidence
Austedo Nitoman Xenazine	Poor metabolizer	CYP2D6	*4/*4	FDA 1[34]

ReviewGx

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Tetrabenazine to less active compounds

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

2 Consider a reduction of maximum daily dose

2 This drug has an FDA therapeutic recommendation, refer to drug monograph or FDA labelling for dosing recommendations

Thioridazine	Phenotype	Genetic Test	Results	Source/Evidence
	Poor metabolizer	CYP2D6	*4/*4	FDA 1[34]

TreatGx
ReviewGx

Implication: 3 FDA PGx Table Section 1 – CYP2D6 Therapeutic Management Recommendations: Results in higher systemic concentrations and higher adverse reaction risk (QT prolongation). Predicted effect based on experience with CYP2D6 inhibitors. Contraindicated in poor metabolizers.

Tolterodine	Phenotype	Genetic Test	Results	Source/Evidence
Detrol	Poor metabolizer	CYP2D6	*4/*4	FDA 2[34]

TreatGx
ReviewGx

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Tolterodine

Higher plasma concentrations may increase the risk of QT prolongation

2 Data indicate a potential impact on patient safety

Tramadol	Phenotype	Genetic Test	Results	Source/Evidence
Conzip Durela Ralivia Ultram Zytram XL	Poor metabolizer	CYP2D6	*4/*4	CPIC A[8]; FDA 1[34]; FDA 2[34]

TreatGx
ReviewGx

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Tramadol to active metabolite may result in diminished analgesia

3 Avoid Tramadol use due to possibility of diminished analgesia. If opioid use is warranted, consider an opioid other than tramadol or codeine (per CPIC strong recommendation). Refer to TreatGx for alternatives and specific dosing recommendations.

Trimipramine	Phenotype	Genetic Test	Results	Source/Evidence
Surmontil	Poor metabolizer	CYP2D6	*4/*4	CPIC B[16]; FDA 3[34]
ReviewGx	Normal metabolizer	CYP2C19	*1/*1	CPIC B[16]

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Trimipramine to less active compounds
Higher plasma concentrations of active drug may increase the risk of adverse drug reactions

3 Avoid Trimipramine use. If use is warranted, consider a reduction of recommended starting dose (per CPIC optional recommendation). Refer to TreatGx for alternatives and specific dosing recommendations.

Valbenazine	Phenotype	Genetic Test	Results	Source/Evidence
Ingrezza	Poor metabolizer	CYP2D6	*4/*4	FDA 1[34]
ReviewGx				

Implication: CYP2D6 poor metabolizer: greatly reduced metabolism of Valbenazine to less active compounds

Higher plasma concentrations of active drug may increase the risk of QT prolongation

2 Consider a reduction of the recommended dose

2 This drug has an FDA therapeutic recommendation, refer to drug monograph or FDA labelling for dosing recommendations

Venlafaxine	Phenotype	Genetic Test	Results	Source/Evidence
Effexor XR	Poor metabolizer	CYP2D6	*4/*4	CPIC B[5]; FDA 1[34]
TreatGx ReviewGx				

Implication: Decreased metabolism of venlafaxine to the active metabolite O-desmethylvenlafaxine (desvenlafaxine) and greatly decreased O-desmethylvenlafaxine: venlafaxine ratio as compared with CYP2D6 normal and intermediate metabolizers. The clinical impact of increased venlafaxine and decreased O-desmethylvenlafaxine: venlafaxine ratio in CYP2D6 poor metabolizers is unclear, but CYP2D6 PM genotype has been associated with adverse effects.

2 Consider a clinically appropriate alternative antidepressant not predominantly metabolized by CYP2D6 (per CPIC optional recommendation).

Viloxazine	Phenotype	Genetic Test	Results	Source/Evidence
Qelbree	Poor metabolizer	CYP2D6	*4/*4	FDA 3[34]
ReviewGx				

Implication: FDA PGx Table Section 3 – Potential Impact on Pharmacokinetic Properties Only: May result in higher systemic concentrations.

Voriconazole	Phenotype	Genetic Test	Results	Source/Evidence
Vfend	Normal metabolizer	CYP2C19	*1/*1	CPIC A[24]; FDA 2[34]
ReviewGx				

Implication: CYP2C19 alleles do not indicate changes from recommended dose

Vortioxetine	Phenotype	Genetic Test	Results	Source/Evidence
Trintellix TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	CPIC A[5]; FDA 1[34]

Implication: Greatly reduced metabolism of vortioxetine to inactive compounds when compared with CYP2D6 normal metabolizers. Higher plasma concentrations may increase the probability of side effects.

2 Initiate 50% of starting dose (e.g., 5 mg) and titrate to the maximum recommended dose of 10 mg or consider a clinically appropriate alternative antidepressant not predominantly metabolized by CYP2D6 (per CPIC moderate recommendation).

Warfarin	Phenotype	Genetic Test	Results	Source/Evidence
Coumadin Jantoven TreatGx ReviewGx	Intermediate metabolizer	CYP2C9	*1/*3	CPIC A[17]; FDA 1[34]
	Reduced response	VKORC1	G/G	CPIC A[17]; FDA 1[34]

Implication: 2 The algorithm in TreatGx includes pharmacogenetics and other clinical factors in calculating initial warfarin dose

Zuclopenthixol	Phenotype	Genetic Test	Results	Source/Evidence
Clopixol TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	DPWG[10]

Implication: 2 DWPG – CYP2D6 Description: The risk of side effects may be elevated. The genetic variation results in a decreased conversion of zuclopenthixol, which causes the plasma concentration to be approximately 1.6-fold higher.
DWPG – CYP2D6 Recommendation: Use with 50% of the normal dose.

Table of Available References

Drug	Genetic Test	Sources
Abrocitinib	CYP2C19	FDA[27,34]
Alfentanil	OPRM1 rs1799971	PharmGKB
Amitriptyline	CYP2D6	CPIC[16]; FDA[34]
Amitriptyline	CYP2C19	CPIC[16]
Amoxapine	CYP2D6	FDA[34]
Amphetamine	CYP2D6	FDA[34]
Aripiprazole	CYP2D6	DPWG[10]; FDA[34]
Aripiprazole lauroxil	CYP2D6	FDA[34]
Atomoxetine	CYP2D6 (Activity Score)	CPIC[6]; FDA[34]
Atorvastatin	SLCO1B1	CPIC[7]; FDA[34]
Avatrombopag	CYP2C9	FDA[34]
Avatrombopag	Factor II rs1799963	FDA[1]
Avatrombopag	Factor V rs6025	FDA[1]
Brexpiprazole	CYP2D6	DPWG[10]; FDA[34]
Brivaracetam	CYP2C19	FDA[34]
Bupropion	ANKK1/DRD2 rs1800497	PharmGKB
Carisoprodol	CYP2C19	FDA[34]
Carvedilol	CYP2D6	FDA[34]
Celecoxib	CYP2C9 (Star Alleles)	CPIC[33]; FDA[34]
Cevimeline	CYP2D6	FDA[34]
Citalopram	CYP2C19	CPIC[5]; FDA[34]
Clobazam	CYP2C19	FDA[21,34]; Product monograph (actionable)[21]
Clomipramine	CYP2D6	CPIC[16]; FDA[34]
Clomipramine	CYP2C19	CPIC[16]
Clopidogrel	CYP2C19	CPIC[19]; FDA[34]
Clozapine	CYP2D6	FDA[34]
Codeine	CYP2D6	CPIC[8]; FDA[34]
Cyclosporine	CYP3A5	PharmGKB
Darifenacin	CYP2D6	FDA[34]
Desipramine	CYP2D6	CPIC[16]; FDA[34]
Deutetrabenazine	CYP2D6	FDA[34]
Dexlansoprazole	CYP2C19	CPIC[20]; FDA[34]
Diazepam	CYP2C19	FDA[34]
Donepezil	CYP2D6	FDA[34]
Doxepin	CYP2D6	CPIC[16]; FDA[34]
Doxepin	CYP2C19	CPIC[16]; FDA[34]
Dronabinol	CYP2C9	FDA[34]
Efavirenz	CYP2B6	CPIC[9]; DPWG[10]; FDA[34]
Elagolix	SLCO1B1	FDA[34]
Eliglustat	CYP2D6	DPWG[10]; FDA[34]
Eltrombopag	Factor V rs6025	FDA[26]
Erdafitinib	CYP2C9 (Star Alleles)	FDA[34]
Escitalopram	CYP2C19	CPIC[5]; FDA[34]

Drug	Genetic Test	Sources
Esomeprazole	CYP2C19	FDA[34]
Fentanyl	OPRM1 rs1799971	PharmGKB
Fesoterodine	CYP2D6	FDA[34]
Flecainide	CYP2D6	DPWG[10]
Flibanserin	CYP2C19	FDA[34]
Flurbiprofen	CYP2C9 (Star Alleles)	CPIC[33]; FDA[34]
Fluvastatin	CYP2C9	CPIC[7]
Fluvastatin	SLCO1B1	CPIC[7]
Fluvoxamine	CYP2D6	CPIC[5]; FDA[34]
Fosphenytoin	CYP2C9	CPIC[18]; FDA[34]
Galantamine	CYP2D6	FDA[34]
Gefitinib	CYP2D6	FDA[34]
Haloperidol	CYP2D6	DPWG[10]
Hydrocodone	CYP2D6	CPIC[8]
Ibuprofen	CYP2C9 (Star Alleles)	CPIC[33]; FDA[34]
Iloperidone	CYP2D6	FDA[34]
Imipramine	CYP2D6	CPIC[16]; FDA[34]
Imipramine	CYP2C19	CPIC[16]
Lansoprazole	CYP2C19	CPIC[20]; FDA[34]
Lofexidine	CYP2D6	FDA[34]
Lovastatin	SLCO1B1	CPIC[7]
Lusutrombopag	Factor II rs1799963	FDA[31]
Lusutrombopag	Factor V rs6025	FDA[31]
Mavacamten	CYP2C19	FDA[34]
Meclizine	CYP2D6	FDA[34]
Meloxicam	CYP2C9 (Star Alleles)	CPIC[33]; FDA[34]
Methotrexate	MTHFR rs1801133	PharmGKB
Methylphenidate	COMT rs4680	PharmGKB
Metoclopramide	CYP2D6	FDA[34]
Metoprolol	CYP2D6	DPWG[10]; FDA[34]
Mirabegron	CYP2D6	FDA[34]
Morphine	OPRM1 rs1799971	PharmGKB
Nateglinide	CYP2C9	FDA[34]
Nebivolol	CYP2D6	FDA[34]
Nicotine replacement therapy	ANKK1/DRD2 rs1800497	PharmGKB
Nortriptyline	CYP2D6	CPIC[16]; FDA[34]
Oliceridine	CYP2D6	FDA[34]
Omeprazole	CYP2C19	CPIC[20]; FDA[34]
Ondansetron	CYP2D6	CPIC[3]
Oral contraceptives	Factor II rs1799963	PharmGKB
Oral contraceptives	Factor V rs6025	PharmGKB
Pantoprazole	CYP2C19	CPIC[20]; FDA[34]
Paroxetine	CYP2D6	CPIC[5]; FDA[34]
Perphenazine	CYP2D6	FDA[34]
Phenytoin	CYP2C9	CPIC[18]; FDA[34]
Pimozide	CYP2D6	DPWG[10]; FDA[34]
Piroxicam	CYP2C9 (Star Alleles)	CPIC[33]; FDA[34]
Pitavastatin	SLCO1B1	CPIC[7]

Drug	Genetic Test	Sources
Pitolisant	CYP2D6	FDA[15,34]
Pravastatin	SLCO1B1	CPIC[7]
Propafenone	CYP2D6	DPWG[10]; FDA[34]
Propranolol	CYP2D6	FDA[34]
Protriptyline	CYP2D6	FDA[34]
Quetiapine	CYP3A4	DPWG[10]
Rabeprazole	CYP2C19	FDA[34]
Risperidone	CYP2D6	DPWG[10]; FDA[34]
Risperidone	ANKK1/DRD2 rs1800497	PharmGKB
Rosuvastatin	SLCO1B1	CPIC[7]; FDA[34]
Sertraline	CYP2B6	CPIC[5]
Sertraline	CYP2C19	CPIC[5]
Simvastatin	SLCO1B1	CPIC[7]; FDA[34]
Siponimod	CYP2C9 (Star Alleles)	FDA[34]
Tacrolimus	CYP3A5	CPIC[4]; FDA[34]
Tacrolimus	CYP3A4	PharmGKB
Tamoxifen	CYP2D6 (Activity Score)	CPIC[13]; FDA[34]
Tamsulosin	CYP2D6	FDA[34]
Tenoxicam	CYP2C9 (Star Alleles)	CPIC[33]
Tetrabenazine	CYP2D6	FDA[34]
Thioridazine	CYP2D6	FDA[34]
Tolterodine	CYP2D6	FDA[34]
Tramadol	CYP2D6	CPIC[8]; FDA[34]
Trimipramine	CYP2D6	CPIC[16]; FDA[34]
Trimipramine	CYP2C19	CPIC[16]
Valbenazine	CYP2D6	FDA[34]
Venlafaxine	CYP2D6	CPIC[5]; FDA[34]
Viloxazine	CYP2D6	FDA[34]
Voriconazole	CYP2C19	CPIC[24]; FDA[34]
Vortioxetine	CYP2D6	CPIC[5]; FDA[34]
Warfarin	CYP2C9	CPIC[17]; FDA[34]
Warfarin	VKORC1	CPIC[17]; FDA[34]
Zuclopenthixol	CYP2D6	DPWG[10]

References

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

Methods

DNA was extracted from dried blood spot (DBS) card by Chemagic 360 system (Revvity) and processed in a Biomark X platform (Standard Biotools) with Advanta™ Pharmacogenomics Assay.

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 Juha Matilainen, Laboratory Director, PhD

13/Aug/2024

Date of Signature

Laboratory Report

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

Gene	rsID	HGVS	HGVS Reference	Result
ABCB1	rs1045642	c.3645T>C	NM_001348945.2	G/G
ANKK1/DRD2	rs1800497	c.2137G>A	NM_178510.1	G/A
APOE	rs429358	c.388T>C	NM_000041.3	T/T
APOE	rs7412	c.526C>T	NM_000041.3	C/C
COMT	rs4680	c.472G>A	NM_000754.3	G/A
CYP1A2	rs12720461	c.-10+113C>T	NM_000761.4	C/C
CYP1A2	rs2069514	g.74745879G>A	NC_000015.10	G/G
CYP1A2	rs56107638	g.9427G>A	NG_061543.1	G/G
CYP1A2	rs72547513	c.558C>T	NM_000761.4	C/C
CYP1A2	rs762551	c.-9-154A>C	NM_000761.3	A/A
CYP2B6	rs28399499	c.983T>C	NM_000767.4	T/T
CYP2B6	rs3745274	c.516G>T	NM_000767.5	G/G
CYP2C19	rs12248560	g.94761900C>T	NC_000010.11	C/C
CYP2C19	rs12769205	c.332-23A>G	NM_000769.2	A/A
CYP2C19	rs17884712	c.431G>A	NM_000769.4	G/G
CYP2C19	rs28399504	c.1A>G	NM_000769.4	A/A
CYP2C19	rs4244285	c.681G>A	NM_000769.4	G/G
CYP2C19	rs4986893	c.636G>A	NM_000769.4	G/G
CYP2C19	rs56337013	c.1297C>T	NM_000769.4	C/C
CYP2C19	rs6413438	c.680C>T	NM_000769.4	C/C
CYP2C19	rs72552267	c.395G>A	NM_000769.4	G/G
CYP2C19	rs72558186	g.94781999T>A	NC_000010.11	T/T
CYP2C9	rs1057910	c.1075A>C	NM_000771.4	A/C
CYP2C9	rs1799853	c.430C>T	NM_000771.4	C/C
CYP2C9	rs28371685	c.1003C>T	NM_000771.4	C/C
CYP2C9	rs28371686	c.1080C>G	NM_000771.4	C/C
CYP2C9	rs56165452	c.1076T>C	NM_000771.4	T/T
CYP2C9	rs72558187	c.269T>C	NM_000771.4	T/T
CYP2C9	rs72558190	c.485C>A/T	NM_000771.4	C/C
CYP2C9	rs7900194	c.449G>A/C/T	NM_000771.4	G/G
CYP2C9	rs9332131	c.818del	NM_000771.4	A/A
CYP2C9	rs9332239	c.1465C>T	NM_000771.4	C/C
CYP2D6	rs1065852	c.100C>T	NM_000106.6	A/A
CYP2D6	rs1135822	c.358T>A	NM_000106.6	A/A
CYP2D6	rs1135840	c.1457G>C	NM_000106.6	G/G
CYP2D6	rs16947	c.886C>T	NM_000106.6	G/G
CYP2D6	rs201377835	g.42129910C>G	NC_000022.11	C/C
CYP2D6	rs267608319	c.1319G>A	NM_000106.6	C/C
CYP2D6	rs28371706	c.320C>T	NM_000106.6	G/G
CYP2D6	rs28371725	c.985+39G>A	NM_000106.5	C/C
CYP2D6	rs35742686	c.775del	NM_000106.6	T/T
CYP2D6	rs3892097	g.42128945C>T	NC_000022.11	T/T
CYP2D6	rs5030655	c.454del	NM_000106.6	A/A
CYP2D6	rs5030656	c.841_843del	NM_000106.6	CTT/CTT
CYP2D6	rs5030862	c.124G>A	NM_000106.6	C/C
CYP2D6	rs5030865	c.505G>T/C/A	NM_000106.6:	C/C
CYP2D6	rs5030867	c.971A>C	NM_000106.6	T/T
CYP2D6	rs59421388	c.971A>C	NM_000106.6	C/C

Gene	rsID	HGVS	HGVS Reference	Result
CYP2D6	rs72549356	c.514_522dup	NM_000106.6	-/-
CYP2D6	rs72549346	c.1088_1089dup	NM_000106.6	-/-
CYP2D6	rs72549347	c.1030C>T	NM_000106.6	G/G
CYP2D6	rs72549352	c.805dup	NM_000106.6:	-/-
CYP2D6	rs72549353	c.765_768del	NM_000106.6	AGTT/AGTT
CYP2D6	rs72549354	c.635dup	NM_000106.6	-/-
CYP2D6	rs79292917	c.975G>A	NM_000106.6	C/C
CYP3A4	rs35599367	c.522-191C>T	NM_017460.6	G/G
CYP3A4	rs4987161	c.566T>C	NM_017460.6	A/A
CYP3A4	rs55785340	c.664T>C	NM_017460.6	A/A
CYP3A5	rs10264272	c.624G>A	NM_000777.5	C/C
CYP3A5	rs28365083	c.1193C>A	NM_000777.5	G/G
CYP3A5	rs41303343	c.1035dup	NM_000777.5	-/-
CYP3A5	rs776746	c.219-237A>G	NM_000777.5	C/C
Factor II	rs1799963	c.*97G>A	NM_000506.5	G/G
Factor V	rs6025	c.1601G>A	NM_000130.4	C/C
MTHFR	rs1801131	c.1286A>C	NM_005957.5	T/G
MTHFR	rs1801133	c.665C>T	NM_005957.5	A/G
OPRM1	rs1799971	c.118A>G	NM_000914.5	A/A
SLCO1B1	rs4149056	c.521T>C	NM_006446.5	T/T
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.

Copy Number Variation

Gene	Reference	Result (Copy/Copies)
CYP2D6	NG_008376.3 exon 9	2
CYP2D6_intron6	NG_008376.3 intron 6	3
CYP2D6_5pFlank	NG_008376.3 CYP2D6_5pFlank	3

Phenotype Table

Gene	Allele Result	Phenotype Result
CYP3A4	*1/*1	Normal Metabolizer
CYP2D6	*4/*4	Poor Metabolizer
CYP2C9	*1/*3	Intermediate Metabolizer
CYP2C19	*1/*1	Normal Metabolizer
SLCO1B1	*1/*1	Normal Function
CYP2B6	*1/*1	Normal Metabolizer
CYP3A5	*3/*3	Poor Metabolizer