AttoDiagnostics

PATIENT INFORMATION
NAME: Claire DDI-DGI
DOB: 23/Nov/1993
SEX AT BIRTH: Female

SPECIMEN DETAILS
BARCODE: 4124081155
SAMPLE ID: 4124081155
TYPE: Swab
COLLECTED: 28/Mar/2025
RECEIVED: 30/Mar/2025
QUALITY: Good

ORDERED BY
ATTOD Demo
AMENDED: 27/Nov/2025 (GMT)

Personalised Pharmacogenetics Report

Overview

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

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

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

TreatGx

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

ReviewGx

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

Components of the Medication Report

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

Example:

Generic Name → Codeine

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

Potential Kidney or Liver Interaction → TreatGx ReviewGx

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

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

3 Avoid Codeine use

Source/Evidence Level for Drug-Gene Interactions:

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

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

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

AttoDiagnostics Ltd Scottow Enterprise Park, Lamas Road, Badersfield, Norfolk, NR10 5FB +44 1603 327 053
info@attodiagnostics.com www.attodiagnostics.com

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

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

Clopidogrel	Phenotype	Genetic Test	Results	Source/Evidence Level
Plavix TreatG ReviewG	Poor metabolizer	CYP2C19	*2/*2	CPIC A[28]; FDA 1[46]

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

3 CPIC – Implication: Significantly reduced clopidogrel active metabolite formation; increased on-treatment platelet reactivity; increased risk for adverse cardiac and cerebrovascular events.

3 CPIC – Strong Recommendation for acute coronary syndrome (ACS) and/or percutaneous coronary intervention (PCI); Moderate Recommendation for non-ACS/non-PCI cardiovascular indications: Avoid clopidogrel if possible. Use prasugrel or ticagrelor at standard dose if no contraindication.
ACS and/or PCI includes patients undergoing PCI for an ACS or non-ACS (elective) indication. Non-ACS, non-PCI cardiovascular indications include peripheral arterial disease and stable coronary artery disease following a recent myocardial infarction outside the setting of PCI.

Drug-Drug Interactions:

2 (Clopidogrel)-(Rosuvastatin) The metabolism of Rosuvastatin can be decreased when combined with Clopidogrel.

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

2 (Sertraline)-(Clopidogrel) The serum concentration of the active metabolites of Clopidogrel can be reduced when Clopidogrel is used in combination with Sertraline resulting in a loss in efficacy.

Codeine	Phenotype	Genetic Test	Results	Source/Evidence Level
Codeine Contin Tylenol with Codeine No. 2/3/4 TreatG ReviewG	Poor metabolizer	CYP2D6	*4/*4	CPIC A[14]; FDA 1[46]; FDA 2[46]

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

3 CPIC – Implication: Greatly reduced morphine formation leading to diminished analgesia.

3 CPIC – Strong Recommendation: Avoid codeine use because of possibility of diminished analgesia. If opioid use is warranted, consider a non-tramadol opioid.

Drug-Drug Interactions:

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

2 (Codeine)-(Sertraline) The risk or severity of serotonin syndrome can be increased when Codeine is combined with Sertraline.

1 (Atomoxetine)-(Codeine) The metabolism of Codeine can be decreased when combined with Atomoxetine.

Sertraline	Phenotype	Genetic Test	Results	Source/Evidence Level
Zoloft	Poor metabolizer	CYP2B6	*6/*6	CPIC B[10]
TreatGx ReviewGx	Poor metabolizer	CYP2C19	*2/*2	CPIC A[10]

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

3 Greatly reduced metabolism of sertraline to less active compounds when compared with CYP2B6 normal metabolizers. Higher plasma concentrations may increase the probability of side effects.

3 Greatly reduced metabolism of sertraline to less active compounds when compared with CYP2C19 normal metabolizers. Higher plasma concentrations may increase the probability of side effects.

3 Select an alternative antidepressant not primarily metabolized by CYP2C19 or CYP2B6 (per CPIC optional recommendation).

Drug-Drug Interactions:

2 (Codeine)-(Sertraline) The risk or severity of serotonin syndrome can be increased when Codeine is combined with Sertraline.

2 (Sertraline)-(Clopidogrel) The serum concentration of the active metabolites of Clopidogrel can be reduced when Clopidogrel is used in combination with Sertraline resulting in a loss in efficacy.

2 (Sertraline)-(Atomoxetine) The metabolism of Atomoxetine can be decreased when combined with Sertraline.

2 (Sertraline)-(Rosuvastatin) The metabolism of Rosuvastatin can be decreased when combined with Sertraline.

Atomoxetine	Phenotype	Genetic Test	Results	Source/Evidence Level
Strattera	Poor metabolizer	CYP2D6 (Activity Score)	*4/*4	CPIC A[11]; FDA 1[46]
TreatGx ReviewGx				

Drug-Gene Interactions:

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

2 CPIC – Implication: Significantly decreased metabolism of atomoxetine may result in higher concentrations as compared to non-poor metabolizers. This may increase the occurrence of side effects, but also a greater improvement of ADHD symptoms as compared to non-poor metabolizers in those who tolerate treatment. Poor metabolizer status is associated with lower final dose requirements as compared to non-poor metabolizers.

2 ADULTS: CPIC – Moderate Recommendation: Initiate with a dose of 40 mg/day and if no clinical response and in the absence of adverse events after 2 weeks increase dose to 80 mg/day. If response is inadequate after 2 weeks, consider obtaining a plasma concentration 2 to 4 hours after dosing. If concentration is <200 ng/mL, consider a proportional dose increase to achieve a concentration to approach 400 ng/mL. If unacceptable side effects are present at any time, consider a reduction in dose.
PEDIATRICS: CPIC – Strong Recommendation: Initiate with a dose of 0.5 mg/kg/day and if no clinical response and in the absence of adverse events after 2 weeks, consider obtaining a plasma concentration 4 hours 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. If unacceptable side effects are present at any time, consider a reduction in dose.
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.

Drug-Drug Interactions:

2 (Sertraline)-(Atomoxetine) The metabolism of Atomoxetine can be decreased when combined with Sertraline.

1 (Atomoxetine)-(Codeine) The metabolism of Codeine can be decreased when combined with Atomoxetine.

Rosuvastatin	Phenotype	Genetic Test	Results	Source/Evidence Level
Crestor	Normal function	SLCO1B1	*37/*37	CPIC A[13]; FDA 3[46]
	Normal function	ABCG2 rs2231142	G/G	CPIC A[13]

TreatGx
ReviewGx

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: Typical myopathy risk and rosuvastatin exposure.

1 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 Asian ancestry should be evaluated prior to initiating rosuvastatin.

Drug-Drug Interactions:

2 (Clopidogrel)-(Rosuvastatin) The metabolism of Rosuvastatin can be decreased when combined with Clopidogrel.

2 (Sertraline)-(Rosuvastatin) The metabolism of Rosuvastatin can be decreased when combined with Sertraline.

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

Analgesia

1
- Carisoprodol
- Celecoxib
- Flurbiprofen
- Hydrocodone
- Ibuprofen
- Meloxicam
- Methadone
- Oxycodone
- Piroxicam
- Tenoxicam

2
- Alfentanil
- Fentanyl
- Morphine
- Oliceridine
- Venlafaxine

3
- Amitriptyline
- Codeine
- Desipramine
- Imipramine
- Nortriptyline
- Tramadol

Autoimmune

1
- Azathioprine

...Autoimmune

1
- Mercaptopurine
- Methotrexate
- Siponimod
- Thioguanine

2
- Cyclosporine
- Tacrolimus

Cancer

1
- Capecitabine
- Erdafitinib
- Fluorouracil
- Mercaptopurine
- Methotrexate
- Thioguanine

2
- Gefitinib

3
- Tamoxifen

Cardiovascular

1
- Fluvastatin
- Lovastatin
- Nebivolol
- Pitavastatin
- Pravastatin
- Propranolol
- Rosuvastatin

...Cardiovascular

1
- Simvastatin

2
- Atorvastatin
- Carvedilol
- Flecainide
- Mavacamten
- Metoprolol
- Propafenone
- Warfarin

3
- Clopidogrel

Endocrinology

1
- Nateglinide

Gastroenterology

1
- Dronabinol
- Esomeprazole
- Methotrexate
- Ondansetron
- Rabeprazole

2
- Dexlansoprazole
- Lansoprazole
- Meclizine
- Metoclopramide
- Omeprazole
- Pantoprazole

Infection

1
- Nevirapine

2
- Efavirenz

3
- Voriconazole

Mental Health

1
- Amoxapine
- Diazepam
- Methadone
- Methylphenidate
- Mirtazapine
- Nicotine replacement therapy
- Olanzapine
- Protriptyline
- Quetiapine
- Viloxazine

2
- Amphetamine
- Aripiprazole
- Aripiprazole lauroxil
- Atomoxetine
- Brexpiprazole
- Bupropion
- Citalopram
- Clozapine

...Mental Health

2

Dextromethorphan/ Bupropion
Escitalopram
Fluoxetine
Fluvoxamine
Haloperidol
Iloperidone
Lofexidine
Paroxetine
Perphenazine
Pimozide
Risperidone
Venlafaxine
Vortioxetine
Zuclopenthixol

3

Amitriptyline
Clomipramine
Desipramine
Doxepin
Imipramine
Nortriptyline
Sertraline
Thioridazine
Trimipramine

Neurology

1

Aducanumab
Diazepam
Donepezil
Fosphenytoin
Galantamine
Lecanemab
Phenytoin
Propranolol

2

Brivaracetam

...Neurology

2

Clobazam
Deutetrabenazine
Dextromethorphan/ Quinidine
Metoprolol
Pitolisant
Tetrabenazine
Valbenazine
Venlafaxine

3

Amitriptyline

Respiratory

1

Salmeterol

Rheumatology

1

Allopurinol
Azathioprine
Celecoxib
Flurbiprofen
Ibuprofen
Meloxicam
Methotrexate
Piroxicam
Tenoxicam

Urology

1

Darifenacin
Fesoterodine
Mirabegron
Tamsulosin

2

Tolterodine

Other

1

Avatrombopag
Elagolix
Eltrombopag
Lusutrombopag
Oral contraceptives

2

Abrocitinib
Cevimeline
Flibanserin

3

Eliglustat

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
Analgesia	Carisoprodol Celecoxib Flurbiprofen Hydrocodone Ibuprofen Meloxicam Methadone Oxycodone Piroxicam Tenoxicam	Venlafaxine		Alfentanil Fentanyl Morphine Oliceridine	Fentanyl	Oliceridine Venlafaxine	Amitriptyline **Codeine *** Desipramine Imipramine Nortriptyline Tramadol
Autoimmune	Azathioprine Mercaptopurine Methotrexate Siponimod Thioguanine		Tacrolimus	Cyclosporine			
Cancer	Capecitabine Erdafitinib Fluorouracil Mercaptopurine Methotrexate Thioguanine					Gefitinib	Tamoxifen
Cardiovascular	Fluvastatin Lovastatin Nebivolol Pitavastatin Pravastatin Propranolol **Rosuvastatin ***	Metoprolol	Warfarin	Flecainide Metoprolol Propafenone Warfarin	Atorvastatin Warfarin	Carvedilol Flecainide Mavacamten Metoprolol Propafenone Warfarin	**Clopidogrel ***

	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
	Simvastatin						
Endocrinology	Nateglinide						
Gastroenterology	Dronabinol Esomeprazole Methotrexate Ondansetron Rabeprazole			Dexlansoprazole Lansoprazole Meclizine Metoclopramide Omeprazole Pantoprazole		Dexlansoprazole Lansoprazole Meclizine Metoclopramide Omeprazole Pantoprazole	
Infection	Nevirapine			Efavirenz		Efavirenz	Voriconazole
Mental Health	Amoxapine Diazepam Methadone Methylphenidate Mirtazapine Nicotine replacement therapy Olanzapine Protriptyline Quetiapine Viloxazine	Citalopram Escitalopram Fluvoxamine Venlafaxine Vortioxetine		Amphetamine Aripiprazole Aripiprazole lauroxil **Atomoxetine *** Brexpiprazole Citalopram Clozapine Dextromethorphan/ Bupropion Escitalopram Fluvoxamine Haloperidol Iloperidone Lofexidine Paroxetine Pimozide Risperidone Vortioxetine Zuclopenthixol	Bupropion	Amphetamine Aripiprazole **Atomoxetine *** Citalopram Escitalopram Fluoxetine Fluvoxamine Haloperidol Iloperidone Lofexidine Paroxetine Perphenazine Risperidone Venlafaxine Vortioxetine Zuclopenthixol	Amitriptyline Clomipramine Desipramine Doxepin Imipramine Nortriptyline **Sertraline *** Thioridazine Trimipramine
Neurology	Aducanumab Diazepam Donepezil	Dextromethorphan/ Quinidine Metoprolol Venlafaxine		Brivaracetam Clobazam Deutetrabenazine	Brivaracetam	Brivaracetam Clobazam Deutetrabenazine	Amitriptyline

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	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
	Fosphenytoin			Metoprolol		Dextromethorphan/ Quinidine	
	Galantamine			Pitolisant		Metoprolol	
	Lecanemab			Tetrabenazine		Tetrabenazine	
	Phenytoin			Valbenazine		Valbenazine	
	Propranolol					Venlafaxine	
Respiratory	Salmeterol						
Rheumatology	Allopurinol						
	Azathioprine						
	Celecoxib						
	Flurbiprofen						
	Ibuprofen						
	Meloxicam						
	Methotrexate						
	Piroxicam						
	Tenoxicam						
Urology	Darifenacin				Tolterodine	Tolterodine	
	Fesoterodine						
	Mirabegron						
	Tamsulosin						
Other	Avatrombopag			Abrocitinib	Flibanserin	Abrocitinib	Eliglustat
	Elagolix			Flibanserin		Cevimeline	
	Eltrombopag					Flibanserin	
	Lusutrombopag						
	Oral contraceptives						

AttoDiagnostics Ltd Scottow Enterprise Park, Lamas Road, Badersfield, Norfolk, NR10 5FB +44 1603 327 053
info@attodiagnostics.com www.attodiagnostics.com

Summary of Genetic and Phenotype Data

Copy Number Variation

Gene	Reference	Result (Copy/Copies)
CYP2D6_exon9	NG_008376.3 exon 9	2
CYP2D6_intron2	NG_008376.3 intron 2	Unreported

Phenotype Table

Gene	Genotype Result	Phenotype Result OR Genotype Explanation
APOE	E3/E4	E3/E4 Heterozygous
CYP3A4	*1A/*1B	Normal Metabolizer
CYP2D6	*4/*4	Poor Metabolizer
CYP2C9	*1/*1	Normal Metabolizer
CYP2C19	*2/*2	Poor Metabolizer
SLCO1B1	*37/*37	Normal Function
CYP2B6	*6/*6	Poor Metabolizer
CYP3A5	*6/*7	Poor Metabolizer
DPYD	*1/*1	Normal Metabolizer
NUDT15	*1/*1	Normal Metabolizer
TPMT	*1/*1	Normal Metabolizer

Definition of Phenotype

Phenotype Term	Functional Definition	Genetic Definition
Ultrarapid metabolizer	Increased enzyme activity compared to rapid metabolizers	Two increased function alleles, or more than two normal function alleles
Rapid metabolizer	Increased enzyme activity compared to normal metabolizers but less than ultrarapid metabolizers	Combinations of normal function and increased function alleles
Normal metabolizer	Fully functional enzyme activity	Combinations of normal function and decreased function alleles
Intermediate metabolizer	Decreased enzyme activity (activity between normal and poor metabolizers)	Combinations of normal function, decreased function and/or no function alleles
Poor metabolizer	Little to no enzyme activity	Combination of no function alleles and/or decreased function alleles

*Adapted from Caudle KE, et al. Standardizing terms for clinical pharmacogenetic test results: consensus terms from the Clinical Pharmacogenetics Implementation Consortium (CPIC). Genet Med. 2017;19(2):215-223.

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 Level
Cibinqo ReviewGx	Poor metabolizer	CYP2C19	*2/*2	FDA 1[46]; Product monograph (actionable)[36]

Drug-Gene Interactions:

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

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

2 Product Monograph – Actionable PGx: In patients who are known or suspected to be CYP2C19 poor metabolizers, the recommended dosage of abrocitinib is 50 mg once daily. If an adequate response is not achieved with abrocitinib 50 mg once daily, consider increasing the dosage to 100 mg once daily. Discontinue therapy if inadequate response is seen after dosage increase to 100 mg once daily.

Aducanumab	Phenotype	Genetic Test	Results	Source/Evidence Level
Aduhelm ReviewGx	E3/E4 Heterozygous	ApoE	E3/E4	Product monograph (actionable)[8]

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 ReviewGx	Increased analgesic response	OPRM1 rs1799971	A/A	PharmGKB 3[47,48]

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 TreatGx ReviewGx	Normal function	ABCG2 rs2231142	G/G	DPWG[17]

Drug-Gene Interactions:

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

1 DPWG: no recommendation for this ABCG2 genotype.

Amitriptyline	Phenotype	Genetic Test	Results	Source/Evidence Level
Elavil Levate TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	CPIC A[24]; FDA 3[46]
	Poor metabolizer	CYP2C19	*2/*2	CPIC A[24]

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

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

3 CPIC – CYP2C19 Implication: Greatly reduced metabolism of tertiary amines compared to normal metabolizers. Decreased conversion of tertiary amines to secondary amines may affect response or side effects.

3 CPIC – Optional Recommendation: Avoid Amitriptyline use. If use is warranted, utilize therapeutic drug monitoring to guide dose adjustment (strongly recommended for a patient with CYP2D6 ultrarapid, intermediate, or poor metabolism in combination with CYP2C19 ultrarapid, intermediate, or poor metabolism). Recommendations above only apply to higher initial doses of TCAs for treatment of conditions such as depression. Lower dosages are often used for neuropathic pain compared to depressive disorders. Although there is sparse information on how to adjust initial TCA doses based on combined CYP2D6 and CYP2C19 genetic results when treating neuropathic pain, caution should be used when patients have a combination of poor or ultrarapid phenotypes (e.g., a CYP2D6 poor metabolizer also having CYP2C19 ultrarapid or poor metabolism).
Because of the lower dosage, it is less likely that CYP2C19 or CYP2D6 poor metabolizers will experience adverse effects due to supratherapeutic plasma concentrations. Therefore, CPIC recommends no dose modifications for poor metabolizers when prescribed a lower dose for treatment of neuropathic pain, but these patients should be monitored closely for side effects. If larger doses are warranted, CPIC recommends following the gene-based guidelines presented above.

Amoxapine	Phenotype	Genetic Test	Results	Source/Evidence Level
ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 3[46]

Drug-Gene Interactions:

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

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

Amphetamine	Phenotype	Genetic Test	Results	Source/Evidence Level
Adderall Adzenys Dyanavel Evekeo Mydayis TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]

Drug-Gene Interactions:

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

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

2 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 Level
Abilify TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]; Product monograph (actionable)[42]

Drug-Gene Interactions:

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

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 Product Monograph for specific dosing recommendations.

2 FDA Product Monograph – CYP2D6 Actionable PGx: For known CYP2D6 Poor Metabolizers, administer half of usual dose of aripiprazole oral tablet. For known CYP2D6 Poor Metabolizers taking concomitant strong CYP3A4 inhibitors, administer a quarter of the usual dose.

Aripiprazole lauroxil	Phenotype	Genetic Test	Results	Source/Evidence Level
Aristada TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]; Product monograph (actionable)[2]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require a reduced dose

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

2 FDA Product Monograph – CYP2D6 Actionable PGx: For patients known to be poor metabolizers of CYP2D6: Reduce dose of aripiprazole lauroxil injection to 441 mg from 662 mg, 882 mg, or 1064 mg. No dosage adjustment is necessary in patients taking 441 mg aripiprazole lauroxil injection, if tolerated.

Atomoxetine	Phenotype	Genetic Test	Results	Source/Evidence Level
Strattera TreatGx ReviewGx	Poor metabolizer	CYP2D6 (Activity Score)	*4/*4	CPIC A[11]; FDA 1[46]

Drug-Gene Interactions:

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

2 CPIC – Implication: Significantly decreased metabolism of atomoxetine may result in higher concentrations as compared to non-poor metabolizers. This may increase the occurrence of side effects, but also a greater improvement of ADHD symptoms as compared to non-poor metabolizers in those who tolerate treatment. Poor metabolizer status is associated with lower final dose requirements as compared to non-poor metabolizers.

2 ADULTS: CPIC – Moderate Recommendation: Initiate with a dose of 40 mg/day and if no clinical response and in the absence of adverse events after 2 weeks increase dose to 80 mg/day. If response is inadequate after 2 weeks, consider obtaining a plasma concentration 2 to 4 hours after dosing. If concentration is <200 ng/mL, consider a proportional dose increase to achieve a concentration to approach 400 ng/mL. If unacceptable side effects are present at any time, consider a reduction in dose.
PEDIATRICS: CPIC – Strong Recommendation: Initiate with a dose of 0.5 mg/kg/day and if no clinical response and in the absence of adverse events after 2 weeks, consider obtaining a plasma concentration 4 hours 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. If unacceptable side effects are present at any time, consider a reduction in dose.
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.

Atorvastatin	Phenotype	Genetic Test	Results	Source/Evidence Level
Lipitor	Normal function	SLCO1B1	*37/*37	CPIC A[13]; FDA 3[46]
	Decreased response	ApoE rs7412	C/C	PharmGKB 2B[47,48]

TreatGx
ReviewGx

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	Normal metabolizer	CYP2C9	*1/*1	FDA 3[46]
	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]

ReviewGx

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 phenotype in the FDA PGx Table

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

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

Azathioprine	Phenotype	Genetic Test	Results	Source/Evidence Level
Azasan Imuran	Normal metabolizer	TPMT	*1/*1	CPIC A[39,44]; FDA 1[46]
	Normal metabolizer	NUDT15	*1/*1	CPIC A[39,44]; FDA 1[46]

TreatGx
ReviewGx

Drug-Gene Interactions:

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

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

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

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

Brexpiprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Rexulti	Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]; Product monograph (actionable)[3]

TreatGx
ReviewGx

Drug-Gene Interactions:

Moderate drug-gene interaction: may require a reduced dose

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

2 FDA Product Monograph – CYP2D6 Actionable PGx: For known CYP2D6 Poor Metabolizers, administer half of usual dose of brexpiprazole oral tablet. For known CYP2D6 Poor Metabolizers taking concomitant strong CYP3A4 inhibitors, administer a quarter of the usual dose.

Brivaracetam	Phenotype	Genetic Test	Results	Source/Evidence Level
Briviact Brivlera	Poor metabolizer	CYP2C19	*2/*2	FDA 1[46]

ReviewGx

Drug-Gene Interactions:

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

2 CYP2C19 poor metabolizer: greatly reduced metabolism of Brivaracetam to less active compounds

2 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

Bupropion	Phenotype	Genetic Test	Results	Source/Evidence Level
Wellbutrin Zyban	Less likely to quit smoking compared to G/G	ANKK1/DRD2 rs1800497	A/A	PharmGKB 3[47,48]

TreatGx
ReviewGx

Drug-Gene Interactions:

Moderate drug-gene interaction: may reduce efficacy

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

Capecitabine	Phenotype	Genetic Test	Results	Source/Evidence Level
Xeloda	Normal metabolizer	DPYD	*1/*1	CPIC A[4]; FDA 1[46]

ReviewGx

Drug-Gene Interactions:

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

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

1 DPYD alleles do not indicate changes from recommended dose

Carisoprodol	Phenotype	Genetic Test	Results	Source/Evidence Level

ReviewGx

	Poor metabolizer	CYP2C19	*2/*2	FDA 3[46]

Drug-Gene Interactions:

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

1 CYP2C19 poor metabolizer: reduced metabolism of Carisoprodol to less active compounds leads to higher plasma concentrations of active drug

1 There is a potential impact on pharmacokinetic properties. The impact of CYP2C19 variants on the safety of Carisoprodol has not been established

Carvedilol	Phenotype	Genetic Test	Results	Source/Evidence Level
Coreg	Poor metabolizer	CYP2D6	*4/*4	FDA 2[46]

TreatGx
ReviewGx

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

- 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)
- Data indicate a potential impact on patient safety

Celecoxib	Phenotype	Genetic Test	Results	Source/Evidence Level
Celebrex	Normal metabolizer	CYP2C9 (Star Alleles)	*1/*1	CPIC A[45]; FDA 1[46]

TreatGx
ReviewGx

Drug-Gene Interactions:

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

- CYP2C9 alleles do not indicate changes from recommended dose

Cevimeline	Phenotype	Genetic Test	Results	Source/Evidence Level
Evoxac	Poor metabolizer	CYP2D6	*4/*4	FDA 2[46]

ReviewGx

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

- 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
- Data indicate a potential impact on patient safety

Citalopram	Phenotype	Genetic Test	Results	Source/Evidence Level
Celexa	Poor metabolizer	CYP2C19	*2/*2	CPIC A[10]; FDA 1[46]

TreatGx
ReviewGx

Drug-Gene Interactions:

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

- Reduced metabolism of citalopram and escitalopram to less active compounds when compared with CYP2C19 normal and intermediate metabolizers. Higher plasma concentrations may increase the probability of side effects.
- Consider a clinically appropriate antidepressant not predominantly metabolized by CYP2C19. If citalopram or escitalopram are clinically appropriate, consider a lower starting dose, slower titration schedule, and 50% reduction of the standard maintenance dose as compared with normal metabolizers (per CPIC strong recommendation).

Clobazam	Phenotype	Genetic Test	Results	Source/Evidence Level
Onfi Sympazan	Poor metabolizer	CYP2C19	*2/*2	FDA 1[46]; Product monograph (actionable)[30]

ReviewGx

Drug-Gene Interactions:

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

2 FDA PGx Table Section 1 – CYP2C19 Therapeutic Management Recommendations: Results in higher systemic active metabolite concentrations. Poor metabolism results in higher adverse reaction risk. Dosage adjustment is recommended. Refer to FDA Product Monograph for specific dosing recommendations.

2 FDA Product Monograph – Actionable PGx: In CYP2C19 poor metabolizers, levels of N-desmethylclobazam, clobazam's active metabolite, will be increased. Therefore, in patients known to be CYP2C19 poor metabolizers, the starting dose should be 5 mg/day and dose titration should proceed slowly according to weight, but to half the standard dose, as tolerated. If necessary and based upon clinical response, an additional titration to the maximum dose (20 mg/day or 40 mg/day, depending on the weight group) may be started on day 21.

Clomipramine	Phenotype	Genetic Test	Results	Source/Evidence Level
Anafranil	Poor metabolizer	CYP2D6	*4/*4	CPIC B[24]; FDA 3[46]
	Poor metabolizer	CYP2C19	*2/*2	CPIC B[24]

ReviewGx

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

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

3 CPIC – CYP2C19 Implication: Greatly reduced metabolism of tertiary amines compared to normal metabolizers. Decreased conversion of tertiary amines to secondary amines may affect response or side effects.

3 CPIC – Optional Recommendation: Avoid Clomipramine use. If use is warranted, utilize therapeutic drug monitoring to guide dose adjustment (strongly recommended for a patient with CYP2D6 ultrarapid, intermediate, or poor metabolism in combination with CYP2C19 ultrarapid, intermediate, or poor metabolism).
Recommendations above only apply to higher initial doses of TCAs for treatment of conditions such as depression. Lower dosages are often used for neuropathic pain compared to depressive disorders.
Although there is sparse information on how to adjust initial TCA doses based on combined CYP2D6 and CYP2C19 genetic results when treating neuropathic pain, caution should be used when patients have a combination of poor or ultrarapid phenotypes (e.g., a CYP2D6 poor metabolizer also having CYP2C19 ultrarapid or poor metabolism).
Because of the lower dosage, it is less likely that CYP2C19 or CYP2D6 poor metabolizers will experience adverse effects due to supratherapeutic plasma concentrations. Therefore, CPIC recommends no dose modifications for poor metabolizers when prescribed a lower dose for treatment of neuropathic pain, but these patients should be monitored closely for side effects. If larger doses are warranted, CPIC recommends following the gene-based guidelines presented above.

Clopidogrel	Phenotype	Genetic Test	Results	Source/Evidence Level
Plavix	Poor metabolizer	CYP2C19	*2/*2	CPIC A[28]; FDA 1[46]

TreatGx
ReviewGx

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

3 CPIC – Implication: Significantly reduced clopidogrel active metabolite formation; increased on-treatment platelet reactivity; increased risk for adverse cardiac and cerebrovascular events.

3 CPIC – Strong Recommendation for acute coronary syndrome (ACS) and/or percutaneous coronary intervention (PCI); Moderate Recommendation for non-ACS/non-PCI cardiovascular indications: Avoid clopidogrel if possible. Use prasugrel or ticagrelor at standard dose if no contraindication.
ACS and/or PCI includes patients undergoing PCI for an ACS or non-ACS (elective) indication. Non-ACS, non-PCI cardiovascular indications include peripheral arterial disease and stable coronary artery disease following a recent myocardial infarction outside the setting of PCI.

Clozapine	Phenotype	Genetic Test	Results	Source/Evidence Level
Clozaril Fazaclo ODT Versacloz TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]
	Decreased risk of developing metabolic syndrome	HTR2C rs1414334	G/G	PharmGKB 3[47,48]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require a reduced dose

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. 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 Level
Codeine Contin Tylenol with Codeine No. 2/3/4 TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	CPIC A[14]; FDA 1[46]; FDA 2[46]

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

3. CPIC – Implication: Greatly reduced morphine formation leading to diminished analgesia.
3. CPIC – Strong Recommendation: Avoid codeine use because of possibility of diminished analgesia. If opioid use is warranted, consider a non-tramadol opioid.

Cyclosporine	Phenotype	Genetic Test	Results	Source/Evidence Level
Neoral Sandimmune ReviewGx	Poor metabolizer	CYP3A5	*6/*7	PharmGKB 3[47,48]

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).

Darifenacin	Phenotype	Genetic Test	Results	Source/Evidence Level
Enablex TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 3[46]

Drug-Gene Interactions:

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

1. CYP2D6 poor metabolizer: reduced metabolism of Darifenacin leads to higher plasma concentrations
1. 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 Level
Norpramin TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	CPIC B[24]; FDA 3[46]

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

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

2 CPIC – Optional Recommendation: Avoid tricyclic use due to potential for side effects. Consider alternative drug not metabolized by CYP2D6. If a TCA is warranted, consider a 50% reduction of recommended starting dose. Patients may receive an initial low dose of a tricyclic, which is then increased over several days to the recommended steady-state dose. The starting dose in this guideline refers to the recommended steady-state dose. Utilize therapeutic drug monitoring to guide dose adjustments. Titrate dose to observed clinical response with symptom improvement and minimal (if any) side effects.
Recommendations above only apply to higher initial doses of TCAs for treatment of conditions such as depression. Lower dosages are often used for neuropathic pain compared to depressive disorders.
Because of the lower dosage, it is less likely that CYP2D6 poor metabolizers will experience adverse effects due to supratherapeutic plasma concentrations. Therefore, CPIC recommends no dose modifications for poor metabolizers when prescribed a lower dose for treatment of neuropathic pain, but these patients should be monitored closely for side effects. If larger doses are warranted, CPIC recommends following the gene-based guidelines presented above.

Deutetrabenazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Austedo ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]

Drug-Gene Interactions:

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

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

2 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 Level
Dexilant TreatGx ReviewGx	Poor metabolizer	CYP2C19	*2/*2	CPIC B[29]; FDA 3[46]

Drug-Gene Interactions:

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

2 CPIC – Implication: Likely increased plasma concentration of PPI compared with CYP2C19 NMs; likely increased chance of efficacy and potentially toxicity.

2 CPIC – Moderate Recommendation: Initiate standard starting daily dose. For chronic therapy (>12 weeks) and efficacy achieved, consider 50% reduction in daily dose and monitor for continued efficacy.

Dextromethorphan/ Bupropion	Phenotype	Genetic Test	Results	Source/Evidence Level
Auvelity ReviewGx	Poor metabolizer	CYP2D6	*4/*4	Product monograph (actionable)[6]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require a reduced dose

2 FDA Product Monograph – CYP2D6 Actionable PGx: The recommended dosage for patients known to be CYP2D6 poor metabolizers (PMs) is one tablet once daily in the morning (45 mg of dextromethorphan hydrobromide and 105 mg of bupropion hydrochloride), reduced from usual maintenance dose of one tablet twice daily for non-PMs. Dosage adjustment is recommended because CYP2D6 PMs have higher dextromethorphan concentrations than extensive/intermediate metabolizers.

Dextromethorphan/ Quinidine	Phenotype	Genetic Test	Results	Source/Evidence Level
Nuedexta ReviewGx	Poor metabolizer	CYP2D6	*4/*4	Product monograph (actionable)[5]

Drug-Gene Interactions:

Moderate drug-gene interaction: consider alternative medications, may increase adverse events

2 FDA Product Monograph – CYP2D6 Testing Recommended: The quinidine component is intended to inhibit CYP2D6 so that higher exposure to dextromethorphan can be achieved compared to when dextromethorphan is given alone. The quinidine component is not expected to contribute to the effectiveness in CYP2D6 poor metabolizers (PMs), but adverse events of the quinidine are still possible. In those patients who may be at risk of significant toxicity due to quinidine, genotyping to determine if they are PMs should be considered prior to making the decision to treat.

Diazepam	Phenotype	Genetic Test	Results	Source/Evidence Level
Diastat Valium TreatGx ReviewGx	Poor metabolizer	CYP2C19	*2/*2	FDA 3[46]

Drug-Gene Interactions:

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

1 FDA PGx Table Section 3 – CYP2C19 Potential Impact on Pharmacokinetic Properties Only: May affect systemic concentrations.

Donepezil	Phenotype	Genetic Test	Results	Source/Evidence Level
Aricept TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 3[46]

Drug-Gene Interactions:

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

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

1 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 Level
Silenor Sinequan TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	CPIC B[24]; FDA 3[46]
	Poor metabolizer	CYP2C19	*2/*2	CPIC B[24]; FDA 3[46]

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

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

3 CPIC – CYP2C19 Implication: Greatly reduced metabolism of tertiary amines compared to normal metabolizers. Decreased conversion of tertiary amines to secondary amines may affect response or side effects.

3 CPIC – Optional Recommendation: Avoid Doxepin use. If use is warranted, utilize therapeutic drug monitoring to guide dose adjustment (strongly recommended for a patient with CYP2D6 ultrarapid, intermediate, or poor metabolism in combination with CYP2C19 ultrarapid, intermediate, or poor metabolism).
Recommendations above only apply to higher initial doses of TCAs for treatment of conditions such as depression. Lower dosages are often used for neuropathic pain compared to depressive disorders.
Although there is sparse information on how to adjust initial TCA doses based on combined CYP2D6 and CYP2C19 genetic results when treating neuropathic pain, caution should be used when patients have a combination of poor or ultrarapid phenotypes (e.g., a CYP2D6 poor metabolizer also having CYP2C19 ultrarapid or poor metabolism).
Because of the lower dosage, it is less likely that CYP2C19 or CYP2D6 poor metabolizers will experience adverse effects due to supratherapeutic plasma concentrations. Therefore, CPIC recommends no dose modifications for poor metabolizers when prescribed a lower dose for treatment of neuropathic pain, but these patients should be monitored closely for side effects. If larger doses are warranted, CPIC recommends following the gene-based guidelines presented above.

Dronabinol	Phenotype	Genetic Test	Results	Source/Evidence Level
Marinol Syndros	Normal metabolizer	CYP2C9	*1/*1	FDA 1[46]

ReviewGx

Drug-Gene Interactions:

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

1 CYP2C9 alleles do not indicate changes from recommended dose

Efavirenz	Phenotype	Genetic Test	Results	Source/Evidence Level
Sustiva	Poor metabolizer	CYP2B6	*6/*6	CPIC A[15]; FDA 2[46]

ReviewGx

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; significantly increased risk of CNS adverse events and treatment discontinuation.

2 CPIC – Moderate Recommendation: Consider initiating efavirenz with decreased dose of 400 or 200 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	*37/*37	FDA 3[46]

ReviewGx

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	Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]

ReviewGx

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

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

2 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 Level
Promacta Revolade	Normal Factor V Leiden	Factor V rs6025	C/C	Product monograph (actionable)[35]

ReviewGx

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 ReviewGx	Normal metabolizer	CYP2C9 (Star Alleles)	*1/*1	FDA 1[46]

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 TreatGx ReviewGx	Poor metabolizer	CYP2C19	*2/*2	CPIC A[10]; FDA 3[46]

Drug-Gene Interactions:

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

2 Reduced metabolism of citalopram and escitalopram to less active compounds when compared with CYP2C19 normal and intermediate metabolizers. Higher plasma concentrations may increase the probability of side effects.

2 Consider a clinically appropriate antidepressant not predominantly metabolized by CYP2C19. If citalopram or escitalopram are clinically appropriate, consider a lower starting dose, slower titration schedule, and 50% reduction of the standard maintenance dose as compared with normal metabolizers (per CPIC strong recommendation).

Esomeprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Nexium TreatGx ReviewGx	Poor metabolizer	CYP2C19	*2/*2	FDA 3[46]

Drug-Gene Interactions:

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

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

Fentanyl	Phenotype	Genetic Test	Results	Source/Evidence Level
Actiq Duragesic Fentora Sublimaze ReviewGx	Decreased analgesic response	OPRM1 rs1799971	A/A	PharmGKB 3[47,48]

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 TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 3[46]

Drug-Gene Interactions:

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

1 CYP2D6 poor metabolizer: reduced metabolism of Fesoterodine leads to higher plasma concentrations

1 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 Level
Tambocor	Poor metabolizer	CYP2D6	*4/*4	DPWG[17]

TreatGx
ReviewGx

Drug-Gene Interactions:

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

- 2 CYP2D6 poor metabolizer: greatly reduced metabolism of Flecainide to less active compounds
- 2 Higher plasma concentrations of active drug may increase the risk of adverse drug reactions
- 2 Reduce the standard dose by 50%, record electrocardiogram, and monitor plasma concentration

Flibanserin	Phenotype	Genetic Test	Results	Source/Evidence Level
Addyi	Poor metabolizer	CYP2C19	*2/*2	FDA 1[46]

ReviewGx

Drug-Gene Interactions:

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

- 2 CYP2C19 poor metabolizer: greatly reduced metabolism of Flibanserin to less active compounds
- 2 Higher plasma concentrations of active drug may increase the risk of hypotension, syncope, and CNS depression
- 2 This drug has an FDA therapeutic recommendation, refer to drug monograph or FDA labelling for dosing recommendations

Fluorouracil	Phenotype	Genetic Test	Results	Source/Evidence Level
Carac Efudex Fluoroplex Tolak	Normal metabolizer	DPYD	*1/*1	CPIC A[4]; FDA 1[46]

ReviewGx

Drug-Gene Interactions:

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

- 1 DPYD alleles indicate normal DPD activity and typical risk for Fluorouracil toxicity
- 1 DPYD alleles do not indicate changes from recommended dose

Fluoxetine	Phenotype	Genetic Test	Results	Source/Evidence Level
Prozac	Poor metabolizer	CYP2D6	*4/*4	Product monograph (actionable)[12]

TreatGx
ReviewGx

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

- 2 FDA Product Monograph – Actionable PGx: Fluoxetine is primarily metabolized by CYP2D6. It should be used with caution in patients with congenital long QT syndrome; a previous history of QT prolongation and other conditions that predispose to QT prolongation and ventricular arrhythmia, which include conditions that predispose to increased fluoxetine exposure (overdose, hepatic impairment, use of CYP2D6 inhibitors, CYP2D6 poor metabolizer status, or use of other highly protein-bound drugs).

Flurbiprofen	Phenotype	Genetic Test	Results	Source/Evidence Level
Ansaid	Normal metabolizer	CYP2C9 (Star Alleles)	*1/*1	CPIC A[45]; FDA 1[46]

TreatGx
ReviewGx

Drug-Gene Interactions:

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

- 1 CYP2C9 alleles do not indicate changes from recommended dose

Fluvastatin	Phenotype	Genetic Test	Results	Source/Evidence Level
Lescol	Normal metabolizer	CYP2C9	*1/*1	CPIC A[13]
	Normal function	SLCO1B1	*37/*37	CPIC A[13]

TreatGx
ReviewGx

Drug-Gene Interactions:

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

1. CPIC – CYP2C9 Implication: Normal exposure.
1. CPIC – SLCO1B1 Implication: Typical myopathy risk and Fluvastatin exposure.
1. CPIC – Strong Recommendation: Prescribe desired starting dose and adjust doses of fluvastatin based on disease-specific guidelines.
   The potential for drug-drug interactions and dose limits based on renal and hepatic function and ancestry should be evaluated prior to initiating a statin.

Fluvoxamine	Phenotype	Genetic Test	Results	Source/Evidence Level
Luvox	Poor metabolizer	CYP2D6	*4/*4	CPIC B[10]; FDA 3[46]

TreatGx
ReviewGx

Drug-Gene Interactions:

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

2. 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 Level
Cerebyx	Normal metabolizer	CYP2C9	*1/*1	CPIC A[27]; FDA 1[46]

ReviewGx

Drug-Gene Interactions:

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

1. CPIC – CYP2C9 Implication: Normal Fosphenytoin metabolism
1. CPIC – Strong Recommendation: No adjustments needed from typical dosing strategies. Subsequent doses should be adjusted according to therapeutic drug monitoring, response, and side effects. An HLA-B*15:02 negative test does not eliminate the risk of Fosphenytoin-induced Stevens-Johnson syndrome and toxic epidermal necrolysis (SJS/TEN), and patients should be carefully monitored according to standard practice.

Galantamine	Phenotype	Genetic Test	Results	Source/Evidence Level
Razadyne	Poor metabolizer	CYP2D6	*4/*4	FDA 3[46]

TreatGx
ReviewGx

Drug-Gene Interactions:

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

1. CYP2D6 poor metabolizer: reduced metabolism of Galantamine to less active compounds leads to higher plasma concentrations of active drug
1. There is a potential impact on pharmacokinetic properties. The impact of CYP2D6 variants on the safety of Galantamine has not been established
1. Titrate dose based on tolerability

Gefitinib	Phenotype	Genetic Test	Results	Source/Evidence Level
Iressa	Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]

ReviewGx

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 Level
Haldol	Poor metabolizer	CYP2D6	*4/*4	DPWG[17]

TreatGx
ReviewGx

Drug-Gene Interactions:

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

2 DPWG – 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 Level
Anexsia Hycodan Hysingla Zohydro	Poor metabolizer	CYP2D6	*4/*4	CPIC B[14]

Drug-Gene Interactions:

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

1 CPIC – Implication: Decreased metabolism of hydrocodone to active metabolite, hydromorphone, but there is insufficient evidence to determine if these effects on pharmacokinetics translate into decreased analgesia or side effects.

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

TreatGx
ReviewGx

Ibuprofen	Phenotype	Genetic Test	Results	Source/Evidence Level
Advil Caldolor Duexis Motrin IB NeoProfen	Normal metabolizer	CYP2C9 (Star Alleles)	*1/*1	CPIC A[45]; FDA 3[46]

Drug-Gene Interactions:

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

1 CYP2C9 alleles do not indicate changes from recommended dose

TreatGx
ReviewGx

Iloperidone	Phenotype	Genetic Test	Results	Source/Evidence Level
Fanapt	Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]

Drug-Gene Interactions:

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

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%.

TreatGx
ReviewGx

Imipramine	Phenotype	Genetic Test	Results	Source/Evidence Level
Tofranil	Poor metabolizer	CYP2D6	*4/*4	CPIC B[24]; FDA 3[46]
TreatG: ReviewG:	Poor metabolizer	CYP2C19	*2/*2	CPIC B[24]

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

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

3 CPIC – CYP2C19 Implication: Greatly reduced metabolism of tertiary amines compared to normal metabolizers. Decreased conversion of tertiary amines to secondary amines may affect response or side effects.

3 CPIC – Optional Recommendation: Avoid Imipramine use. If use is warranted, utilize therapeutic drug monitoring to guide dose adjustment (strongly recommended for a patient with CYP2D6 ultrarapid, intermediate, or poor metabolism in combination with CYP2C19 ultrarapid, intermediate, or poor metabolism). Recommendations above only apply to higher initial doses of TCAs for treatment of conditions such as depression. Lower dosages are often used for neuropathic pain compared to depressive disorders. Although there is sparse information on how to adjust initial TCA doses based on combined CYP2D6 and CYP2C19 genetic results when treating neuropathic pain, caution should be used when patients have a combination of poor or ultrarapid phenotypes (e.g., a CYP2D6 poor metabolizer also having CYP2C19 ultrarapid or poor metabolism).
Because of the lower dosage, it is less likely that CYP2C19 or CYP2D6 poor metabolizers will experience adverse effects due to supratherapeutic plasma concentrations. Therefore, CPIC recommends no dose modifications for poor metabolizers when prescribed a lower dose for treatment of neuropathic pain, but these patients should be monitored closely for side effects. If larger doses are warranted, CPIC recommends following the gene-based guidelines presented above.

Lansoprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Prevacid	Poor metabolizer	CYP2C19	*2/*2	CPIC A[29]; FDA 3[46]
TreatG: ReviewG:				

Drug-Gene Interactions:

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

2 CPIC – Implication: Likely increased plasma concentration of PPI compared with CYP2C19 NMs; likely increased chance of efficacy and potentially toxicity.

2 CPIC – Moderate Recommendation: Initiate standard starting daily dose. For chronic therapy (>12 weeks) and efficacy achieved, consider 50% reduction in daily dose and monitor for continued efficacy.

Lecanemab	Phenotype	Genetic Test	Results	Source/Evidence Level
Leqembi ReviewG:	E3/E4 Heterozygous	ApoE	E3/E4	Product monograph (actionable)[18]

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	Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]
ReviewG:				

Drug-Gene Interactions:

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

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

2 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 Level
Altoprev	Normal function	SLCO1B1	*37/*37	CPIC A[13]

TreatGx
ReviewGx

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.

Lusutrombopag	Phenotype	Genetic Test	Results	Source/Evidence Level
Mupleta	Normal Factor II	Factor II rs1799963	G/G	Product monograph (actionable)[43]
	Normal Factor V Leiden	Factor V rs6025	C/C	Product monograph (actionable)[43]

ReviewGx

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	Poor metabolizer	CYP2C19	*2/*2	FDA 2[46]

ReviewGx

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

2 FDA PGx Table Section 2 – Potential Impact on Safety or Response: Results in higher systemic concentrations and may have higher adverse reaction risk (heart failure). Dosage is based on individual response. The dose titration and monitoring schedule accounts for differences due to CYP2C19 genetic variation, so adjustments based on CYP2C19 genotype are not necessary. Refer to FDA Product Monograph for specific dosing recommendations and monitoring.

Meclizine	Phenotype	Genetic Test	Results	Source/Evidence Level
Antivert	Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]

ReviewGx

Drug-Gene Interactions:

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

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

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

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

Meloxicam	Phenotype	Genetic Test	Results	Source/Evidence Level
Anjeso Mobic Qmiiz ODT Vivlodex	Normal metabolizer	CYP2C9 (Star Alleles)	*1/*1	CPIC A[45]; FDA 1[46]

TreatGx
ReviewGx

Drug-Gene Interactions:

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

1 CYP2C9 alleles do not indicate changes from recommended dose

Mercaptopurine	Phenotype	Genetic Test	Results	Source/Evidence Level
Purinethol Purixan	Normal metabolizer	TPMT	*1/*1	CPIC A[39,44]; FDA 1[46]
	Normal metabolizer	NUDT15	*1/*1	CPIC A[39,44]; FDA 1[46]

TreatGx
ReviewGx

Drug-Gene Interactions:

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

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

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

1 CPIC – Strong Recommendation: Start with normal starting dose (e.g., 75 mg/m2/day or 1.5 mg/kg/day) and adjust doses of mercaptopurine (and of any other myelosuppressive therapy) without any special emphasis on mercaptopurine compared to other agents. Allow at least 2 weeks to reach steady-state after each dose adjustment.
Normal starting doses vary by race/ethnicity and treatment regimens.

Methadone	Phenotype	Genetic Test	Results	Source/Evidence Level
Methadose	Poor metabolizer	CYP2B6	*6/*6	PharmGKB 2A[40,47,48]

ReviewGx

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	Decreased risk of toxicity	MTHFR rs1801133	G/G	PharmGKB 2A[47,48]

TreatGx
ReviewGx

Drug-Gene Interactions:

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

1 PharmGKB – Clinical Annotation (Level 2A Toxicity): Patients with the MTHFR rs1801133 G/G genotype and cancer or arthritis who are treated with methotrexate may have a decreased risk of toxicity as compared to patients with the A/G or 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

Aptensio
Concerta
Cotempla
Daytrana
Jornay
Metadate
Methylin
Quillichew
Quillivant
Relexxiii
Ritalin

TreatG
ReviewG

Phenotype	Genetic Test	Results	Source/Evidence Level
Better response compared to C/C	ADRA2A rs1800544	G/G	PharmGKB 4[47,48]
No significant association to response	COMT rs4680	G/A	PharmGKB 4[47,48]

Drug-Gene Interactions:

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

1 PharmGKB – Clinical Annotation (Level 4 Efficacy): Patients (mainly pediatric patients) with the ADRA2A rs1800544 G/G genotype and attention deficit hyperactivity disorder (ADHD) may have a better response to methylphenidate treatment as compared to patients with the C/C 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.

1 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

Metonia
Reglan

TreatG
ReviewG

Phenotype	Genetic Test	Results	Source/Evidence Level
Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]

Drug-Gene Interactions:

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

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

2 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

Metoprolol

Kapspargo Sprinkle
Lopressor
Toprol-XL

TreatG
ReviewG

Phenotype	Genetic Test	Results	Source/Evidence Level
Poor metabolizer	CYP2D6	*4/*4	CPIC B[16]

Drug-Gene Interactions:

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

2 CPIC – Implication: Decreased metabolism of metoprolol leading to markedly increased drug concentrations; this leads to greater heart rate and blood pressure reductions, but the effect on clinical outcomes is unclear.

2 CPIC – Moderate Recommendation: Initiate therapy with lowest recommended starting dose. Carefully titrate dose upward to clinical effect or guideline-recommended dose; monitor more closely for bradycardia. Alternatively, consider selecting another beta-blocker.

Mirabegron

Myrbetriq

TreatG
ReviewG

Phenotype	Genetic Test	Results	Source/Evidence Level
Poor metabolizer	CYP2D6	*4/*4	FDA 3[46]

Drug-Gene Interactions:

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

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

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

Mirtazapine	Phenotype	Genetic Test	Results	Source/Evidence Level
Remeron	Poor metabolizer	CYP2D6	*4/*4	DPWG[17]; PharmGKB 2A[47,48]

TreatG
ReviewG

Drug-Gene Interactions:

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

1 DPWG: The higher plasma concentration of mirtazapine does not result - or hardly results - in an increase in the side effects.

1 DPWG "No Recommendation": NO action is required for this gene-drug interaction.

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[47,48]

TreatG
ReviewG

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
	Normal metabolizer	CYP2C9	*1/*1	FDA 1[46]

ReviewG

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	Poor metabolizer	CYP2D6	*4/*4	FDA 3[46]

TreatG
ReviewG

Drug-Gene Interactions:

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

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

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

Nevirapine	Phenotype	Genetic Test	Results	Source/Evidence Level
Viramune	Decreased risk for SJS/TEN	CYP2B6 rs28399499	T/T	PharmGKB 2A[47,48]
	Decreased clearance and increased exposure compared to G/G	CYP2B6 rs3745274	T/T	PharmGKB 2A[47,48]

ReviewGx

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 T/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	Increased likelihood of smoking cessation compared to G/G	ANKK1/DRD2 rs1800497	A/A	PharmGKB 3[47,48]

TreatGx
ReviewGx

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 A/A 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 Level
Aventyl Pamelor	Poor metabolizer	CYP2D6	*4/*4	CPIC A[24]; FDA 3[46]

TreatGx
ReviewGx

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

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

3 CPIC – Strong Recommendation: Avoid tricyclic use due to potential for side effects. Consider alternative drug not metabolized by CYP2D6. If a TCA is warranted, consider a 50% reduction of recommended starting dose. Patients may receive an initial low dose of a tricyclic, which is then increased over several days to the recommended steady-state dose. The starting dose in this guideline refers to the recommended steady-state dose. Utilize therapeutic drug monitoring to guide dose adjustments. Titrate dose to observed clinical response with symptom improvement and minimal (if any) side effects.
Recommendations above only apply to higher initial doses of TCAs for treatment of conditions such as depression. Lower dosages are often used for neuropathic pain compared to depressive disorders.
Because of the lower dosage, it is less likely that CYP2D6 poor metabolizers will experience adverse effects due to supratherapeutic plasma concentrations. Therefore, CPIC recommends no dose modifications for poor metabolizers when prescribed a lower dose for treatment of neuropathic pain, but these patients should be monitored closely for side effects. If larger doses are warranted, CPIC recommends following the gene-based guidelines presented above.

Olanzapine	Phenotype	Genetic Test	Results	Source/Evidence Level
Zyprexa TreatG ReviewG	May have decreased time until response compared to T/T	DRD2 rs1799978	C/T	PharmGKB 3[47,48]

Drug-Gene Interactions:

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

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

Oliceridine	Phenotype	Genetic Test	Results	Source/Evidence Level
Olinvyk ReviewG	Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]

Drug-Gene Interactions:

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

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 Level
Losec Olex Prilosec TreatG ReviewG	Poor metabolizer	CYP2C19	*2/*2	CPIC A[29]; FDA 3[46]

Drug-Gene Interactions:

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

2 CPIC – Implication: Likely increased plasma concentration of PPI compared with CYP2C19 NMs; likely increased chance of efficacy and potentially toxicity.

2 CPIC – Moderate Recommendation: Initiate standard starting daily dose. For chronic therapy (>12 weeks) and efficacy achieved, consider 50% reduction in daily dose and monitor for continued efficacy.

Ondansetron	Phenotype	Genetic Test	Results	Source/Evidence Level
Zofran Zuplenz ReviewG	Poor metabolizer	CYP2D6	*4/*4	CPIC A[7]

Drug-Gene Interactions:

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

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

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

Oral contraceptives	Phenotype	Genetic Test	Results	Source/Evidence Level
ReviewGx	Decreased risk for DVT	Factor II rs1799963	G/G	PharmGKB 2B[47,48]
	Decreased risk of thrombosis (normal Factor V)	Factor V rs6025	C/C	PharmGKB 2B[47,48]

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.

Oxycodone	Phenotype	Genetic Test	Results	Source/Evidence Level
OxyContin Roxicodone Roxybond Xtampza ER	Poor metabolizer	CYP2D6	*4/*4	PharmGKB 2A[14,47,48]
TreatGx ReviewGx				

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): Decreased metabolism of oxycodone to active metabolite oxymorphone, but this does not appear to translate into decreased analgesia or side effects.
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: No recommendation for this CYP2D6 phenotype and oxycodone therapy because of weak evidence regarding adverse events or analgesia. Inconsistent evidence indicates both supporting and non-supporting evidence for an association between oxycodone use and adverse events, or analgesia.

Pantoprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Pantoloc Protonix Tecta	Poor metabolizer	CYP2C19	*2/*2	CPIC A[29]; FDA 1[46]
TreatGx ReviewGx				

Drug-Gene Interactions:

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

2. CPIC – Implication: Likely increased plasma concentration of PPI compared with CYP2C19 NMs; likely increased chance of efficacy and potentially toxicity.

2. CPIC – Moderate Recommendation: Initiate standard starting daily dose. For chronic therapy (>12 weeks) and efficacy achieved, consider 50% reduction in daily dose and monitor for continued efficacy.

Paroxetine	Phenotype	Genetic Test	Results	Source/Evidence Level
Brisdelle Paxil Pexeva	Poor metabolizer	CYP2D6	*4/*4	CPIC A[10]; FDA 3[46]
TreatGx ReviewGx				

Drug-Gene Interactions:

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

2. 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.

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 Level
TreatG ReviewG	Poor metabolizer	CYP2D6	*4/*4	FDA 2[46]

Drug-Gene Interactions:

Moderate drug-gene interaction: may increase adverse events

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 Level
Dilantin Tremytoine Phenytek ReviewG	Normal metabolizer	CYP2C9	*1/*1	CPIC A[27]; FDA 1[46]

Drug-Gene Interactions:

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

1 CPIC – CYP2C9 Implication: Normal Phenytoin metabolism

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

Pimozide	Phenotype	Genetic Test	Results	Source/Evidence Level
Orap TreatG ReviewG	Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require a reduced dose

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 Level
Feldene TreatG ReviewG	Normal metabolizer	CYP2C9 (Star Alleles)	*1/*1	CPIC A[45]; FDA 1[46]

Drug-Gene Interactions:

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

1 CYP2C9 alleles do not indicate changes from recommended dose

Pitavastatin	Phenotype	Genetic Test	Results	Source/Evidence Level
Livalo Zypitamag TreatG ReviewG	Normal function	SLCO1B1	*37/*37	CPIC A[13]

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 ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]; Product monograph (actionable)[23]

Drug-Gene Interactions:

Moderate drug-gene interaction: may require a reduced dose

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

2 FDA Product Monograph – Actionable PGx: 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 Level
Pravachol TreatGx ReviewGx	Normal function	SLCO1B1	*37/*37	CPIC A[13]

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.

Propafenone	Phenotype	Genetic Test	Results	Source/Evidence Level
Rythmol TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	DPWG[17]; FDA 1[46]

Drug-Gene Interactions:

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

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

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

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

Propranolol	Phenotype	Genetic Test	Results	Source/Evidence Level
Inderal Innopran TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 3[46]

Drug-Gene Interactions:

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

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

1 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 Level
Vivactil ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 3[46]

Drug-Gene Interactions:

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

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

1 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 Level
Seroquel	Normal metabolizer	CYP3A4	*1A/*1B	DPWG[17]

TreatGx
ReviewGx

Drug-Gene Interactions:

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

1 DPWG: no recommendation for this CYP3A4 phenotype.

Rabeprazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Aciphex Pariet	Poor metabolizer	CYP2C19	*2/*2	FDA 3[46]

TreatGx
ReviewGx

Drug-Gene Interactions:

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

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

Risperidone	Phenotype	Genetic Test	Results	Source/Evidence Level
Perseris Risperdal	Poor metabolizer	CYP2D6	*4/*4	DPWG[17]; FDA 3[46]
	Increased prolactin compared to G/G	ANKK1/DRD2 rs1800497	A/A	PharmGKB 3[47,48]
	May have decreased time until response compared to T/T	DRD2 rs1799978	C/T	PharmGKB 3[47,48]
	Decreased risk of developing metabolic syndrome	HTR2C rs1414334	G/G	PharmGKB 3[47,48]

TreatGx
ReviewGx

Drug-Gene Interactions:

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

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

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.

2 DPWG – 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.
DPWG – 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.

1 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/A 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 Level
Crestor	Normal function	SLCO1B1	*37/*37	CPIC A[13]; FDA 3[46]
	Normal function	ABCG2 rs2231142	G/G	CPIC A[13]

TreatGx ReviewGx

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: Typical myopathy risk and rosuvastatin exposure.

1 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 Asian ancestry should be evaluated prior to initiating rosuvastatin.

Salmeterol	Phenotype	Genetic Test	Results	Source/Evidence Level
Serevent	Increased response compared to A/A	ADRB2 rs1042713	A/G	PharmGKB 2A[47,48]

TreatGx ReviewGx

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.

Sertraline	Phenotype	Genetic Test	Results	Source/Evidence Level
Zoloft	Poor metabolizer	CYP2B6	*6/*6	CPIC B[10]
	Poor metabolizer	CYP2C19	*2/*2	CPIC A[10]

TreatGx ReviewGx

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

3 Greatly reduced metabolism of sertraline to less active compounds when compared with CYP2B6 normal metabolizers. Higher plasma concentrations may increase the probability of side effects.

3 Greatly reduced metabolism of sertraline to less active compounds when compared with CYP2C19 normal metabolizers. Higher plasma concentrations may increase the probability of side effects.

3 Select an alternative antidepressant not primarily metabolized by CYP2C19 or CYP2B6 (per CPIC optional recommendation).

Simvastatin	Phenotype	Genetic Test	Results	Source/Evidence Level
Zocor Flolipid	Normal function	SLCO1B1	*37/*37	CPIC A[13]; FDA 2[46]

TreatGx ReviewGx

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	Normal metabolizer	CYP2C9 (Star Alleles)	*1/*1	FDA 1[46]

ReviewGx

Drug-Gene Interactions:

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

1 CYP2C9 alleles do not indicate changes from recommended dose

Tacrolimus	Phenotype	Genetic Test	Results	Source/Evidence Level
Advagraf Astagraf XL Envarsus XR Prograf Protopic	Poor metabolizer	CYP3A5	*6/*7	CPIC A[9]; FDA 1[46]
	Normal metabolizer	CYP3A4	*1A/*1B	PharmGKB 2A[47,48]

ReviewGx

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.

Tamoxifen	Phenotype	Genetic Test	Results	Source/Evidence Level
Nolvadex Soltamox	Poor metabolizer	CYP2D6 (Activity Score)	*4/*4	CPIC A[21]; FDA 3[46]

ReviewGx

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

3 CPIC – Implication: Lower endoxifen concentrations compared to normal metabolizers; higher risk of breast cancer recurrence, event-free and recurrence-free survival compared to normal metabolizers.

3 CPIC – Strong Recommendation: Recommend alternative hormonal therapy such as an aromatase inhibitor for postmenopausal women or aromatase inhibitor along with ovarian function suppression in premenopausal women given that these approaches are superior to tamoxifen regardless of CYP2D6 genotype (PMID 26211827) and based on knowledge that CYP2D6 poor metabolizers switched from tamoxifen to anastrozole do not have an increased risk of recurrence (PMID 23213055). Note, 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 (PMID 27226358, 21768473).

1 FDA PGx Table Section 3 – Potential Impact on Pharmacokinetic Properties Only: Results in lower systemic active metabolite concentrations. The impact of CYP2D6 intermediate or poor metabolism on efficacy is not well established.

Tamsulosin	Phenotype	Genetic Test	Results	Source/Evidence Level
Flomax	Poor metabolizer	CYP2D6	*4/*4	FDA 3[46]

ReviewGx

Drug-Gene Interactions:

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

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

1 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 Level
Mobiflex	Normal metabolizer	CYP2C9 (Star Alleles)	*1/*1	CPIC A[45]

ReviewGx

Drug-Gene Interactions:

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

1 CYP2C9 alleles do not indicate changes from recommended dose

Tetrabenazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Austedo Nitoman Xenazine ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]

Drug-Gene Interactions:

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

- 2 CYP2D6 poor metabolizer: greatly reduced metabolism of Tetrabenazine to less active compounds
- 2 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

Thioguanine	Phenotype	Genetic Test	Results	Source/Evidence Level
Lanvis ReviewGx	Normal metabolizer	TPMT	*1/*1	CPIC A[39,44]; FDA 1[46]
	Normal metabolizer	NUDT15	*1/*1	CPIC A[39,44]; FDA 1[46]

Drug-Gene Interactions:

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

- 1 CPIC – TPMT Implication: Lower concentrations of thioguanine nucleotides (TGN) metabolites, but note that TGN after thioguanine are 5-10X higher than TGN after mercaptopurine or azathioprine. Normal risk of thiopurine-related leukopenia, neutropenia, myelosuppression.
- 1 CPIC – NUDT15 Implication: Normal risk of thiopurine-related leukopenia, neutropenia, myelosuppression.
- 1 CPIC – Strong Recommendation: Start with normal starting dose (e.g., 40-60 mg/m2/day) and adjust doses of thioguanine and of other myelosuppressive therapy without any special emphasis on thioguanine. Allow 2 weeks to reach steady-state after each dose adjustment.
  Normal starting doses vary by race/ethnicity and treatment regimens.

Thioridazine	Phenotype	Genetic Test	Results	Source/Evidence Level
TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

- 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 Level
Detrol TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	FDA 2[46]

Drug-Gene Interactions:

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

- 2 CYP2D6 poor metabolizer: greatly reduced metabolism of Tolterodine
- 2 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 Level
Conzip Durela Ralivia Ultram Zytram XL	Poor metabolizer	CYP2D6	*4/*4	CPIC A[14]; FDA 1[46]; FDA 2[46]

TreatGx
ReviewGx

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

3 CPIC – Implication: Greatly reduced O-desmethyltramadol (active metabolite) formation leading to diminished analgesia.

3 CPIC – Strong Recommendation: Avoid tramadol use because of possibility of diminished analgesia. If opioid use is warranted, consider a non-codeine opioid.

Trimipramine	Phenotype	Genetic Test	Results	Source/Evidence Level
Surmontil	Poor metabolizer	CYP2D6	*4/*4	CPIC B[24]; FDA 3[46]
	Poor metabolizer	CYP2C19	*2/*2	CPIC B[24]

ReviewGx

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

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

3 CPIC – CYP2C19 Implication: Greatly reduced metabolism of tertiary amines compared to normal metabolizers. Decreased conversion of tertiary amines to secondary amines may affect response or side effects.

3 CPIC – Optional Recommendation: Avoid Trimipramine use. If use is warranted, utilize therapeutic drug monitoring to guide dose adjustment (strongly recommended for a patient with CYP2D6 ultrarapid, intermediate, or poor metabolism in combination with CYP2C19 ultrarapid, intermediate, or poor metabolism).
Recommendations above only apply to higher initial doses of TCAs for treatment of conditions such as depression. Lower dosages are often used for neuropathic pain compared to depressive disorders.
Although there is sparse information on how to adjust initial TCA doses based on combined CYP2D6 and CYP2C19 genetic results when treating neuropathic pain, caution should be used when patients have a combination of poor or ultrarapid phenotypes (e.g., a CYP2D6 poor metabolizer also having CYP2C19 ultrarapid or poor metabolism).
Because of the lower dosage, it is less likely that CYP2C19 or CYP2D6 poor metabolizers will experience adverse effects due to supratherapeutic plasma concentrations. Therefore, CPIC recommends no dose modifications for poor metabolizers when prescribed a lower dose for treatment of neuropathic pain, but these patients should be monitored closely for side effects. If larger doses are warranted, CPIC recommends following the gene-based guidelines presented above.

Valbenazine	Phenotype	Genetic Test	Results	Source/Evidence Level
Ingrezza	Poor metabolizer	CYP2D6	*4/*4	FDA 1[46]

ReviewGx

Drug-Gene Interactions:

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

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

2 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 Level
Effexor XR	Poor metabolizer	CYP2D6	*4/*4	CPIC B[10]; FDA 1[46]

TreatGx
ReviewGx

Drug-Gene Interactions:

Moderate drug-gene interaction: consider alternative medications, may increase adverse events

2 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 Level
Qelbree	Poor metabolizer	CYP2D6	*4/*4	FDA 3[46]

ReviewGx

Drug-Gene Interactions:

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

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

Voriconazole	Phenotype	Genetic Test	Results	Source/Evidence Level
Vfend	Poor metabolizer	CYP2C19	*2/*2	CPIC A[33]; FDA 2[46]

ReviewGx

Drug-Gene Interactions:

Serious drug-gene interaction: avoid/select alternative

3 CPIC – Implication: Higher dose-adjusted trough concentrations of voriconazole and may increase probability of adverse events.

3 ADULTS: CPIC – Moderate Recommendation: Choose an alternative agent that is not dependent on CYP2C19 metabolism as primary therapy in lieu of voriconazole. Such agents include isavuconazole, liposomal amphotericin B, and posaconazole. In the event that voriconazole is considered to be the most appropriate agent, based on clinical advice, for a patient with poor metabolizer genotype, voriconazole should be administered at a preferably lower than standard dosage with careful therapeutic drug monitoring
PEDIATRICS <18yo: CPIC – Moderate Recommendation: Choose an alternative agent that is not dependent on CYP2C19 metabolism as primary therapy in lieu of voriconazole. Such agents include liposomal amphotericin B and posaconazole. In the event that voriconazole is considered to be the most appropriate agent, based on clinical advice, for a patient with poor metabolizer genotype, voriconazole should be administered at a preferably lower than standard dosage with careful therapeutic drug monitoring.
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. Pediatric recommendations based upon data extrapolated from adults.

Vortioxetine	Phenotype	Genetic Test	Results	Source/Evidence Level
Trintellix	Poor metabolizer	CYP2D6	*4/*4	CPIC A[10]; FDA 1[46]

TreatGx
ReviewGx

Drug-Gene Interactions:

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

2 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 Level
Coumadin Jantoven TreatGx ReviewGx	Normal metabolizer	CYP2C9	*1/*1	CPIC A[26]; FDA 1[46]
	Reduced response	VKORC1 rs9923231	G/G	CPIC A[26]; FDA 1[46]
	Typical response	CYP4F2 rs2108622	C/C	CPIC A[26]; FDA 1[46]
	Increased response	CYP2C rs12777823	A/A	CPIC A[26]

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 – Moderate Recommendation for African ancestry: CYP2C cluster rs12777823 A carriers: Decrease dose by 10-25%.

Zuclopenthixol	Phenotype	Genetic Test	Results	Source/Evidence Level
Clopixol TreatGx ReviewGx	Poor metabolizer	CYP2D6	*4/*4	DPWG[17]

Drug-Gene Interactions:

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

2 DPWG – 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.
DPWG – CYP2D6 Recommendation: Use with 50% of the normal dose.

Laboratory Report

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

Gene	rsID	HGVS	HGVS Reference	Result
ABCB1	rs1045642	c.3645G>A	NC_000007.14	G/G
ABCB1	rs1128503	c.1446G>A	NC_000007.14	G/G
ABCB1	rs2032582	c.2887C>A/T	NC_000007.14	C/C
ABCG2	rs2231142	c.421G>T	NC_000004.12	G/G
ADRA2A	rs11195419	c.*216C>A	NC_000010.11	A/C
ADRA2A	rs1800544	c.-1252G>C	NC_000010.11	G/G
ADRA2A	rs553668	c.*427G>A	NC_000010.11	A/G
ADRB2	rs1042713	c.46G>A	NC_000005.10	A/G
ANKK1	rs1800497	c.2137G>A	NC_000011.10	A/A
APOE	rs429358	c.388T>C	NC_000019.10	C/T
APOE	rs7412	c.526C>T	NC_000019.10	C/C
COMT	rs4680	c.472G>A	NC_000022.11	A/G
CYP1A2	rs12720461	c.-10+113C>T	NC_000015.10	C/C
CYP1A2	rs2069514	g.74745879G>A	NC_000015.10	G/A
CYP1A2	rs2069526	c.-10+103T>G	NC_000015.10	T/T
CYP1A2	rs35694136	c.-1635T>-	NC_000015.10	T/-
CYP1A2	rs762551	c.-9-154A>C	NC_000015.10	C/A
CYP2B6	rs28399499	c.983T>C	NC_000019.10	T/T
CYP2B6	rs3211371	c.1459C>T	NC_000019.10	C/C
CYP2B6	rs34223104	c.-82T>C	NC_000019.10	T/T
CYP2B6	rs2279343	c.785A>G	NC_000019.10	G/G
CYP2B6	rs3745274	c.516G>T	NC_000019.10	T/T
CYP2C	rs12777823	g.94645745G>A	NC_000010.11	A/A
CYP2C19	rs12248560	c.-806C>T	NC_000010.11	C/C
CYP2C19	rs12769205	c.332-23A>G	NC_000010.11	G/G
CYP2C19	rs28399504	c.1A>G	NC_000010.11	A/A
CYP2C19	rs41291556	c.358T>C	NC_000010.11	T/T
CYP2C19	rs4244285	c.681G>A	NC_000010.11	A/A
CYP2C19	rs4986893	c.636G>A	NC_000010.11	G/G
CYP2C19	rs72552267	c.395G>A	NC_000010.11	G/G
CYP2C19	rs17884712	c.431G>A	NC_000010.11	G/G
CYP2C19	rs56337013	c.1297C>T	NC_000010.11	C/C
CYP2C19	rs6413438	c.680C>T	NC_000010.11	C/C
CYP2C19	rs72558186	c.819+2T>A	NC_000010.11	T/T
CYP2C8	rs10509681	c.1196T>C	NC_000010.11	T/T
CYP2C8	rs1058930	c.792G>C	NC_000010.11	G/G
CYP2C8	rs11572103	c.805T>A	NC_000010.11	T/T
CYP2C8	rs17110453	c.-370A>C	NC_000010.11	A/A
CYP2C9	rs1057910	c.1075A>C	NC_000010.11	A/A
CYP2C9	rs1799853	c.430C>T	NC_000010.11	C/C
CYP2C9	rs28371685	c.1003C>T	NC_000010.11	C/C
CYP2C9	rs28371686	c.1080C>G	NC_000010.11	C/C
CYP2C9	rs72558187	c.269T>C	NC_000010.11	T/T
CYP2C9	rs9332131	c.818AA>-	NC_000010.11	A/A
CYP2C9	rs56165452	c.1076T>C	NC_000010.11	T/T
CYP2C9	rs7900194	c.449G>A/T	NC_000010.11	G/G
CYP2D6	rs1065852	c.100G>A	NC_000022.11	A/A
CYP2D6	rs1135840	c.1457G>C	NC_000022.11	G/G
CYP2D6	rs16947	c.886G>A	NC_000022.11	G/G
CYP2D6	rs28371706	c.320G>A	NC_000022.11	G/G
CYP2D6	rs28371725	c.985+39C>T	NC_000022.11	C/C

Gene	rsID	HGVS	HGVS Reference	Result
CYP2D6	rs35742686	c.775T>-	NC_000022.11	T/T
CYP2D6	rs3892097	c.506-1C>T	NC_000022.11	T/T
CYP2D6	rs5030655	c.454A>-	NC_000022.11	A/A
CYP2D6	rs5030656	c.841_843CTT>-	NC_000022.11	CTT/CTT
CYP2D6	rs5030867	c.971T>G	NC_000022.11	T/T
CYP2D6	rs59421388	c.1012C>T	NC_000022.11	C/C
CYP2D6	rs5030862	c.124C>T	NC_000022.11	C/C
CYP2D6	rs5030865	c.505C>T/A	NC_000022.11	C/C
CYP2D6	rs769258	c.31C>T	NC_000022.11	C/C
CYP2D6	rs201377835	c.181-1C>G	NC_000022.11	C/C
CYP2D6	rs774671100	c.137->A	NC_000022.11	A/A (-/-)[1]
CYP2D6	rs72549356	c.514_522GGGGCGAAA>-	NC_000022.11	-/-
CYP2D6	rs267608319	c.1319C>T	NC_000022.11	C/C
CYP2D6	rs72549346	c.1088_1089AC>-	NC_000022.11	-/-
CYP2D6	rs1135822	c.358A>T	NC_000022.11	A/A
CYP2D6	rs79292917	c.975C>T	NC_000022.11	C/C
CYP3A4	rs12721629	c.1117G>A	NC_000007.14	G/G
CYP3A4	rs2740574	c.-392T>C	NC_000007.14	C/T
CYP3A4	rs35599367	c.522-191G>A	NC_000007.14	G/G
CYP3A4	rs4986910	c.1334A>G	NC_000007.14	A/A
CYP3A4	rs4987161	c.566A>G	NC_000007.14	A/A
CYP3A4	rs55785340	c.664A>G	NC_000007.14	A/A
CYP3A5	rs10264272	c.624C>T	NC_000007.14	C/T
CYP3A5	rs41303343	c.1035_1036->A	NC_000007.14	A/AA (A/-)[1]
CYP3A5	rs776746	c.219-237C>T	NC_000007.14	T/T
CYP3A5	rs28365083	c.1193G>T	NC_000007.14	G/G
CYP3A5	rs28383468	c.88G>A	NC_000007.14	G/G
CYP3A5	rs28383479	c.1009C>T	NC_000007.14	C/C
CYP3A5	rs55817950	c.82G>A	NC_000007.14	G/G
CYP4F2	rs2108622	c.1297C>T	NC_000019.10	C/C
C11orf65	rs11212617	c.175-5285C>A	NC_000011.10	A/C
DPYD	rs3918290	c.1905+1C>T	NC_000001.11	C/C
DPYD	rs67376798	c.2846T>A	NC_000001.11	T/T
DPYD	rs56038477	c.1236C>T	NC_000001.11	C/C
DPYD	rs55886062	c.1679A>C	NC_000001.11	A/A
DPYD	rs115232898	c.557T>C	NC_000001.11	T/T
DRD2	rs1079598	c.-31-870A>G	NC_000011.10	A/A
DRD2	rs1799732	c.-486_-485G>-	NC_000011.10	G/G
DRD2	rs1799978	c.-32+29T>C	NC_000011.10	C/T
DRD2	rs2734841	c.1139-134C>A	NC_000011.10	C/C
EPHX1	rs1051740	c.337T>C	NC_000001.11	T/T
Factor II	rs1799963	c.*97G>A	NC_000011.10	G/G
Factor V	rs6025	c.1601C>T	NC_000001.11	C/C
GRIK4	rs1954787	c.83-10039C>T	NC_000011.10	T/T
HTR1A	rs6295	c.-1019G>C	NC_000005.10	C/G
HTR2A	rs6311	c.-510C>T	NC_000013.11	C/T
HTR2A	rs7997012	c.614-2211G>A	NC_000013.11	G/G
HTR2C	rs1414334	c.551-3008G>C	NC_000023.11	G/G
HTR2C	rs3813929	c.-759C>T	NC_000023.11	C/C
ITGB3	rs5918	c.176T>C	NC_000017.11	T/T
MTHFR	rs1801131	c.1286A>C	NM_005957.5	T/T
MTHFR	rs1801133	c.665C>T	NM_005957.5	G/G
NUDT15	rs116855232	c.415C>T	NC_000013.11	C/C
NUDT15	rs147390019	c.416G>A	NC_000013.11	G/G
NUDT15	rs186364861	c.52G>A	NC_000013.11	G/G

AttoDiagnostics

PATIENT INFORMATION

NAME: Claire DDI-DGI
DOB: 23/Nov/1993
SEX AT BIRTH: Female

SPECIMEN DETAILS

BARCODE: 4124081155
SAMPLE ID: 4124081155
TYPE: Swab
COLLECTED: 28/Mar/2025
RECEIVED: 30/Mar/2025
QUALITY: Good

ORDERED BY

ATTOD Demo
AMENDED: 27/Nov/2025 (GMT)

Gene	rsID	HGVS	HGVS Reference	Result
OPRM1	rs1799971	c.118A>G	NC_000006.12	A/A
SLCO1B1	rs4149056	c.521T>C	NC_000012.12	T/T
SLCO1B1	rs2306283	c.388G>A	NC_000012.12	G/G
SLC6A2	rs12708954	c.1261-210C>A	NC_000016.10	A/C
SLC6A2	rs3785143	c.274+4226C>T	NC_000016.10	C/C
TPMT	rs1142345	c.719T>C	NC_000006.12	T/T
TPMT	rs1800460	c.460C>T	NC_000006.12	C/C
TPMT	rs1800462	c.238C>G	NC_000006.12	C/C
TPMT	rs1800584	c.626-1C>T	NC_000006.12	C/C
UGT2B15	rs1902023	c.253C>A	NC_000004.12	C/C
VKORC1	rs9923231	c.-1639C>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.

AttoDiagnostics Ltd Scottow Enterprise Park, Lamas Road, Badersfield, Norfolk, NR10 5FB +44 1603 327 053
info@attodiagnostics.com www.attodiagnostics.com

Table of Available References

Drug	Genetic Test	Sources
Abrocitinib	CYP2C19	FDA[36,46]
Aducanumab	ApoE	FDA[8]
Alfentanil	OPRM1 rs1799971	PharmGKB[47,48]
Allopurinol	ABCG2 rs2231142	DPWG[17]
Amitriptyline	CYP2D6	CPIC[24]; FDA[46]
Amitriptyline	CYP2C19	CPIC[24]
Amoxapine	CYP2D6	FDA[46]
Amphetamine	CYP2D6	FDA[46]
Aripiprazole	CYP2D6	FDA[46]; Product monograph (actionable)[42]
Aripiprazole lauroxil	CYP2D6	FDA[46]; Product monograph (actionable)[2]
Atomoxetine	CYP2D6 (Activity Score)	CPIC[11]; FDA[46]
Atorvastatin	SLCO1B1	CPIC[13]; FDA[46]
Atorvastatin	ApoE rs7412	PharmGKB[47,48]
Avatrombopag	CYP2C9	FDA[46]
Avatrombopag	Factor II rs1799963	Product monograph (actionable)[1]
Avatrombopag	Factor V rs6025	Product monograph (actionable)[1]
Azathioprine	TPMT	CPIC[39,44]; FDA[46]
Azathioprine	NUDT15	CPIC[39,44]; FDA[46]
Brexpiprazole	CYP2D6	FDA[46]; Product monograph (actionable)[3]
Brivaracetam	CYP2C19	FDA[46]
Bupropion	ANKK1/DRD2 rs1800497	PharmGKB[47,48]
Capecitabine	DPYD	CPIC[4]; FDA[46]
Carisoprodol	CYP2C19	FDA[46]
Carvedilol	CYP2D6	FDA[46]
Celecoxib	CYP2C9 (Star Alleles)	CPIC[45]; FDA[46]
Cevimeline	CYP2D6	FDA[46]
Citalopram	CYP2C19	CPIC[10]; FDA[46]
Clobazam	CYP2C19	FDA[46]; Product monograph (actionable)[30]
Clomipramine	CYP2D6	CPIC[24]; FDA[46]
Clomipramine	CYP2C19	CPIC[24]
Clopidogrel	CYP2C19	CPIC[28]; FDA[46]
Clozapine	CYP2D6	FDA[46]
Clozapine	HTR2C rs1414334	PharmGKB[47,48]
Codeine	CYP2D6	CPIC[14]; FDA[46]
Cyclosporine	CYP3A5	PharmGKB[47,48]
Darifenacin	CYP2D6	FDA[46]
Desipramine	CYP2D6	CPIC[24]; FDA[46]
Deutetrabenazine	CYP2D6	FDA[46]
Dexlansoprazole	CYP2C19	CPIC[29]; FDA[46]
Dextromethorphan/ Bupropion	CYP2D6	FDA[6]
Dextromethorphan/ Quinidine	CYP2D6	FDA[5]
Diazepam	CYP2C19	FDA[46]
Donepezil	CYP2D6	FDA[46]

Drug	Genetic Test	Sources
Doxepin	CYP2D6	CPIC[24]; FDA[46]
Doxepin	CYP2C19	CPIC[24]; FDA[46]
Dronabinol	CYP2C9	FDA[46]
Efavirenz	CYP2B6	CPIC[15]; FDA[46]
Elagolix	SLCO1B1	FDA[46]
Eliglustat	CYP2D6	DPWG[17]; FDA[46]
Eltrombopag	Factor V rs6025	Product monograph (actionable)[35]
Erdafitinib	CYP2C9 (Star Alleles)	FDA[46]
Escitalopram	CYP2C19	CPIC[10]; FDA[46]
Esomeprazole	CYP2C19	FDA[46]
Fentanyl	OPRM1 rs1799971	PharmGKB[47,48]
Fesoterodine	CYP2D6	FDA[46]
Flecainide	CYP2D6	DPWG[17]
Flibanserin	CYP2C19	FDA[46]
Fluorouracil	DPYD	CPIC[4]; FDA[46]
Fluoxetine	CYP2D6	Product monograph (actionable)[12]
Flurbiprofen	CYP2C9 (Star Alleles)	CPIC[45]; FDA[46]
Fluvastatin	CYP2C9	CPIC[13]
Fluvastatin	SLCO1B1	CPIC[13]
Fluvoxamine	CYP2D6	CPIC[10]; FDA[46]
Fosphenytoin	CYP2C9	CPIC[27]; FDA[46]
Galantamine	CYP2D6	FDA[46]
Gefitinib	CYP2D6	FDA[46]
Haloperidol	CYP2D6	DPWG[17]
Hydrocodone	CYP2D6	CPIC[14]
Ibuprofen	CYP2C9 (Star Alleles)	CPIC[45]; FDA[46]
Iloperidone	CYP2D6	FDA[46]
Imipramine	CYP2D6	CPIC[24]; FDA[46]
Imipramine	CYP2C19	CPIC[24]
Lansoprazole	CYP2C19	CPIC[29]; FDA[46]
Lecanemab	ApoE	FDA[18]
Lofexidine	CYP2D6	FDA[46]
Lovastatin	SLCO1B1	CPIC[13]
Lusutrombopag	Factor II rs1799963	Product monograph (actionable)[43]
Lusutrombopag	Factor V rs6025	Product monograph (actionable)[43]
Mavacamten	CYP2C19	FDA[46]
Meclizine	CYP2D6	FDA[46]
Meloxicam	CYP2C9 (Star Alleles)	CPIC[45]; FDA[46]
Mercaptopurine	TPMT	CPIC[39,44]; FDA[46]
Mercaptopurine	NUDT15	CPIC[39,44]; FDA[46]
Methadone	CYP2B6	CPIC[40]; PharmGKB[47,48]
Methotrexate	MTHFR rs1801133	PharmGKB[47,48]
Methylphenidate	ADRA2A rs1800544	PharmGKB[47,48]
Methylphenidate	COMT rs4680	PharmGKB[47,48]
Metoclopramide	CYP2D6	FDA[46]
Metoprolol	CYP2D6	CPIC[16]
Mirabegron	CYP2D6	FDA[46]
Mirtazapine	CYP2D6	DPWG[17]; PharmGKB 2A[47,48]
Morphine	OPRM1 rs1799971	PharmGKB[47,48]

Drug	Genetic Test	Sources
Nateglinide	CYP2C9	FDA[46]
Nebivolol	CYP2D6	FDA[46]
Nevirapine	CYP2B6 rs28399499	PharmGKB[47,48]
Nevirapine	CYP2B6 rs3745274	PharmGKB[47,48]
Nicotine replacement therapy	ANKK1/DRD2 rs1800497	PharmGKB[47,48]
Nortriptyline	CYP2D6	CPIC[24]; FDA[46]
Olanzapine	DRD2 rs1799978	PharmGKB[47,48]
Oliceridine	CYP2D6	FDA[46]
Omeprazole	CYP2C19	CPIC[29]; FDA[46]
Ondansetron	CYP2D6	CPIC[7]
Oral contraceptives	Factor II rs1799963	PharmGKB[47,48]
Oral contraceptives	Factor V rs6025	PharmGKB[47,48]
Oxycodone	CYP2D6	CPIC[14]; PharmGKB[47,48]
Pantoprazole	CYP2C19	CPIC[29]; FDA[46]
Paroxetine	CYP2D6	CPIC[10]; FDA[46]
Perphenazine	CYP2D6	FDA[46]
Phenytoin	CYP2C9	CPIC[27]; FDA[46]
Pimozide	CYP2D6	FDA[46]
Piroxicam	CYP2C9 (Star Alleles)	CPIC[45]; FDA[46]
Pitavastatin	SLCO1B1	CPIC[13]
Pitolisant	CYP2D6	FDA[46]; Product monograph (actionable)[23]
Pravastatin	SLCO1B1	CPIC[13]
Propafenone	CYP2D6	DPWG[17]; FDA[46]
Propranolol	CYP2D6	FDA[46]
Protriptyline	CYP2D6	FDA[46]
Quetiapine	CYP3A4	DPWG[17]
Rabeprazole	CYP2C19	FDA[46]
Risperidone	CYP2D6	DPWG[17]; FDA[46]
Risperidone	ANKK1/DRD2 rs1800497	PharmGKB[47,48]
Risperidone	DRD2 rs1799978	PharmGKB[47,48]
Risperidone	HTR2C rs1414334	PharmGKB[47,48]
Rosuvastatin	SLCO1B1	CPIC[13]; FDA[46]
Rosuvastatin	ABCG2 rs2231142	CPIC[13]
Salmeterol	ADRB2 rs1042713	PharmGKB[47,48]
Sertraline	CYP2B6	CPIC[10]
Sertraline	CYP2C19	CPIC[10]
Simvastatin	SLCO1B1	CPIC[13]; FDA[46]
Siponimod	CYP2C9 (Star Alleles)	FDA[46]
Tacrolimus	CYP3A5	CPIC[9]; FDA[46]
Tacrolimus	CYP3A4	PharmGKB[47,48]
Tamoxifen	CYP2D6 (Activity Score)	CPIC[21]; FDA[46]
Tamsulosin	CYP2D6	FDA[46]
Tenoxicam	CYP2C9 (Star Alleles)	CPIC[45]
Tetrabenazine	CYP2D6	FDA[46]
Thioguanine	TPMT	CPIC[39,44]; FDA[46]
Thioguanine	NUDT15	CPIC[39,44]; FDA[46]
Thioridazine	CYP2D6	FDA[46]
Tolterodine	CYP2D6	FDA[46]

Drug	Genetic Test	Sources
Tramadol	CYP2D6	CPIC[14]; FDA[46]
Trimipramine	CYP2D6	CPIC[24]; FDA[46]
Trimipramine	CYP2C19	CPIC[24]
Valbenazine	CYP2D6	FDA[46]
Venlafaxine	CYP2D6	CPIC[10]; FDA[46]
Viloxazine	CYP2D6	FDA[46]
Voriconazole	CYP2C19	CPIC[33]; FDA[46]
Vortioxetine	CYP2D6	CPIC[10]; FDA[46]
Warfarin	CYP2C9	CPIC[26]; FDA[46]
Warfarin	VKORC1 rs9923231	CPIC[26]; FDA[46]
Warfarin	CYP4F2 rs2108622	CPIC[26]; FDA[46]
Warfarin	CYP2C rs12777823	CPIC[26]
Zuclopenthixol	CYP2D6	DPWG[17]

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2: Alkermes Inc. Aristada (aripiprazole lauroxil) FDA Product Monograph. Reference ID: NDA207533, 12/2023.

3: Amneal Pharmaceuticals NY LLC. Brexpiprazole FDA Product Monograph. Reference ID: ANDA213562, 03/2024.

4: Amstutz U, et al. Clinical Pharmacogenetics Implementation Consortium (CPIC) Guideline for Dihydropyrimidine Dehydrogenase Genotype and Fluoropyrimidine Dosing: 2017 Update. Clin Pharmacol Ther. 2018;103(2):210-216.

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7: Bell GC, et al. Clinical Pharmacogenetics Implementation Consortium (CPIC) guideline for CYP2D6 genotype and use of ondansetron and tropisetron. Clin Pharmacol Ther. 2017;102(2):213-218.

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9: Birdwell KA, et al. Clinical Pharmacogenetics Implementation Consortium (CPIC) Guidelines for CYP3A5 Genotype and Tacrolimus Dosing. Clin Pharmacol Ther. 2015;98(1):19-24.

10: Bousman CA, et al. Clinical Pharmacogenetics Implementation Consortium (CPIC) Guideline for CYP2D6, CYP2C19, CYP2B6, SLC6A4, and HTR2A Genotypes and Serotonin Reuptake Inhibitor Antidepressants. Clin Pharmacol Ther. 2023;114(1):51-68.

11: Brown JT, et al. Clinical Pharmacogenetics Implementation Consortium Guideline for Cytochrome P450 (CYP)2D6 Genotype and Atomoxetine Therapy. Clin Pharmacol Ther. 2019;106(1):94-102.

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Methods

The results meet stringent quality control metrics for DNA isolation and genotyping. SNPs are processed in an OpenArray platform. Each call has an estimated quality value >95%, based on the autocaller algorithm in the TaqMan® Genotyper software (ThermoFisher Scientific). Copy number calls are accepted when confidence values are >95%.

Limitations

The annotations and interpretations provided in this report are based on scientific literature and do not take into account all potential drug-drug interactions, medical condition or clinical factors that may affect medication response. Gene-drug interactions are ranked according to guidelines, level of evidence and clinical utility. The AttoDiagnostics pharmacogenetics platform, genetic markers, reports and GenXys Health Care System 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.

Other genetic and clinical factors which are not included in this report can also affect medication response.

Other medications are covered by pharmacodynamic and pharmacokinetic genes which are not included in this report but may have an impact on the medications reported.

Additional genes and medications will be added in future updates based on availability of clinically actionable guidelines.

Liability Disclaimer

This test was developed by ThermoFisher Scientific and its performance characteristics has been validated by AttoDiagnostics Ltd and verified on the GenXys Health Care Systems. It has not been cleared or approved by the US Food and Drug Administration or the European Medicines Agency. The report is not a diagnostic test, and TreatGx is not a prescribing system. You should discuss your pharmacogenetics information with a Physician or Healthcare Professional before you act upon the pharmacogenetics information within 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.

DO NOT MAKE ANY CHANGES TO YOUR CURRENT MEDICATION(S) WITHOUT TALKING TO YOUR DOCTOR FIRST.

While genetics is important, other factors also contribute to how you react to medications. The final choice of medication used will be based on your healthcare provider's professional judgement and may be different from what is recommended in this report. This test does not determine your risk of any health problem. It only evaluates select portions of your DNA that help predict how you may react to the medications covered in this report.

Laboratory Director



P Agyirey-Kwakye, Authorised Scientist,
Laboratory Director

27/Nov/2025 (GMT)

Date of Signature

Note: AttoDiagnostics Ltd is a leading precision healthcare genetic testing company. We provide research and diagnostic testing services for brands, medical institutions and healthcare service providers to deliver life-changing personalised healthcare solutions.