Interpretation Guide

Pharmacogenomic (PGx) Report - for your healthcare provider

Biron

The following PGx report is a clinical decision support tool based on individual genetic results. It contributes to a better understanding and prediction of medication response and tolerability. This test does not predict the risk of any health problem. **Since response to medications is multifactorial, clinical judgment supersedes any recommendations provided.**

The report notifies you if the patient carries any genetic variant that can alter the following pharmacological parameters:

- pharmacokinetics: overall **exposure** to a medication depending on metabolic and efflux pump function;
- pharmacodynamics: the potential **efficacy** of a drug and whether the patient is predisposed to certain **atypical effects**.

These results do not change with age, but their interpretation can evolve as new data becomes available. Therefore, the Biron PGx reports are updated periodically. These results can also be useful for other medications, not covered by the report.

How to use pharmacogenomic recommendations

1. Only medications relevant for your patient need to be consulted.
2. Use the **Exposure** column to adjust doses for adequate plasma concentrations.
3. Use the **Efficacy** and **Risk of atypical effect** columns to choose the most compatible medication.

The **Exposure**, **Efficacy** and **Risk of atypical effect** columns are interpreted i ndependently from each other. Medications are ordered by class with the most compatible options listed first within each class.

Exposure

- ↑ A higher dose may be required to achieve adequate plasma concentrations.
- ↓ A lower dose may be required to achieve adequate plasma concentrations.
- ∿ Several metabolic pathways are involved, but their capacities are opposed (e.g., PM and UM). Thus, a calculation of dose adjustments is not possible based on current data and closer monitoring is recommended.
- ✗ Drug not recommended by peer-reviewed guidelines due to a risk of toxicity or lack of efficacy.

Efficacy

When choosing between multiple clinically appropriate medications, you may give preference to a medication in which a lower number of variants have been identified (e.g., 2/2 is better than 4/6), in terms of their association with an increased likelihood of a poorer response or an atypical effect.

- ! Signifies the presence of a high-impact gene variant, which increases the probability of a poorer response.
- ✓ Signifies that all of the tested variants predict an increased likelihood of a better response, compared to non-carriers. This medication may be a good option.

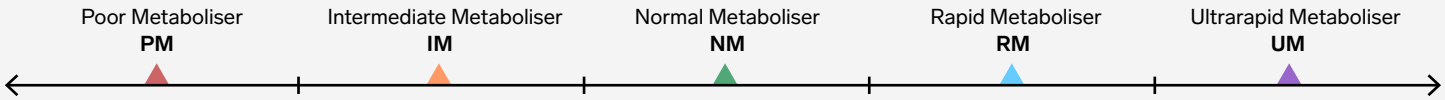
Risk of atypical effect

- ! Signifies the presence of variants associated with an increased risk of particular side effects, compared to non-carriers.
- ✗ Medication not recommended by peer-reviewed guidelines due to a risk of severe side effects,

The notification Normal efficacy* or Normal risk* signifies that there is currently no available data allowing for a genetically-based prediction of medication effect.

Nomenclature for enzyme phenotypes

(e.g., cytochrome P450s or CYP)



NM is generally used to establish standard doses. This dose may be too high for **PM/IM** or too low for **RM/UM**, warranting a dose adjustments or the consideration of an alternative agent. For a pro-drug (e.g., clopidogrel, tramadol), phenotype variability will have the opposite effect.

Inducible Metaboliser **(Ind)** - Specific for CYP1A2, which can have increased function in the presence of an inducer, such as tobacco smoke, comparable to **RM/UM**.

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biron.com/en/genetics/pharmacogenomics

PHARMACOGENOMIC REPORT

Biron

Cardiology

To download the latest version, go here: secur.biron.com/login.

YOUR RESULTS ARE CONFIDENTIAL. As per the Genetic Non-Discrimination Act (S-201), no person, company or institution, including insurers and employers, can force you to share this report.

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 health care provider's professional judgement and may be different than 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. For more information, visit biron.com/pgxtest.

ADMINISTRATIVE DATA

Patient Name
Test-Firstname Test-Lastname

Sex assigned at birth: Female
Date of birth: 1999-01-01
Phone Number: (418) 999-9999
Email: test-sample.BIO2409071186@biron.local

Clinical Support
Email: genetique@biron.com

Ordering Clinician
Meredith Grey

Patient Address
1212 some street
Ste-foy, Québec
G2J 4M5

Phone: 1-855-943-6379

Sample ID: BIO2409071186
Sample Type: test

Date ordered: 2022-08-21
Date of sample reception: 2025-10-20
Date of report: 2025-10-20

Fax: (514) 317-2241

ATYPICAL PHENOTYPES

CYP1A2 IND, CYP2B6 PM, CYP2C19 IM, CYP2D6 IM, DPYD IM, POR PA, SLCO1B1 Reduced function, UGT1A1 IM, UGT2B7 variable, UGT2B15 PM.

NM: Normal Metaboliser, IM: Intermediate Metaboliser, PM: Poor Metaboliser, RM: Rapid Metaboliser, UM: Ultrarapid Metaboliser, Ind: Inducible Metaboliser, NA: Normal Activity, IA: Intermediate Activity, PA: Poor Activity.

CAUTIONARY INFORMATION - MEDICATIONS TO AVOID OR USE WITH CAUTION

Medication	Identified risk	Recommendation
Clopidogrel Plavix®	Reduced clopidogrel active metabolite formation; increased on-treatment platelet reactivity; increased risk for adverse cardiac and cerebrovascular events (CYP2C19 IM).	Cardiovascular indications: avoid standard dose (75mg/day) if possible; use prasugrel or ticagrelor at standard dose if no contraindication. Neurovascular: consider alternative P2Y12 inhibitor at standard dose if clinically indicated and no contraindication.[1]
Tamoxifen	Lower endoxifen concentrations compared to normal metabolizers; higher risk of breast cancer recurrence, reduced probability of event-free and recurrence-free survival (CYP2D6 IM).	Consider hormonal therapy such as an aromatase inhibitor for post-menopausal women or aromatase inhibitor along with ovarian function suppression in premenopausal women. If aromatase inhibitor use is contraindicated, consider using a higher dose of tamoxifen (40mg/day). Avoid CYP2D6 inhibitors.[2]
Capecitabine, 5-fluorouracil	Decreased DPD activity (DPD activity at 30% to 70% that of the normal population) and increased risk for severe or even fatal drug toxicity when treated with fluoropyrimidines.	Reduce starting dose by 25%-50% followed by titration of dose based on toxicity or therapeutic drug monitoring, if available. Increase the dose in patients experiencing no or clinically tolerable toxicity in the first two cycles to maintain efficacy; decrease the dose in patients who do not tolerate the starting dose to minimize toxicities (DPYD IM).[3]

PGx RECOMMENDATIONS - CARDIOLOGY

Dose increase may be required.

Dose reduction may be required.

Exposure is difficult to predict, insufficient data to calculate dose adjustments.

Increased probability of a better response.

Greater potential for a poorer response or atypical effect.

Medication not recommended by peer-reviewed guidelines.

Normal exposure*, Normal efficacy* or Normal risk*: Based on currently available genetic data, the efficacy or risk of an atypical effect is likely similar to that of most other individuals; further research is needed to better understand genetic influence.

Medications	Genetic Associations Identified: Exposure	Efficacy	Risk of atypical effect
Angiotensin-Converting Enzyme Inhibitors (ACEI)			
Benazepril (Lotensin®)	Initiate with recommended starting dose, normal exposure (CES1 NM).	Normal efficacy*	Normal risk*
Captopril (Capoten®)	Normal exposure*	Normal efficacy*	Normal risk*
Cilazapril (Inhibace®)	Initiate with recommended starting dose, normal exposure (CES1 NM).	Normal efficacy*	Normal risk*
Enalapril (Vasotec®)	Initiate with recommended starting dose (CES1 NM, SLCO1B1 reduced function).	Normal efficacy*	Normal risk*
Fosinopril (Monopril®)	Initiate with recommended starting dose, normal exposure (CES1 NM).	Normal efficacy*	Normal risk*
Lisinopril (Prinivil/Zestril®)	Normal exposure*	Normal efficacy*	Normal risk*
Perindopril (Coversyl®)	Initiate with recommended starting dose, normal exposure (CES1 NM).	Normal efficacy*	Normal risk*
Quinapril (Accupril®)	Initiate with recommended starting dose, normal exposure (CES1 NM).	Normal efficacy*	Normal risk*
Ramipril (Altace®)	Initiate with recommended starting dose, normal exposure (CES1 NM).	Normal efficacy*	Normal risk*
Trandolapril (Mavik®)	Initiate with recommended starting dose, normal exposure (CES1 NM).	Normal efficacy*	Normal risk*
Antiarrhythmics			
Amiodarone (Pacerone®/ Cordarone®)	Initiate therapy with recommended starting dose (CYP3A4 NM).	Normal efficacy*	Normal risk*
Digoxin (Lanoxin®)	Initiate with recommended dose; a low dose may be adequate (ABCB1).	Normal efficacy*	Normal risk*
Disopyramide (Rythmodan®)	Initiate therapy with recommended starting dose (CYP3A4 NM).	Normal efficacy*	Normal risk*
Propafenone (Rythmol®)	Initiate with recommended starting dose but, as a precaution, monitor the plasma concentration and record an ECG due to increased risk of side effects. Alternatively, select another agent that is not metabolized by CYP2D6 (or to a lesser extent), including sotalol, disopyramide, quinidine and amiodarone (CYP2D6 IM).[4]	Normal efficacy*	Normal risk*
Flecainide (Tambocor®)	Indications other than diagnosis of Brugada syndrome: reduce the dose to 75% of the standard dose and record an ECG and monitor the plasma	Normal efficacy*	Normal risk*

Genetic Associations Identified			
Medications	Exposure	Efficacy	Risk of atypical effect
	concentration (CYP2D6 IM). Provocation test for diagnosis of Brugada syndrome: No action required.[4]		
Anticoagulants			
Acenocoumarol-Nicoumalone (Sintrom®)	Initiate with recommended dose (VKORC1 -1639 AG).[4]	Normal efficacy*	Normal risk*
Apixaban (Eliquis®)	Initiate with recommended starting dose, normal exposure (CYP3A4 NM, ABCB1).	Normal efficacy*	Normal risk*
Edoxaban (Lixiana®/Savaysa®)	Initiate with recommended starting dose (SLCO1B1 IA).	Normal efficacy*	Normal risk*
Dabigatran (Pradaxa®)	↓ Initiate with recommended starting dose but monitor more closely for potential bleeding risks (CES1 NM, UGT2B15 PM).	Normal efficacy*	Normal risk*
Rivaroxaban (Xarelto®)	! Initiate with recommended starting dose but monitor more closely for potential bleeding risks (ABCB1).	Normal efficacy*	Normal risk*
Warfarin (Coumadin®)	! A dose adjustment may be required; go to warfarindosing.org to calculate the average recommended dose using a validated pharmacogenetic algorithm (CYP2C9 NM, VKORC1 -1639 GA, CYP4F2 CC, rs12777823 GG).[5]	Normal efficacy*	Normal risk*
Antiplatelets			
Prasugrel (Effient®)	Normal exposure*	Normal efficacy*	Normal risk*
Ticagrelor (Brilinta®)	Normal exposure*	Normal efficacy*	Normal risk*
Clopidogrel (Plavix®)	✗ Cardiovascular indications: if possible, avoid standard dose clopidogrel (75mg/day) due to reduced active metabolite formation; use prasugrel or ticagrelor at standard dose if no contraindication. Neurovascular indications: consider alternative P2Y12 inhibitor at standard dose if clinically indicated and no contraindication. (CYP2C19 IM)[1]	Normal efficacy*	Normal risk*
Angiotensin II Receptor Blockers (ARBs)			
Azilsartan (Edarbi®)	Initiate with recommended starting dose, normal exposure (CYP2C9 NM).	Normal efficacy*	Normal risk*
Irbesartan (Avapro®)	Initiate with recommended starting dose, normal exposure (CYP2C9 NM).	Normal efficacy*	Normal risk*
Losartan (Cozaar®)	Initiate with recommended starting dose, normal exposure (CYP2C9 NM).	Normal efficacy*	Normal risk*
Olmesartan (Olmetec®/Benicar®)	Normal exposure*	Normal efficacy*	Normal risk*
Telmisartan (Micardis®)	Normal exposure*	Normal efficacy*	Normal risk*
Valsartan (Diovan®)	Initiate with recommended starting dose, normal exposure (ABCB1).	Normal efficacy*	Normal risk*
Beta-blockers			
Acebutolol (Sectral®/Monitan®)	Initiate with recommended dose (CYP2C19 IM).	Normal efficacy*	Normal risk*

Genetic Associations Identified

Medications	Exposure	Efficacy	Risk of atypical effect
Atenolol (Tenormin®)	Normal exposure; eliminated largely unchanged by the kidneys.	Normal efficacy*	Normal risk*
Bisoprolol (Monocor®/Zebeta®)	Initiate with recommended starting dose (CYP2D6 IM, CYP3A4 NM, CYP3A5 PM).	Normal efficacy*	Normal risk*
Carvedilol (Coreg®)	Initiate with recommended starting dose but monitor bradycardia more closely; a low dose may be adequate (CYP2D6 IM, CYP2C9 NM, UGT1A1 IM).	Normal efficacy*	Normal risk*
Labetalol (Trandate®)	CYP2C19 IM - Initiate with recommended starting dose but monitor bradycardia more closely; a low dose may be adequate.	Normal efficacy*	Normal risk*
Metoprolol (Lopresor/Betaloc®/ Toprol XL®)	Initiate with recommended starting dose; a lower target dose may be adequate (CYP2D6 IM).	Normal efficacy*	Normal risk*
Nadolol (Corgard®)	Initiate with recommended starting dose but monitor bradycardia more closely; a low dose may be adequate (ABCB1).	Normal efficacy*	Normal risk*
Sotalol (Sotacor®/Betapace®)	Normal exposure; eliminated largely unchanged by the kidneys.	Normal efficacy*	Normal risk*
Propranolol (Inderal®)	Initiate therapy with recommended starting dose but monitor response and tolerance more closely; insufficient data to calculate dose adjustments (CYP1A2 Ind, CYP2D6 IM).	Normal efficacy*	Normal risk*
Calcium channel blockers (CCBs)			
Amlodipine (Norvasc®)	Initiate with recommended starting dose, normal exposure (CYP3A4 NM, CYP3A5 PM).	Normal efficacy*	Normal risk*
Diltiazem (Cardizem®/Tiazac®)	Initiate with recommended starting dose, normal exposure (CYP3A4 NM).	Normal efficacy*	Normal risk*
Felodipine (Plendil®)	Initiate with recommended starting dose, normal exposure (CYP3A4 NM).	Normal efficacy*	Normal risk*
Nifedipine (Adalat/Procardia®)	Initiate with recommended starting dose, normal exposure (CYP3A4 NM, CYP3A5 PM).	Normal efficacy*	Normal risk*
Verapamil (Isoptin®/Verelan®/ Calan®)	Initiate with recommended starting dose, normal exposure (CYP3A4 NM).	Normal efficacy*	Normal risk*
Diuretics			
Acetazolamide (Diamox®)	Normal exposure; eliminated largely unchanged by the kidneys.	Normal efficacy*	Normal risk*
Amiloride (Midamor®)	Normal exposure; eliminated largely unchanged by the kidneys.	Normal efficacy*	Normal risk*
Chlorthalidone (Hygroton®)	Normal exposure; eliminated largely unchanged by the kidneys.	Normal efficacy*	Normal risk*
Eplerenone (Inspra®)	Initiate with recommended starting dose, normal exposure (CYP3A4 NM, CYP3A5 PM).	Normal efficacy*	Normal risk*
Furosemide (Lasix®)	Normal exposure*	Normal efficacy*	Normal risk*
Hydrochlorothiazide (HCTZ) (Hydrodiuril®/ Microzide®)	Normal exposure; eliminated largely unchanged by the kidneys.	Normal efficacy*	Normal risk*
Indapamide (lozide®/Lozol®)	Initiate with recommended starting dose, normal exposure (CYP3A4 NM).	Normal efficacy*	Normal risk*

Genetic Associations Identified			
Medications	Exposure	Efficacy	Risk of atypical effect
Spironolactone (Aldactone®)	Normal exposure; eliminated largely unchanged by the kidneys.	Normal efficacy*	Normal risk*
Triamterene (Dyrenium®)	↑ Initiate with recommended starting dose but monitor efficacy more closely, especially with CYP1A2 inducers, such as smoke (CYP1A2 Ind).	Normal efficacy*	Normal risk*
Lipid-lowering agent			
Fenofibrate (Lipidil®/Tricor®)	Normal exposure*	Normal efficacy*	Normal risk*
Selective cholesterol-absorption inhibitor			
Ezetimibe (Ezetrol®/Zetia®)	Normal exposure*	Normal efficacy*	Normal risk*
Statins			
Fluvastatin (Lescol®)	Prescribe desired starting dose and adjust doses based on disease-specific guidelines. Be aware of possible increased risk for myopathy especially for doses >40 mg per day (SLCO1B1 decreased function, CYP2C9 NM).[6]	Normal efficacy*	Normal risk*
Pravastatin (Pravachol®)	Prescribe desired starting dose and adjust doses based on disease-specific guidelines. Be aware of possible increased risk for myopathy with pravastatin especially with doses >40 mg per day (SLCO1B1 reduced function).[6]	Normal efficacy*	Normal risk*
Rosuvastatin (Crestor®)	Prescribe desired starting dose and adjust doses based on disease-specific and population-specific guidelines. Be aware of possible increased risk for myopathy especially for doses >20 mg (SLCO1B1 decreased function, ABCG2 normal function).[7]	Normal efficacy*	Normal risk*
Atorvastatin (Lipitor®/Caduet®)	↓ Prescribe ≤40 mg as a starting dose and adjust doses based on disease-specific guidelines. Be aware of possible increased risk for myopathy especially for 40-mg dose. If dose >40 mg needed for desired efficacy, consider combination therapy (i.e., atorvastatin plus nonstatin guideline-directed medical therapy) (SLCO1B1 reduced function).[6]	Normal efficacy*	Normal risk*
Pitavastatin (Livalo®)	↓ Prescribe ≤2 mg as a starting dose and adjust doses based on disease-specific guidelines. Be aware of possible increased risk for myopathy especially for doses >1 mg. If dose >2 mg needed for desired efficacy, consider an alternative statin or combination therapy (i.e., pitavastatin plus nonstatin guideline-directed medical therapy) (SLCO1B1 reduced function).[6]	Normal efficacy*	Normal risk*
Lovastatin (Mevacor®)	✖ Prescribe an alternative statin depending on the desired potency. If lovastatin therapy is warranted, limit dose to ≤20 mg/day (SLCO1B1 reduced function).[6]	Normal efficacy*	Normal risk*
Simvastatin (Zocor®)	✖ Prescribe an alternative statin depending on the desired potency. If simvastatin	Normal efficacy*	Normal risk*

Genetic Associations Identified

Medications	Exposure	Efficacy	Risk of atypical effect
	therapy is warranted, limit dose to <20 mg/day (SLCO1B1 reduced function).[6]		

PGx RECOMMENDATIONS - COMPLEMENTARY TREATMENTS

Dose increase may be required.

Dose reduction may be required.

Exposure is difficult to predict, insufficient data to calculate dose adjustments.

Increased probability of a better response.

Greater potential for a poorer response or atypical effect.

Medication not recommended by peer-reviewed guidelines.

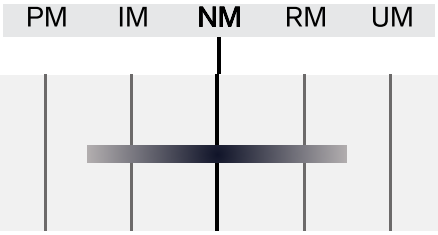
Normal exposure*, Normal efficacy* or Normal risk*: Based on currently available genetic data, the efficacy or risk of an atypical effect is likely similar to that of most other individuals; further research is needed to better understand genetic influence.

Medications	Genetic Associations Identified: Exposure	Efficacy	Risk of atypical effect
Antiemetics			
Dimenhydrinate (Gravol®)	Normal exposure*	Normal efficacy*	Normal risk*
Granisetron (Kytril®)	Normal exposure (CYP3A4 NM, CYP3A5 PM).	Normal efficacy*	Normal risk*
Ondansetron (Zofran®)	Initiate therapy with recommended starting dose; a low dose may be adequate (CYP2D6 IM).	Normal efficacy*	Normal risk*
Palonosetron (Aloxi®)	Initiate with recommended dose; a low dose may be adequate (CYP2D6 IM).	Normal efficacy*	Normal risk*
Proton pump inhibitors (PPI)			
Esomeprazole (Nexium®)	Normal exposure*	Normal efficacy*	Normal risk*
Dexlansoprazole (Dexilant®)	Initiate therapy with standard dose but for chronic therapy (> 12 weeks) and efficacy achieved, consider reducing the daily dose by 50% and monitor for continued efficacy (CYP2C19 IM).[8]	Normal efficacy*	Normal risk*
Lansoprazole (Prevacid®)	Initiate therapy with standard dose but for chronic therapy (> 12 weeks) and efficacy achieved, consider reducing the daily dose by 50% and monitor for continued efficacy (CYP2C19 IM).[8]	Normal efficacy*	Normal risk*
Omeprazole (Losec®)	Initiate therapy with standard dose but for chronic therapy (> 12 weeks) and efficacy achieved, consider reducing the daily dose by 50% and monitor for continued efficacy (CYP2C19 IM).[8]	Normal efficacy*	Normal risk*
Pantoprazole (Pantoloc®)	Initiate therapy with standard dose but for chronic therapy (> 12 weeks) and efficacy achieved, consider reducing the daily dose by 50% and monitor for continued efficacy (CYP2C19 IM).[8]	Normal efficacy*	Normal risk*

PGx ASSOCIATIONS - EXPOSURE

Enzymes	Phenotype (PM IM NM RM UM)	Genotype	Details
CYTOCHROME P450 (CYP450)			
CYP2B6		*6/*6	**Poor metabolizer (PM)** Greatly reduced or absent CYP2B6 activity compared to normal metabolizers.
CYP3A5		*3/*3	**Poor metabolizer (PM)** Low CYP3A5 activity is the most common phenotype and has been used to establish standard doses.
CYP2C19		*1/*2	**Intermediate metabolizer (IM)** Reduced CYP2C19 activity compared to normal metabolizers.
CYP2D6		*1/*4	**Intermediate metabolizer (IM)** Reduced CYP2D6 activity compared to normal metabolizers.
CYP2A6		*1/*1	**Normal metabolizer (NM)** Normal CYP2A6 activity.
CYP2C9		*1/*1	**Normal metabolizer (NM)** Normal CYP2C9 activity.
CYP4F2		*1/*1	**Normal metabolizer (NM)** Normal CYP4F2 activity.
CYP3A4		*1/*1	**Normal metabolizer (NM)** Normal CYP3A4 activity.
CYP1A2		*1A/*1F	**Inducible metabolizer (Ind)** Increased CYP1A2 activity compared to normal metabolizers if the patient is exposed to CYP1A2 inducers, such as smoke.

Enzymes	Phenotype (PM IM **NM** RM UM)	Genotype	Details
OTHER METABOLIC ENZYMES AND FACTORS			
POR		A/A	**Poor activity (PA)** Greatly reduced cytochrome P450 oxicoreductase (POR) activity compared to normal phenotypes (POR participates in CYP2B6 activation).
SLCO1B1		*1/*5	**Intermediate function** Reduced solute carrier organic anion transporter family member 1B1 (SLCO1B1) activity compared to a normal function.
DPYD		REF/c.1129-5923C>G	**Intermediate metabolizer (IM)** Reduced dihydropyrimidine dehydrogenase (DPYD) activity compared to normal metabolizers.
CES1		C/C	**Normal metabolizer (NM)** Normal carboxylesterase-1 (CES1) activity.
TPMT		*1/*1	**Normal metabolizer (NM)** Normal thiopurine S-methyltransferase (TPMT) activity.
NUDT15		*1/*1	**Normal metabolizer (NM)** Normal nudix hydrolase 15 (NUDT15) activity.
UDP-GLUCURONOSYLTRANSFERASES (UGT)			
UGT2B15		*2/*2	**Poor metabolizer (PM)** Greatly reduced or absent UGT2B15 activity compared to normal metabolizers.
UGT1A1		*1/*28	**Intermediate metabolizer (IM)** Reduced UGT1A1 activity compared to normal metabolizers.
UGT1A4		*1/*1	**Normal metabolizer (NM)** Normal UGT1A4 activity.

Enzymes	Phenotype	Genotype	Details
UGT2B7		*1/*2	**Variable metabolizer activity** The function of the *2 allele of UGT2B7 is substrate-dependent. Thus, compared to normal metabolizers, UGT2B7 activity can be increased or reduced depending on the medication substrate.

ANALYTICAL RESULTS

The following analytical results were used to generate the pharmacogenomic interpretations found in this report. Technical limitations inherent with the methods used to produce these results may hinder the attribution of a definitive phenotype (see "TEST METHODOLOGY AND LIMITATIONS").

Genes	Variant Details (GRCH38.p12)		Result
ABCB1	rs1045642	chr7:87509329	A|G
	rs2032582	chr7:87531302	A|C
	rs2032583	chr7:87531245	T|T
ABCG2	rs2231142	chr4:88131171	G|G
ADRA2A	rs1800544	chr10:111076745	C|G
ANKK1	rs1800497	chr11:113400106	A|G
BDNF	rs6265	chr11:27658369	C|C
CACNG2	rs2283967	chr22:36567486	C|T
CES1	rs71647871	chr16:55823658	C|C
CNR1	rs806380	chr6:88154934	G|G
COMT	rs4680	chr22:19963748	A|A
CYP1A2	rs762551	chr15:74749576	C|A
	rs2069514	chr15:74745879	G|G
CYP2A6	rs1801272	chr19:40848628	A|A
	rs28399433	chr19:40850474	A|A
CYP2B6	rs2279343	chr19:41009358	G|G
	rs3745274	chr19:41006936	T|T
	rs28399499	chr19:41012316	T|T
CYP2C cluster	rs12777823	chr10:94645745	G|G
CYP2C9	rs1057910	chr10:94981296	A|A
	rs1799853	chr10:94942290	C|C
	rs7900194	chr10:94942309	G|G
	rs9332131	chr10:94949282-94949283	A|A
	rs9332239	chr10:94989020	C|C
	rs28371685	chr10:94981224	C|C
	rs28371686	chr10:94981301	C|C
	rs72558187	chr10:94941958	T|T
	rs72558190	chr10:94947782	C|C
CYP2C19	rs4244285	chr10:94781859	G|G
	rs4986893	chr10:94780653	G|G
	rs6413438	chr10:94781858	C|C
	rs12248560	chr10:94761900	C|C
	rs12769205	chr10:94775367	A|A
	rs17884712	chr10:94775489	G|G
	rs28399504	chr10:94762706	A|A
	rs41291556	chr10:94775416	T|T
	rs56337013	chr10:94852738	C|C
	rs72552267	chr10:94775453	G|G
	rs72558186	chr10:94781999	T|T
CYP2D6	rs16947	chr22:42127941	G|G
	rs1065852	chr22:42130692	G|G
	rs1135840	chr22:42126611	C|C
	rs3892097	chr22:42128945	C|C
	rs5030655	chr22:42129084	A|A
	rs5030656	chr22:42128174-42128178	A|A
	rs5030862	chr22:42130668	C|C
	rs5030865	chr22:42129033	C|C
	rs5030867	chr22:42127856	T|T
	rs28371725	chr22:42127803	C|C
	rs28371706	chr22:42129770	G|G
	rs35742686	chr22:42128242	T|T
	rs59421388	chr22:42127608	C|C
	rs774671100	chr22:42130555-42130755	G|G
	rs201377835	chr22:42129910	C|C
	Gene Deletion	n/a	Not Detected
	Gene Duplication	n/a	Not Detected
CYP3A4	rs4986907	chr7:99769804	C|C
	rs35599367	chr7:99768693	G|G
	rs55785340	chr7:99768360	A|A
	rs67666821	chr7:99758184-99758188	D|D
	rs72552799	chr7:99770165	C|C

Genes	Variant Details (GRCH38.p12)		Result
CYP3A5	rs776746	chr7:99672916	C|C
	rs10264272	chr7:99665212	C|C
	rs41303343	chr7:99652771	D|D
CYP4F2	rs2108622	chr19:15879621	C|C
DPYD	rs75017182	chr1:97579893	G|C
	rs55886062	chr1:97515787	A|A
	rs3918290	chr1:97450058	C|C
	rs112766203	chr1:97305279	G|G
	rs67376798	chr1:97082391	T|T
	rs115232898	chr1:97699474	T|T
	rs146356975	chr1:97595149	T|T
DRD2	rs6275	chr11:113412755	A|G
DRD3	rs963468	chr3:114144040	G|G
FAAH	rs324420	chr1:46405089	C|C
FKBP5	rs4713916	chr6:35702206	A|G
GNB3	rs5443	chr12:6845711	C|C
GRIK1	rs2832407	chr21:29595188	C|C
GRIK4	rs1954787	chr11:120792654	C|C
*HLA-A*31:01*	rs1061235	chr6:29945521	A|A
*HLA-B*15:02*	rs144012689	chr6:31355003	T|T
HTR2A	rs6311	chr13:46897343	C|T
	rs6313	chr13:46895805	A|G
	rs2770296	chr13:46866425	C|T
HTR2C	rs3813929	chrX:114584047	C|C
HTR7	rs7905446	chr10:90859404	G|T
INSIG2	rs17047764	chr2:118111006	C|G
long non-coding (lnc) RNA	rs74795342	chr21:18954018	G|G
MC4R	rs489693	chr18:60215554	A|C
	rs17782313	chr18:60183864	T|T
MTHFR	rs1801131	chr1:11794419	T|T
	rs1801133	chr1:11796321	G|G
NUDT15	rs116855232	chr13:48045719	C|C
OPRM1	rs1799971	chr6:154039662	A|A
POR	rs2868177	chr7:75960585	A|A
SLC6A2	rs5569	chr16:55697923	A|G
	rs2242446	chr16:55656513	C|T
	rs28386840	chr16:55652906	A|T
SLC6A4	5-HTTLPR	chr17:30190154-30240133	S|L
SLC6A5	rs2298826	chr11:20638211	A|G
SLCO1B1	rs4149056	chr12:21178615	T|T
TH	rs2070762	chr11:2165105	A|G
TPH2	rs1487278	chr12:72007071	T|T
TPMT	rs1800462	chr6:18143724	C|C
	rs1800460	chr6:18138997	C|C
	rs1142345	chr6:18130687	T|T
UGT1A1	rs4148323	chr2:233760498	G|G
	rs34815109	chr2:233760234-233760248	6|7
UGT1A4	rs2011425	chr2:233718962	T|T
UGT2B7	rs7439366	chr4:69098620	T|C
UGT2B15	rs1902023	chr4:68670366	A|A
VKORC1	rs9923231	chr16:31096368	C|T

TEST METHODOLOGY AND LIMITATIONS

The Biron pharmacogenomic test for psychiatry and pain management is a MALDI-TOF-based single nucleotide primer extension genotyping test; laboratory developed and validated test (LDT), not approved by Health Canada. Nucleic acid amplification techniques may be subject to general interference by factors such as reaction inhibitors and low quality or quantity of extracted DNA. Factors influencing the amount and quality of extracted DNA include but are not limited to patient oral hygiene, collection technique and presence of dietary or microbial source of nucleic acids and nuclease. When present, these interferents typically yield no result rather than an inaccurate one. Risk of suboptimal DNA quantity or quality is significantly reduced by automated DNA extraction which uses chemistry without PCR inhibitors (magnetic beads) and systematic dilution, quantitation and normalization of DNA before nucleic acid amplification. Very infrequent variants or polymorphisms occurring in primer-binding regions may also affect testing and could produce an erroneous result or assay failure. The test does not detect all known and unknown variations in the genes tested, nor does absence of a detectable variant (typically reported as *1 for metabolic enzymes) rule out the presence of other, non-detected variants. The test detects CYP2D6 deletion and duplication but cannot differentiate duplication in the presence of deletion. CYP2D6 deletion and duplication assays can translate into equivocal phenotype results where a range of enzyme activity level must be reported. Test results and clinical interpretation may be inaccurate for individuals who have undergone or are receiving non-autologous blood transfusions, tissue, and/or organ transplant therapies.

DISCLAIMER

Biron Health Group developed this pharmacogenomic report. This test does not diagnose any disorder, condition or disease. The interpretations and recommendations provided in this report are intended as a clinical support tool (DST) to be used solely by a healthcare professional. Treatment decisions for the patient remain the sole responsibility of the treating healthcare provider. The interpretations of the results provided by this report were determined by Biron's data curation protocol, which were established as per the current available scientific evidence available at the time this report version was created. As more evidence becomes available in the future, these interpretations may change. Some variants tested may not be used to provide report interpretations due to a lack of clear gene-drug association as determined by Biron's data curation protocol. The presence of a notification within the "Exposure", "Efficacy" or "Adverse Drug Reactions" categories for a given drug indicates that an associated genetic variant was detected. The lack of a notification within these categories for a given drug does not eliminate the requirement for dose adjustments for optimal dosage, does not guarantee effective drug therapy and does not eliminate the risks of adverse drug reactions. Commercial names are indicated as examples and do not consist an exhaustive list.

REFERENCES

For the full list of references, contact pgxinfo@biron.com

Reference(s) cited in this report:

1. *Lee CR, et al. Clinical Pharmacogenetics Implementation Consortium Guideline for CYP2C19 Genotype and Clopidogrel Therapy: 2022 Update. Clin Pharmacol Ther (2022).*
2. *Goetz MP, et al. Clinical Pharmacogenetics Implementation Consortium (CPIC) Guideline for CYP2D6 and Tamoxifen Therapy. Clin Pharmacol Ther (2018).*
3. *Amstutz U, et al. Clinical Pharmacogenetics Implementation Consortium (CPIC) Guideline for Dihydropyrimidine Dehydrogenase Genotype and Fluoropyrimidine Dosing: 2017 Update. Clin Pharmacol Ther (2018).*
4. *Dutch Pharmacogenetics Working Group (DPWG) 2018 update (https://api.pharmgkb.org/v1/download/file/attachment/DPWG_November_2018.pdf)*
5. *Johnson JA, et al. Clinical Pharmacogenetics Implementation Consortium (CPIC) Guideline for Pharmacogenetics-Guided Warfarin Dosing: 2017 Update. Clin Pharmacol Ther (2017).*
6. *Cooper-DeHoff RM, et al. The Clinical Pharmacogenetics Implementation Consortium Guideline for SLCO1B1, ABCG2, and CYP2C9 genotypes and Statin-Associated Musculoskeletal Symptoms. Clin Pharmacol Ther (2022).*
7. *Hicks JK, et al. Clinical pharmacogenetics implementation consortium guideline (CPIC) for CYP2D6 and CYP2C19 genotypes and dosing of tricyclic antidepressants: 2016 update. Clin Pharmacol Ther (2017).*
8. *Lima JJ, et al. Clinical Pharmacogenetics Implementation Consortium (CPIC) Guideline for CYP2C19 and Proton Pump Inhibitor Dosing. Clin Pharmacol Ther (2021).*