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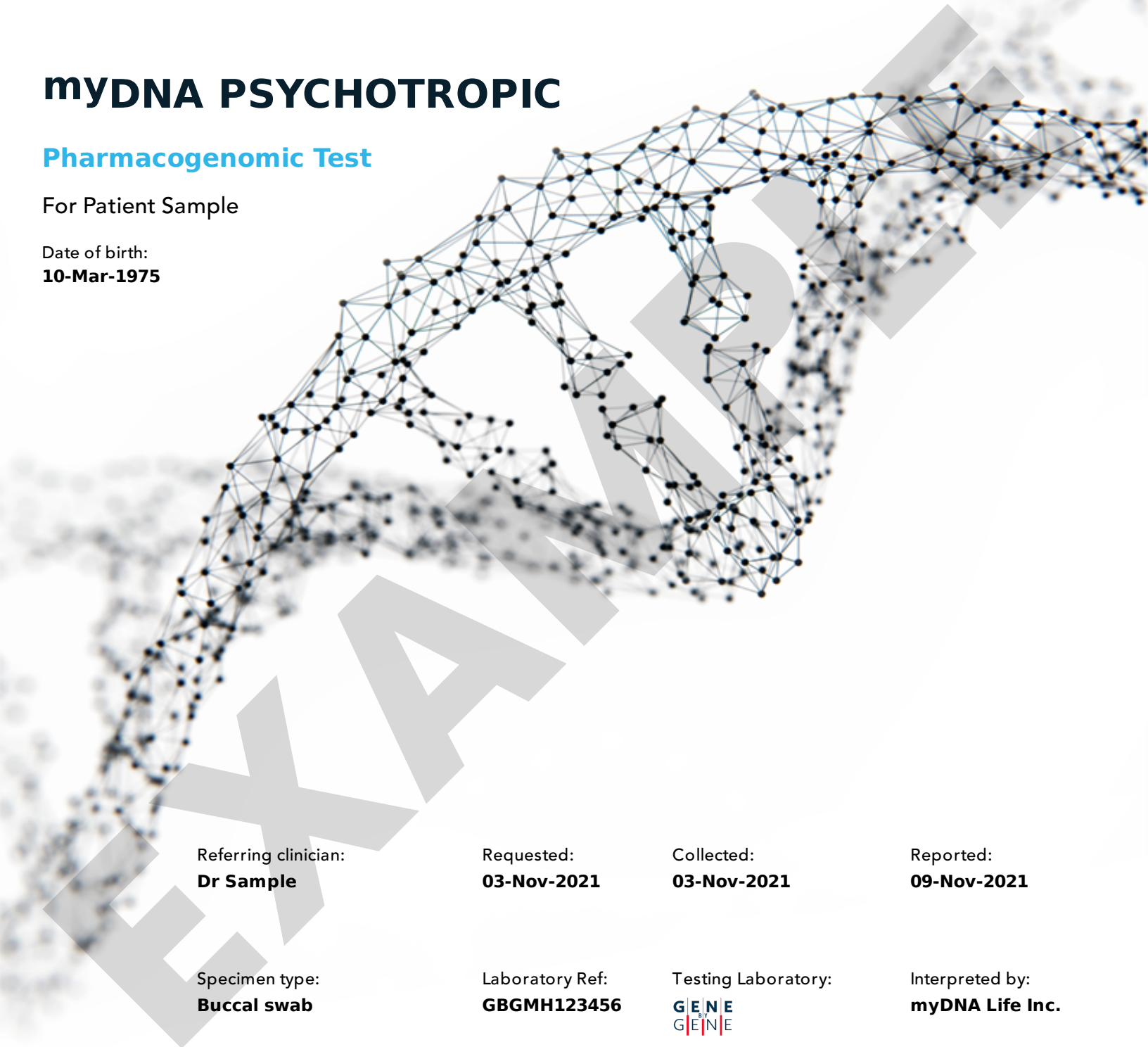
myDNA PSYCHOTROPIC

Pharmacogenomic Test

For Patient Sample

Date of birth:
10-Mar-1975



EXAMPLE

Referring clinician:	Requested:	Collected:	Reported:
Dr Sample	**03-Nov-2021**	**03-Nov-2021**	**09-Nov-2021**
Specimen type:	Laboratory Ref:	Testing Laboratory:	Interpreted by:
Buccal swab	**GBGMH123456**	GENE BY GENE	**myDNA Life Inc.**

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ABOUT THIS REPORT

This report provides clinically relevant information on what the patient's genetic results predict about their response to a number of medications covered by this report.

The information concerns drug metabolism and plasma concentrations (drug exposure), as well as the potential for altered clinical effects.

Based on the available information found in the published literature, each medication has been assigned a category according to the likely clinical significance of each gene-drug interaction.
The four categories are:

MAJOR PRESCRIBING CONSIDERATIONS

A potentially significant effect on drug response is predicted. There may be guidelines or a drug label recommending consideration be given to a change in the dose, the medication type, or further monitoring in order to minimize the risk of the potential clinical issue noted.
Of note, "Major" prescribing considerations do not always preclude the use of a specific medication or necessitate a dosage change if the drug is currently effective and well tolerated, this will be dependent on the individual gene-drug interaction and the clinical circumstances.

MINOR PRESCRIBING CONSIDERATIONS

Altered drug response is possible, but either the clinical significance is thought to be minor or there is currently limited evidence available. Consider monitoring for any potential clinical effects annotated in this report. There are generally no specific recommendations to alter dosage or medication according to current guidelines.

USUAL PRESCRIBING CONSIDERATIONS

Genetic results are not predicted to have a significant effect on drug response, based on the literature currently available, and there are no additional prescribing considerations. Other factors may still influence drug response and therefore usual monitoring for adverse effects and efficacy still applies.

NO PHARAMCOGENOMIC PRESCRIBING CONSIDERATIONS

These medications do not have significant gene-drug interactions identified and standard prescribing considerations apply.

PHARMACOGENOMIC GUIDELINES

For many medications covered in this report, evidence-based guidelines and drug label information are available and where relevant are referenced in this report. Key practice guidelines include:

1. Clinical Pharmacogenetics Implementation Consortium (CPIC)
2. The Royal Dutch Pharmacists Association – Pharmacogenetics Working Group (DPWG).
3. The FDA Table of Pharmacogenetic Associations and drug label information

REPORT BREAKDOWN

The report consists of the following 4 sections:

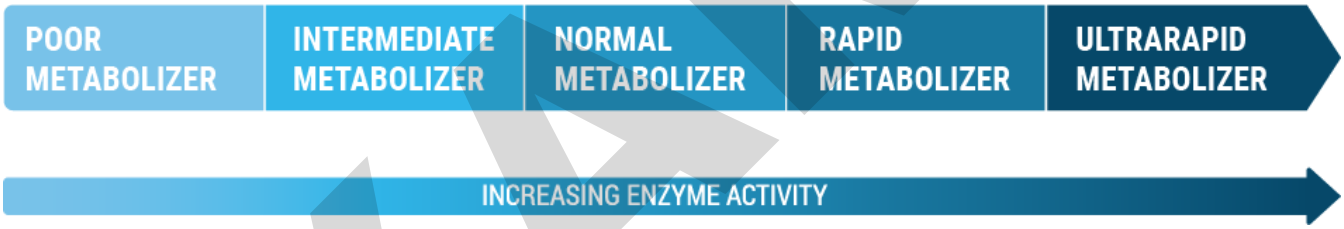
1. Genetic test results summary – presents the patients genotypes for the genes relevant to the medications covered by this report.
2. Medication tables arranged according to the four categories of MAJOR, MINOR, USUAL or NO PHARMACOGENOMIC prescribing considerations.
3. Details of test results – for example, an explanation of how the genotypes have been used to predict CYP enzyme function and the likely general effect on drug metabolism and plasma concentrations (drug exposure).
4. References – list of key peer-reviewed literature that has been used to produce the report.

EXAMPLE

TEST RESULTS SUMMARY

GENE	GENOTYPE	PREDICTED PHENOTYPE
CYP1A2	*1A/*1A	**Normal metabolizer**
CYP2B6	*6/*6	**Poor metabolizer**
CYP2C19	*1/*1	**Normal metabolizer**
CYP2C9	*1/*3	**Intermediate metabolizer**
CYP2D6	*4/*4	**Poor metabolizer**
CYP3A4	*1/*1	**Normal metabolizer**
HLA-A*31:01 (rs1061235)	TT	**Higher risk of certain hypersensitivity reactions**
HLA-B*15:02 (rs144012689)	TT	**Lower risk of certain hypersensitivity reactions**

Detailed interpretations of genetic test results are provided at the end of this report.



EXAMPLE

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ANTIDEPRESSANTS - Important Genes (CYP1A2, CYP2B6, CYP2C19, CYP2C9, CYP2D6)

Each antidepressant below has been allocated to a major, minor, usual, or no prescribing considerations quadrant based on the pharmacogenomic test results. NOTE: These classifications and recommendations do not account for the effect of any inhibitors or inducers and this is not an all-inclusive list of antidepressants.

MAJOR PRESCRIBING CONSIDERATIONS

- AMITRIPTYLINE (TCA)
- CLOMIPRAMINE (TCA)
- DESIPRAMINE (TCA)
- DOTHIEPIN (TCA)
- DOXEPIN (TCA)
- FLUOXETINE (SSRI)
- FLUVOXAMINE (SSRI)
- IMIPRAMINE (TCA)
- NORTRIPTYLINE (TCA)
- PAROXETINE (SSRI)
- TRIMIPRAMINE (TCA)
- VENLAFAXINE (SNRI)
- VORTIOXETINE

MINOR PRESCRIBING CONSIDERATIONS

- AMOXAPINE (TCA)
- BUPROPION
- DULOXETINE (SNRI)
- MIANSERIN
- MIRTAZAPINE
- PROTRIPTYLINE (TCA)

USUAL PRESCRIBING CONSIDERATIONS

- AGOMELATINE
- CITALOPRAM (SSRI)
- ESCITALOPRAM (SSRI)
- MOCLOBEMIDE
- SERTRALINE (SSRI)

NO PHARMACOGENOMIC PRESCRIBING CONSIDERATIONS

- DESVENLAFAXINE (SNRI)
- SELEGILINE
- TRAZODONE
- VILAZODONE
- LEVOMILNACIPRAN

EXAMPLE

ANTIPSYCHOTICS - Important Genes (CYP1A2, CYP2D6, CYP3A4)

Each antipsychotic below has been allocated to a major, minor, usual, or no prescribing considerations quadrant based on the pharmacogenomic test results. NOTE: These classifications and recommendations do not account for the effect of any inhibitors or inducers and this is not an all-inclusive list of antipsychotics.

MAJOR PRESCRIBING CONSIDERATIONS

- ARIPIPRAZOLE
- ARIPIPRAZOLE LAUROXIL
- BREXPIPRAZOLE
- HALOPERIDOL
- ILOPERIDONE
- PIMOZIDE
- RISPERIDONE
- THIORIDAZINE
- ZUCLOPENTHIXOL

MINOR PRESCRIBING CONSIDERATIONS

- CHLORPROMAZINE
- CLOZAPINE
- PERPHENAZINE

USUAL PRESCRIBING CONSIDERATIONS

- OLANZAPINE
- QUETIAPINE

NO PHARMACOGENOMIC PRESCRIBING CONSIDERATIONS

- ASENAPINE
- LURASIDONE
- PALIPERIDONE
- ZIPRASIDONE

EXAMPLE

OTHER PSYCHOTROPICS - Important Genes (CYP2C19, CYP2D6, HLA-A*31:01 (rs1061235), HLA-B*15:02 (rs144012689))

This section includes medications that belong to the following groups: ADHD stimulants and non-stimulants, mood stabilizers, hypnotics and anxiolytics. Each medication below has been allocated to a major, minor, usual, or no prescribing considerations quadrant based on the pharmacogenomic test results. NOTE: These classifications and recommendations do not account for the effect of any inhibitors or inducers and this is not an all-inclusive list of psychotropic medications.

MAJOR PRESCRIBING CONSIDERATIONS

- AMPHETAMINE (PSYCHOSTIMULANT)
- ATOMOXETINE
- CARBAMAZEPINE

MINOR PRESCRIBING CONSIDERATIONS

- DEXTROAMPHETAMINE (PSYCHOSTIMULANT)
- LISDEXAMFETAMINE (PSYCHOSTIMULANT)

USUAL PRESCRIBING CONSIDERATIONS

- CLOBAZAM (BENZODIAZEPINE)
- DIAZEPAM (BENZODIAZEPINE)
- OXCARBAZEPINE

NO PHARMACOGENOMIC PRESCRIBING CONSIDERATIONS

- ALPRAZOLAM (BENZODIAZEPINE)
- CLONAZEPAM (BENZODIAZEPINE)
- CLONIDINE
- DEXMETHYLPHENIDATE (PSYCHOSTIMULANT)
- GUANFACINE
- LORAZEPAM (BENZODIAZEPINE)
- METHYLPHENIDATE (PSYCHOSTIMULANT)
- OXAZEPAM (BENZODIAZEPINE)
- TEMAZEPAM (BENZODIAZEPINE)
- ZOLPIDEM

EXAMPLE

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ANTIDEPRESSANTS

The following tables provide reference information to consider for antidepressants categorized as having major, minor or usual prescribing considerations, based on the genetic test results. This information is intended as a guide and to be considered in addition to other clinical information as part of a comprehensive clinical review by the clinician. NOTE: These classifications and recommendations do not account for the effect of any inhibitors or inducers.

MAJOR PRESCRIBING CONSIDERATIONS

MEDICATION	INTERPRETATION	RECOMMENDATION
AMITRIPTYLINE (TCA)	**CYP2D6 - Poor metabolizer** **CYP2C19 - Normal metabolizer:** Amitriptyline is metabolized by CYP2C19 into an active metabolite, which is further metabolized by CYP2D6 into an inactive metabolite. Normal metabolism of amitriptyline and negligible metabolism (via CYP2D6) of the active metabolite are predicted. Higher plasma concentrations of the active metabolite may increase the risk of adverse effects.	For use at higher doses such as in the treatment of depression, CPIC[1] provides a strong recommendation to avoid amitriptyline use and consider use of an alternative not metabolized by CYP2D6. If a tricyclic is required, consider 50% reduction of the recommended steady-state starting dose. Consider therapeutic drug monitoring to guide dose adjustments. For use at lower doses such as in treatment of neuropathic pain, initial dose adjustments are not recommended but close monitoring for adverse effects is advisable.
CLOMIPRAMINE (TCA)	**CYP2D6 - Poor metabolizer** **CYP2C19 - Normal metabolizer:** Clomipramine is metabolized by CYP2C19 into an active metabolite, which is further metabolized by CYP2D6 into an inactive metabolite. Normal metabolism of clomipramine and negligible metabolism (via CYP2D6) of the active metabolite are predicted. Higher plasma concentrations of the active metabolite may increase the risk of adverse effects.	CPIC[1] provides an optional recommendation to avoid clomipramine use and consider use of an alternative not metabolized by CYP2D6. If a tricyclic is required, consider 50% reduction of the recommended steady-state starting dose. Consider therapeutic drug monitoring to guide dose adjustments. Note that these recommendations only apply to higher initial doses of tricyclic antidepressants for treatment of conditions such as depression.
DESIPRAMINE (TCA)	**CYP2D6 - Poor metabolizer:** Greatly reduced desipramine metabolism and increased drug exposure are predicted. An increased risk of adverse effects is expected.	CPIC guidelines[1] provide an optional recommendation to avoid desipramine and consider an alternative antidepressant not metabolized by CYP2D6. If prescribing desipramine, CPIC guidelines recommend a 50% reduction of the recommended steady-state starting dose, as well as using therapeutic drug monitoring to guide dose adjustments. Note that these recommendations only apply to higher initial doses of tricyclic antidepressants for treatment of conditions such as depression.
DOTHIEPIN (TCA)	**CYP2D6 - Poor metabolizer** **CYP2C19 - Normal metabolizer:** Dothiepin is metabolized by CYP2C19 into an active metabolite, which is further metabolized by CYP2D6 into an inactive metabolite. Normal metabolism of dothiepin and negligible metabolism (via CYP2D6) of the active metabolite are predicted. Higher plasma concentrations of the active metabolite may increase the risk of adverse effects.	CPIC[1] provides an optional recommendation to avoid dothiepin use and consider use of an alternative not metabolized by CYP2D6. If a tricyclic is required, consider 50% reduction of the recommended steady-state starting dose. Consider therapeutic drug monitoring to guide dose adjustments. Note that these recommendations only apply to higher initial doses of tricyclic antidepressants for treatment of conditions such as depression.

EXAMPLE

MEDICATION	INTERPRETATION	RECOMMENDATION
DOXEPIN (TCA)	**CYP2D6 - Poor metabolizer** **CYP2C19 - Normal metabolizer:** Doxepin is metabolized by CYP2C19 into an active metabolite, which is further metabolized by CYP2D6 into an inactive metabolite. Normal metabolism of doxepin and negligible metabolism (via CYP2D6) of the active metabolite are predicted. Higher plasma concentrations of the active metabolite may increase the risk of adverse effects.	CPIC[1] provides an optional recommendation to avoid doxepin use and consider use of an alternative not metabolized by CYP2D6. If a tricyclic is required, consider 50% reduction of the recommended steady-state starting dose. Consider therapeutic drug monitoring to guide dose adjustments. Note that these recommendations only apply to higher initial doses of tricyclic antidepressants for treatment of conditions such as depression.
FLUOXETINE (SSRI)	**CYP2D6 - Poor metabolizer** **CYP2C9 - Intermediate metabolizer:** The metabolism of fluoxetine is complex due to the involvement of several CYP enzymes (especially CYP2D6 and CYP2C9), the formation of active metabolites and the enzyme-inhibiting effect of the parent drug and metabolites (especially on CYP2D6). The CYP2D6 genotype predicts increased fluoxetine exposure and reduced formation of the active S-norfluoxetine metabolite. The CYP2C9 genotype predicts reduced metabolism via this pathway. There may be an increased risk of adverse effects.	Based on the CYP2D6 genotype, DPWG[2] recommends that no specific action on fluoxetine dosing is required for this genotype. Monitor for altered clinical effect, including adverse effects. The FDA[3] has cautioned regarding this genotype and increased risk for QT prolongation with fluoxetine. If adverse effects are a concern, consider an alternative antidepressant for which normal metabolism is predicted.
FLUVOXAMINE (SSRI)	**CYP2D6 - Poor metabolizer** **CYP1A2 - Normal metabolizer:** Fluvoxamine is metabolized by both CYP2D6 (predominant pathway) and CYP1A2. Negligible metabolism by CYP2D6 and normal metabolism by CYP1A2 (not affected by enzyme inducers such as cigarette smoke) are predicted. Fluvoxamine itself will inhibit CYP1A2. There may be increased exposure to fluvoxamine and potentially increased risk of adverse effects.	Based on the CYP2D6 genotype, CPIC[4] provides an optional recommendation to consider a 25-50% reduction of the recommended starting dose and titrate to response. Alternatively, CPIC recommends using an alternative drug not metabolized by CYP2D6. DPWG [5] suggests no specific action on fluvoxamine dosing is required based on this CYP2D6 genotype.
IMIPRAMINE (TCA)	**CYP2D6 - Poor metabolizer** **CYP2C19 - Normal metabolizer:** Imipramine is metabolized by CYP2C19 into an active metabolite, which is further metabolized by CYP2D6 into an inactive metabolite. Normal metabolism of imipramine and negligible metabolism (via CYP2D6) of the active metabolite are predicted. Higher plasma concentrations of the active metabolite may increase the risk of adverse effects.	CPIC[1] provides an optional recommendation to avoid imipramine use and consider use of an alternative not metabolized by CYP2D6. If a tricyclic is required, consider 50% reduction of the recommended steady-state starting dose. Consider therapeutic drug monitoring to guide dose adjustments. Note that these recommendations only apply to higher initial doses of tricyclic antidepressants for treatment of conditions such as depression.

EXAMPLE

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MEDICATION	INTERPRETATION	RECOMMENDATION
NORTRIPTYLINE (TCA)	**CYP2D6 - Poor metabolizer:** Greatly reduced nortriptyline metabolism and increased drug exposure are predicted. An increased risk of adverse effects is expected.	For use at higher doses such as in the treatment of depression, CPIC guidelines[1] provide a strong recommendation to avoid nortriptyline and consider an alternative antidepressant not metabolized by CYP2D6. If prescribing nortriptyline, CPIC guidelines recommend a 50% reduction of the recommended steady-state starting dose, as well as using therapeutic drug monitoring to guide dose adjustments. For use at lower doses such as in treatment of neuropathic pain, initial dose adjustments are not recommended but close monitoring for adverse effects is advisable.
PAROXETINE (SSRI)	**CYP2D6 - Poor metabolizer:** Greatly reduced metabolism by CYP2D6 and greatly increased drug exposure are predicted. There may be increased adverse effects.	CPIC[4] guidelines provide an optional recommendation to select an alternative drug not predominantly metabolized by CYP2D6. If using paroxetine, consider a 50% reduction of the recommended starting dose and titrate to response. It would also be reasonable to monitor for adverse effects.
TRIMIPRAMINE (TCA)	**CYP2D6 - Poor metabolizer** **CYP2C19 - Normal metabolizer:** Trimipramine is metabolized by CYP2C19 into an active metabolite, which is further metabolized by CYP2D6 into an inactive metabolite. Normal metabolism of trimipramine and negligible metabolism (via CYP2D6) of the active metabolite are predicted. Higher plasma concentrations of the active metabolite may increase the risk of adverse effects.	CPIC[1] provides an optional recommendation to avoid trimipramine use and consider use of an alternative not metabolized by CYP2D6. If a tricyclic is required, consider 50% reduction of the recommended steady-state starting dose. Consider therapeutic drug monitoring to guide dose adjustments. Note that these recommendations only apply to higher initial doses of tricyclic antidepressants for treatment of conditions such as depression.
VENLAFAXINE (SNRI)	**CYP2D6 - Poor metabolizer:** Greatly reduced metabolism of venlafaxine into O-desvenlafaxine (also an active drug) is predicted. This will result in increased venlafaxine exposure and reduced O-desvenlafaxine exposure. There may be an increased risk of adverse effects, such as gastrointestinal discomfort. There are indications that the effectiveness of venlafaxine is reduced when used for management of depression in patients with this genotype.	The DPWG[6] recommends: It is not possible to offer adequately substantiated advice for dose reduction based on the literature. 1. Choose an alternative. 2. If an alternative is not an option and side effects occur: a) Reduce the dose b) Check the plasma concentrations of venlafaxine and O-desmethylvenlafaxine (this is not routinely available for venlafaxine). It is not known whether it is possible to reduce the dose to such an extent that effectiveness is maintained without side effects. In general, it is assumed that the effectiveness is determined by the sum of the plasma concentrations of venlafaxine and O-desmethylvenlafaxine. However, the side effects do not appear to be related to this sum.
VORTIOXETINE	**CYP2D6 - Poor metabolizer:** Negligible metabolism by CYP2D6 and increased drug exposure is predicted. This may be associated with an increased risk of concentration-dependent adverse effects.	The TGA approved Product Information[7] states that a dose adjustment is not required. The FDA[8] approved labelling states that the recommended maximum dose is 10mg for CYP2D6 poor metabolizers. Regardless of which dosing advice is followed, be alert for adverse effects.

EXAMPLE

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MINOR PRESCRIBING CONSIDERATIONS

MEDICATION	INTERPRETATION	RECOMMENDATION
AMOXAPINE (TCA)	**CYP2D6 - Poor metabolizer:** Reduced metabolism of amoxapine by CYP2D6 is predicted and therefore increased drug exposure is possible.[9] The clinical significance of this is not known. The FDA notes that systemic concentrations may be altered with this genotype.[10]	No genotype-guided dosing recommendation available. Monitor for adverse effects.
BUPROPION	**CYP2B6 - Poor metabolizer:** Individuals with this genotype may have reduced bupropion metabolism and formation of the active metabolite hydroxybupropion (this is extrapolated mainly from data involving the *6 reduced function allele), as compared with individuals carrying only normal or increased function alleles.[11], [12] Reduced CYP2B6 function may result in reduced effect and/or adverse effects, however, direct evidence is lacking. Other genetic and clinical factors may also affect bupropion metabolism.	Monitor for adequate clinical response and/or adverse effects. No genotype-guided dosing recommendation available. Usual prescribing considerations apply.
DULOXETINE (SNRI)	**CYP2D6 - Poor metabolizer** **CYP1A2 - Normal metabolizer:** Duloxetine is metabolized by both CYP1A2 and CYP2D6, with CYP1A2 likely to have the major role. Negligible metabolism by CYP2D6 and normal metabolism by CYP1A2 (less affected by exposure to enzyme inducers such as tobacco smoke) are predicted. This may lead to a modest increase in exposure to duloxetine. The FDA-approved drug label[13] notes that concomitant administration of duloxetine and a potent CYP1A2 inhibitor to CYP2D6 poor metabolizers resulted in significant increase in drug exposure.	No genotype-guided dosing recommendation available. Be alert to adverse effects.
MIANSERIN	**CYP2D6 - Poor metabolizer:** Negligible metabolism by CYP2D6 and increased drug exposure are predicted. This could increase the risk of adverse effects.	No genotype-guided dosing recommendation is available. Be alert for adverse effects.
MIRTAZAPINE	**CYP2D6 - Poor metabolizer** **CYP1A2 - Normal metabolizer:** Mirtazapine is metabolized by a number of enzymes, including CYP2D6 and CYP1A2. Negligible metabolism by CYP2D6 and normal metabolism by CYP1A2 (less affected by exposure to enzyme inducers such as cigarette smoke) are predicted. There may be increased exposure to mirtazapine and potentially increased clinical effects (therapeutic and/or adverse).	Be alert for adverse effects. Based on the CYP2D6 genotype, DPWG suggests that no specific action on mirtazapine dosing is required.[14]

EXAMPLE

MEDICATION	INTERPRETATION	RECOMMENDATION
PROTRIPTYLINE (TCA)	**CYP2D6 - Poor metabolizer:** Reduced metabolism of protriptyline by CYP2D6 is predicted and therefore increased drug exposure is possible.[15] The clinical significance of this is not known.	No genotype-guided dosing recommendation available. Monitor for adverse effects.

EXAMPLE

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USUAL PRESCRIBING CONSIDERATIONS

MEDICATION	INTERPRETATION	RECOMMENDATION
AGOMELATINE	**CYP1A2 - Normal metabolizer:** Metabolism of agomelatine is less likely to be increased by inducers such as tobacco smoking, daily consumption of cruciferous vegetables or chargrilled meat, and certain medications (e.g. omeprazole).	No genotype-guided dosing recommendation available. Standard dosing and monitoring apply.
CITALOPRAM (SSRI)	**CYP2C19 - Normal metabolizer:** Normal metabolism of citalopram by CYP2C19 is predicted.	CPIC guidelines[4] provide a strong recommendation to initiate therapy with the recommended starting dose.
ESCITALOPRAM (SSRI)	**CYP2C19 - Normal metabolizer:** Normal metabolism of escitalopram by CYP2C19 is predicted.	CPIC guidelines[4] provide a strong recommendation to initiate therapy with the recommended starting dose.
MOCLOBEMIDE	**CYP2C19 - Normal metabolizer:** Normal metabolism of moclobemide is predicted.	Standard dosing and prescribing measures apply.
SERTRALINE (SSRI)	**CYP2C19 - Normal metabolizer:** Normal metabolism of sertraline by CYP2C19 is predicted.	CPIC guidelines[4] provide a strong recommendation to initiate therapy with the recommended starting dose

EXAMPLE

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ANTIPSYCHOTICS

The following tables provide reference information to consider for antipsychotics categorized as having major, minor or usual prescribing considerations, based on the genetic test results. This information is intended as a guide and to be considered in addition to other clinical information as part of a comprehensive clinical review by the clinician. NOTE: These classifications and recommendations do not account for the effect of any inhibitors or inducers.

MAJOR PRESCRIBING CONSIDERATIONS

MEDICATION	INTERPRETATION	RECOMMENDATION
ARIPIPRAZOLE	**CYP2D6 - Poor metabolizer:** Poor metabolism by CYP2D6 and increased drug exposure are predicted. This may increase the risk of concentration-dependent adverse effects.	FDA-approved labelling [16] advises use of 50% of the usual dose. Additionally, if aripiprazole is prescribed together with a strong CYP3A4 inhibiting drug, the dose should be reduced to 25% of the usual dose. For the injectable depot (Abilify Maintena), the FDA- approved label and TGA-approved product information [17] recommends for CYP2D6 poor metabolizers to use a starting and maintenance dose of 300 mg and for CYP2D6 poor metabolizers taking CYP3A4 inhibitors, a 200 mg dose is advised. Note the DPWG[18] recommends administering no more than 10mg/day or 300 mg/month (67-75% of the standard maximum dose), for CYP2D6 poor metabolizers.
ARIPIPRAZOLE LAUROXIL	**CYP2D6 - Poor metabolizer:** Poor metabolism by CYP2D6 and increased drug exposure are predicted. This may increase the risk of concentration-dependent adverse effects.	Aristada Initio®: The FDA-approved drug label[19] advises avoiding use of Aristada Initio in CYP2D6 poor metabolizers. Aristada®: For patients known to be CYP2D6 poor metabolizers and are on concomitant strong CYP3A4 inhibitors for more than 2 weeks, the FDA-approved drug label[20] advises reducing the dose to 441 mg from 662 mg, 882 mg or 1064 mg for poor metabolizers. No dosage adjustment is required in patients tolerating 441 mg of Aristada. For patients known to be CYP2D6 poor metabolizers and on concomitant strong CYP2D6 inhibitors, no dose adjustment is required.
BREXPIPRAZOLE	**CYP2D6 - Poor metabolizer:** Poor metabolism by CYP2D6 and increased drug exposure are predicted. This may increase the risk of concentration-dependent adverse effects.	DPWG guidelines and FDA-approved labelling[21], [22] advise initial treatment with 50% of the usual dose and adjusting according to clinical response. Additionally, if brexpiprazole is prescribed together with a strong CYP3A4 inhibiting drug, the dose should be reduced to 25% of the usual dose.[22]
HALOPERIDOL	**CYP2D6 - Poor metabolizer:** Poor reduced metabolism by CYP2D6 and increased drug exposure are predicted. This may increase the risk of concentration-dependent adverse effects.	The DPWG[23] suggests reducing the initial dose of haloperidol by 50% and adjusting to effect, or using an alternative drug.

EXAMPLE

MEDICATION	INTERPRETATION	RECOMMENDATION
ILOPERIDONE	**CYP2D6 - Poor metabolizer:** Significantly reduced metabolism of iloperidone by CYP2D6 is predicted and therefore increased drug exposure is possible, leading to an increased risk of adverse effects. The FDA-approved drug label notes that poor metabolizers are expected to have higher drug exposures than extensive metabolizers. [24]	The FDA-approved drug label advises that poor metabolizers should have their dose reduced by one-half.[24]
PIMOZIDE	**CYP2D6 - Poor metabolizer:** Negligible metabolism by CYP2D6 and significantly increased drug exposure are predicted. This may increase the risk of concentration dependant adverse effects.	FDA-approved[25] labelling advises: 1) in children, not exceeding a dose of 0.05mg/kg/day and not increasing the dose earlier than 14 days; 2) in adults, not exceeding a dose of 4mg/day and not increasing the dose earlier than 14 days. The DWPG[26] recommends using no more than 50% of the standard maximum dose.
RISPERIDONE	**CYP2D6 - Poor metabolizer:** Poor metabolism and increased drug exposure to risperidone is predicted. This has been associated with both an increased risk of certain adverse effects and a stronger decrease in symptoms when used in schizophrenia. An increased proportion of therapeutic failure has been observed with this genotype.	The DPWG[27] suggests using 67% of the standard dose. If problematic side effects originating from the central nervous system occur despite this reduced dose, a further reduction in dose to 50% of the standard dose is advised.
THIORIDAZINE	**CYP2D6 - Poor metabolizer:** Negligible metabolism by CYP2D6 and significantly increased drug exposure are predicted, with the increased risk of adverse effects. The reduction in clearance of thioridazine is associated with increased risk of Torsades de pointes and/or sudden death. Other factors contributing to this increased risk include: bradycardia, hypokalaemia, concomitant use of other drugs that prolong QTc interval, and presence of congenital prolongation of the QT interval.	The FDA-approved drug label states that thioridazine is contraindicated in patients with reduced activity of CYP2D6.[28]
ZUCLOPENTHIXOL	**CYP2D6 - Poor metabolizer:** Poor metabolism and increased drug exposure are predicted. This has been associated with an increased risk of adverse effects.	The DPWG[29] advises starting with 50% of the standard dose or selecting an alternative drug according to current guidelines.

EXAMPLE

MINOR PRESCRIBING CONSIDERATIONS

MEDICATION	INTERPRETATION	RECOMMENDATION
CHLORPROMAZINE	**CYP2D6 - Poor metabolizer:** Greatly reduced metabolism of chlorpromazine by CYP2D6 and increased drug exposure are predicted. There may be an increased risk of adverse effects.	No genotype-guided dosing recommendation available. Monitor for adverse effects.
CLOZAPINE	**CYP2D6 - Poor metabolizer** **CYP1A2 - Normal metabolizer:** Based on the CYP1A2 genotype, normal metabolism is predicted. The highly inducible genotype *1F/*1F is not present and therefore metabolism is less likely to be increased by inducers such as tobacco smoking, daily consumption of cruciferous vegetables or chargrilled meat, and certain medications (e.g. omeprazole). The FDA-approved drug label[30] states that in CYP2D6 poor metabolizers, plasma concentrations of clozapine may be increased.	Based on the CYP1A2 genotype, standard dosing and prescribing measures apply. Based on the CYP2D6 genotype, the FDA-approved drug label[30] states that it may be necessary to reduce the dose in CYP2D6 poor metabolizers, as they may develop higher than expected plasma concentrations when given usual doses.
PERPHENAZINE	**CYP2D6 - Poor metabolizer:** Significantly reduced metabolism of perphenazine by CYP2D6 is predicted and therefore increased drug exposure is possible, leading to an increased risk of adverse effects. The FDA-approved drug label notes that poor metabolizers are expected to have higher drug concentrations than extensive metabolizers, and that one study has demonstrated an increased risk of adverse effects in poor metabolizers than in extensive metabolizers. [31]	No genotype-guided dosing recommendation available. Monitor closely for adverse effects.

EXAMPLE

USUAL PRESCRIBING CONSIDERATIONS

MEDICATION	INTERPRETATION	RECOMMENDATION
OLANZAPINE	**CYP1A2 - Normal metabolizer:** Normal metabolism by CYP1A2 (note that whilst CYP2D6 is also involved in olanzapine metabolism, it is thought to have a minor role). The highly inducible CYP1A2 genotype *1F/*1F is not present and therefore metabolism is less likely to be increased by inducers such as tobacco smoking, daily consumption of cruciferous vegetables or chargrilled meat, and certain medications (e.g. omeprazole).	Standard dosing and prescribing measures apply.
QUETIAPINE	**CYP3A4 - Normal metabolizer:** Normal metabolism of quetiapine is predicted.	Standard dosing and prescribing measures apply.

EXAMPLE

OTHER PSYCHOTROPICS

The following tables provide reference information to consider for other psychotropics categorized as having major, minor or usual prescribing considerations, based on the genetic test results. This information is intended as a guide and to be considered in addition to other clinical information as part of a comprehensive clinical review by the clinician. NOTE: These classifications and recommendations do not account for the effect of any inhibitors or inducers.

MAJOR PRESCRIBING CONSIDERATIONS

MEDICATION	INTERPRETATION	RECOMMENDATION
AMPHETAMINE (PSYCHOSTIMULANT)	**CYP2D6 - Poor metabolizer:** Although the enzymes involved in amphetamine metabolism have not been clearly defined, CYP2D6 is involved in the formation of an active metabolite 4-hydroxy-amphetamine. Reduced metabolism by CYP2D6 is predicted which could lead to variations in amphetamine metabolism.[32] The increased levels of amphetamine may lead to an increased risk of adverse effects.[10]	The FDA advises consideration of use of a lower starting dosage, or use of an alternative agent.[10] Monitor for adverse effects.
ATOMOXETINE	**CYP2D6 - Poor metabolizer:** Greatly reduced metabolism by CYP2D6 and greatly increased drug exposure is predicted. An increased risk of some side effects has been shown for this genotype (e.g. increased blood pressure and heart rate, QT interval prolongation, dry mouth, erectile dysfunction and insomnia) but also greater improvement of ADHD symptoms as compared to non-poor metabolizers in those who tolerate treatment. This genotype is associated with lower final dose requirements.	CPIC[33] provides a strong recommendation for children and moderate recommendation for adults for dosing of atomoxetine. Refer to CPIC guidelines for details. In summary, Adults: initiate at 40 mg/day. If no clinical response and no adverse events after 2 weeks, increase dose to 80 mg/day. If inadequate response after 2 weeks, consider use of plasma concentrations 2-4 hours after dosing to guide titration. Children: initiate at 0.5mg/kg/day. If no clinical response and no adverse events after 2 weeks, consider use of plasma concentrations 4 hours after dosing to guide titration. Note: FDA-approved drug label[34] recommends maximum doses of 1.4mg/kg/day in children up to 70kg and 100 mg daily in adults or children over 70kg. Note: dosing recommendations should be considered with other clinical factors by the treating clinician(s). For CYP2D6 poor metabolizers or patients on strong CYP2D6 inhibitors, FDA approved labelling[34] advises using a reduced dosing strategy (starting dose 0.5mg/kg/day, and only increasing to 1.2mg/kg/day after 4 weeks if required) in children and adolescent patients with body weight <70kg. For children and adolescents >70kg, and for adults, atomoxetine should be initiated at 40mg/day and only increased to 80mg/day after four weeks if necessary.

EXAMPLE

MEDICATION	INTERPRETATION	RECOMMENDATION
CARBAMAZEPINE	**HLA-A*31:01 (rs1061235) - Higher risk of certain hypersensitivity reactions** **HLA-B*15:02 (rs144012689) - Lower risk of certain hypersensitivity reactions:** The rs1061235 TT result provides a moderate prediction of the presence of the HLA-A*31:01 allele. The rs144012689 TT result provides a high prediction of the absence of the HLA-B*15:02 allele. If the HLA-A*31:01 is present, there is an increased risk of serious cutaneous hypersensitivity reactions (such as Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN)), drug reaction with eosinophilia and systemic symptoms (DRESS) and maculopapular exanthema (MPE) within the first four months of initiating carbamazepine therapy.	If the patient is carbamazepine naive, consider the use of an alternative medication if clinically appropriate. If carbamazepine is the drug of choice, then specific HLA-A*31:01 testing is recommended to confirm the presence of this allele. If confirmed, it is recommended to avoid carbamazepine use. If patient is carbamazepine-naive and alternative agents are not available, CPIC provide an optional recommendation to consider the use of carbamazepine with increased frequency of clinical monitoring. Discontinue therapy at first evidence of a cutaneous adverse reaction. If the patient has previously used carbamazepine for longer than 3 months without any incidence of any cutaneous adverse reactions, CPIC provide an optional recommendation to cautiously consider the use of carbamazepine.[35] If the patient develops any rash or hypersensitivity reactions on carbamazepine, then discontinuation should be considered in accordance with standard prescribing guidelines.[36]

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MINOR PRESCRIBING CONSIDERATIONS

MEDICATION	INTERPRETATION	RECOMMENDATION
DEXTROAMPHETAMINE (PSYCHOSTIMULANT)	**CYP2D6 - Poor metabolizer:** Dextroamphetamine is eliminated by both the kidney (as unchanged drug) and the liver, with CYP2D6 playing a significant role. Negligible metabolism via CYP2D6 and increased dextroamphetamine exposure is predicted. Clinical effects may be increased.	The FDA-approved drug label suggests a lower starting dose and monitoring for adverse effects where there is a lack of CYP2D6 function.[37] .
LISDEXAMFETAMINE (PSYCHOSTIMULANT)	**CYP2D6 - Poor metabolizer:** Lisdexamfetamine is a prodrug of dextroamphetamine (also known as dexamfetamine). Dextroamphetamine is eliminated by both the kidney (as unchanged drug) and the liver, with CYP2D6 playing a significant role. Negligible metabolism via CYP2D6 and increased dextroamphetamine exposure is predicted. Clinical effects may be increased.	The FDA-approved drug label suggests a lower starting dose and monitoring for adverse effects where there is a lack of CYP2D6 function.[38]

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USUAL PRESCRIBING CONSIDERATIONS

MEDICATION	INTERPRETATION	RECOMMENDATION
CLOBAZAM (BENZODIAZEPINE)	**CYP2C19 - Normal metabolizer:** Clobazam is metabolized by CYP3A4 into an active metabolite, N-desmethylclobazam, which is responsible for most of the therapeutic effect. N-desmethylclobazam is further metabolized by CYP2C19 into an inactive metabolite. Normal metabolism of clobazam's active metabolite is predicted. (Note that the effect of variations in CYP3A4 has not been described).	Standard dosing and prescribing measures apply.
DIAZEPAM (BENZODIAZEPINE)	**CYP2C19 - Normal metabolizer:** Diazepam is metabolized by CYP3A4 and CYP2C19 into active metabolites, including desmethyldiazepam, which has a long half-life. The CYP2C19 genotype predicts normal CYP2C19-mediated metabolism of both diazepam and desmethyldiazepam. (Note that the effect of variations in the CYP3A4 gene on diazepam metabolism have not been described).	Standard dosing and prescribing measures apply.
OXCARBAZEPINE	**HLA-B*15:02 (rs144012689) - Lower risk of certain hypersensitivity reactions:** The rs144012689 TT result provides a high prediction of the absence of the HLA-B*15:02 allele. This result is associated with a normal or reduced risk of cutaneous hypersensitivity reactions to oxcarbazepine (such as Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN)).	It would be reasonable to cautiously consider the use of oxcarbazepine as per standard prescribing guidelines.[35] Be aware that this is a screening test only, if the patient develops any rash or hypersensitivity reactions on oxcarbazepine, then discontinuation should be considered in accordance with standard prescribing guidelines.[39]

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GENETIC TEST RESULTS

GENE	GENOTYPE	PREDICTED PHENOTYPE
CYP1A2	*1A/*1A	**Normal metabolizer:** The *1F allele is not present and this individual is predicted to have a normal metabolizer phenotype. Normal metabolism of CYP1A2 substrate drugs is predicted. Furthermore, metabolism is not expected to be increased by exposure to inducers such as tobacco smoking and certain dietary components and drugs.
CYP2B6	*6/*6	**Poor metabolizer:** Due to the presence of two reduced or non-functioning alleles this individual is predicted to have a poor metabolizer phenotype. For a drug extensively metabolized by CYP2B6, drug exposure and clinical effects may either be increased (for an active drug) or decreased (for a prodrug). This individual is at risk of experiencing adverse effects (active drug) or therapeutic failure (prodrug).
CYP2C19	*1/*1	**Normal metabolizer:** Due to the presence of two normal function alleles, this individual is predicted to have a normal metabolizer phenotype. For a drug extensively metabolized by CYP2C19, drug exposure and clinical effects may be expected to lie within the normal range.
CYP2C9	*1/*3	**Intermediate metabolizer:** Due to the presence of one normal function allele and one null allele, this individual is predicted to have an intermediate metabolizer phenotype. For a drug extensively metabolized by CYP2C9, drug exposure and clinical effects may either be increased (for an active drug) or decreased (for a prodrug). This may increase the likelihood of adverse effects (active drug) or therapeutic failure (prodrug).
CYP2D6	*4/*4	**Poor metabolizer:** Due to the presence of two null alleles, this individual is predicted to have a poor metabolizer phenotype. For a drug extensively metabolized by CYP2D6, drug exposure and clinical effects may either be greatly increased (for an active drug) or greatly decreased (for a prodrug). The individual is at risk of experiencing adverse effects (active drug) or therapeutic failure (prodrug).
CYP3A4	*1/*1	**Normal metabolizer:** The *22 allele is not present and this individual is expected to have a normal metabolizer phenotype. Whilst many drugs are known to be metabolized by CYP3A4, relatively few genetic variations have been found that affect metabolism of a limited number of these drugs.

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GENE	GENOTYPE	PREDICTED PHENOTYPE
HLA-A*31:01 (rs1061235)	TT	**Higher risk of certain hypersensitivity reactions:** Testing for a specific rs1061235 variant may be utilized as a screening test for the presence of HLA-A*31:01. HLA-A*31:01 is an allele which, if present, has been associated with hypersensitivity reactions including Stevens-Johnson syndrome, toxic epidermal necrolysis, maculopapular eruptions, and drug reaction with eosinophilia and systemic symptoms (DRESS) with carbamazepine. This TT result provides a moderate prediction of the presence of the HLA-A*31:01 allele. The positive predictive value for this test has been shown to be ~77%.[40] The clinical utility for testing for this variant appears to be particularly relevant for carbamazepine, with the FDA-approved drug label noting that the risks and benefits of carbamazepine therapy should be weighed before considering carbamazepine in patients known to be positive for HLA-A*31:01.[36] As this is a screening test for HLA-A*31:01 only, if a more definitive result is required then DNA sequencing for this allele would be required via another laboratory. The Genetic Testing Registry may be used to identify laboratories that offer this testing: https://www.ncbi.nlm.nih.gov/gtr/
HLA-B*15:02 (rs144012689)	TT	**Lower risk of certain hypersensitivity reactions:** Testing for a specific rs144012689 variant may be utilized as a screening test for the presence of HLA-B*15:02. HLA-B*15:02 is an allele which, if present, is associated with serious cutaneous hypersensitivity reactions (such as Stevens-Johnson syndrome and toxic epidermal necrolysis) for certain medications. This TT result provides a high prediction of the absence of the HLA-B*15:02 allele. The negative predictive value for this test has been shown to be 100%.[41] The clinical utility of testing for this variant appears to be particularly relevant for carbamazepine and oxcarbazepine, as there is more limited evidence for other medications. The FDA-approved drug label notes that HLA-B*1502 is found almost exclusively in patients with Asian ancestry across broad areas of Asia and that patients with ancestry in genetically at risk populations should be screened for the presence of HLA-B*15:02 prior to initiating treatment with carbamazepine.[36] It is noted that this should also be considered prior to initiating treatment with oxcarbazepine.

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ADDITIONAL GENES WITH EMERGING EVIDENCE

This section contains genes that have limited evidence for clinical implementation and are not utilized in how medications are classified under major, minor, usual or no pharmacogenomic prescribing considerations. The data has been included for informational purposes only and there are currently no recommendations to alter prescribing based on genotype.

GENE	GENOTYPE	COMMENTS
ADRA2A	CC	This genetic result may be associated with some reduction in response to methylphenidate compared to GC and GG carriers,[42] however, study results are conflicting. There are currently no recommendations to alter prescribing.
CES1A1	GG	Individuals with this genetic result may have increased metabolism of methylphenidate[43] compared to AA or AG carriers. There are currently no recommendations to alter prescribing.
HTR2A	AA	This genetic result may be associated with a lower risk of side effects for certain SSRIs compared to GG carriers,[44] however, not all studies have shown this and there are currently no recommendations to alter prescribing.
SLC6A4	S/S	This genetic result (two short alleles of the 5HTTLPR) has been associated with some reduction in SSRI response in individuals of Caucasian ancestry compared with LS or LL carriers.[45] However, there are conflicting studies, particularly in other populations. There are currently no recommendations to alter prescribing.
UGT1A4	*1/*1	Individuals with this genetic result may have increased drug exposure of lamotrigine[46] and olanzapine[47] compared to *3/*3 carriers. However, there is conflicting evidence in relation to this effect and there are currently no recommendations to alter prescribing.
UGT2B15	*1/*2	Individuals with this genetic result may have reduced clearance of certain benzodiazepines, such as lorazepam[48] and oxazepam[49] compared to *1/*1 carriers. However there is limited evidence for this effect and there are currently no recommendations to alter prescribing.

EXAMPLE

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Electronic Signature:

This report has been prepared by the myDNA Clinical Team

Laboratory Results provided by:

Gene by Gene Ltd in a CAP and CLIA accredited laboratory (CAP Number 7212851, CLIA Number 45D1102202).

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myDNA DISCLAIMER

Response to medications is complex and may also be influenced by other genetic and non-genetic factors which are not tested for (e.g. patient adherence to prescription regimen, concurrent illness, drug-drug interactions). This report is just one clinical factor which is intended to be considered in addition to other clinical information as part of a comprehensive medical evaluation by the treating clinician. It is advised that medications should not be changed solely based on this report and it is the responsibility of the treating clinician to consider all information relating to the patient to determine the most appropriate course of treatment. Unless instructed by their doctor, patients are advised not to alter the dose or stop any medications based on this report.

Clinical monitoring should occur for all psychotropic medications. It is not intended to imply that drugs listed in this report are approved for certain indications or that they have comparable efficacy or safety. Note that prescribing of some of the listed medications for psychiatric conditions may be considered off-label and approved drug labels should be consulted for guidance regarding their use.

This report outlines gene-drug interactions for the medications listed. Allergic reactions cannot be detected by this test. The test does not detect all known variants in the genes tested.

Genetic counselling is recommended to properly review and explain these results to the tested individual as there may be implications for both the individual in addition to family members. This is not provided by myDNA and responsibility to arrange this is with the ordering clinician or patient.

The pharmacogenomic guidance in this report primarily applies to adult patients over the age of 18 years. Therefore, clinician discretion should be exercised if the guidance in this report is applied to patients under the age of 18 years other than otherwise stated.

Disclaimer of Liability

This myDNA report does not serve as medical advice and does not substitute clinical monitoring. myDNA is not liable for any clinical decisions made based on the results provided in this report as this remains the responsibility of the treating clinician. myDNA strongly believes that this report should be considered as part of a comprehensive medical evaluation by the treating clinician.

The information provided in the report is believed to be accurate and complete at the date reported and is based on the current evidence in the scientific literature. However, the scientific literature is routinely updated as new information becomes available and therefore, the reported drug classifications and clinical considerations may change from the original published version of the report. While myDNA believes the information of this report is accurate and complete, myDNA does not provide any warranties of any kind relating to how the information provided in this report is used or applied by the treating clinician.

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TEST METHODOLOGY AND LIMITATIONS

Gene By Gene is a College of American Pathologists (CAP) accredited and Clinical Laboratory Improvement Amendments (CLIA) certified clinical laboratory qualified to perform high-complexity testing. This test was developed and its performance characteristics determined by Gene by Gene. It has not been cleared or approved by the FDA. FDA does not require this test to go through premarket FDA review. This test is used for clinical purposes. It should not be regarded as investigational or for research. Only the genomic regions listed below were tested; there is a possibility that the tested individual is a carrier for additional, undetected mutations. Direct Sanger sequencing of a gene is unable to identify large deletions of a gene or part of it. Although molecular tests are highly accurate, rare diagnostic errors may occur that interfere with analysis. Sources of these errors include sample mix-up, trace contamination, and other technical errors. The presence of additional variants nearby may interfere with mutation detection. Genetic counseling is recommended to properly review and explain these results to the tested individual.

TEST PANEL OF GENES AND VARIANTS

The current list of reported haplotypes are below. Unless otherwise indicated, the *1 allele denotes the absence of any variant and is designated as the wild type: CYP1A2 *1C (LRG_1274:g.2035G>A), *1F (LRG_1274:g.5732C>A), *1K (LRG_1274:g.[5166C>T; 5732C>A]), *1L (LRG_1274:g.[2035G>A; 5732C>A]), *7 (LRG_1274:g.9427G>A), *11 (LRG_1274:g.6452C>A); CYP2B6 *6 (LRG_1267:g.20638G>T), *18 (LRG_1267:g.26018T>C); CYP2C19 *2 (NG_008384.3:g.24179G>A), *3 (NG_008384.3:g.22973G>A), *4A (NG_008384.3:g.5026A>G), *4B (NG_008384.3:g.[4220C>T; 5026A>G), *5 (NG_008384.3:g. 95058C>T), *6 (NG_008384.3:g.17773G>A), *7 (NG_008384.3:g.24319T>A), *8 (NG_008384.3:g.17736T>C), *17 (NG_008384.3:g.4220C>T); CYP2C9 *2 (LRG_1195:g.9133C>T), *3(LRG_1195:g.48139A>C),*4 (LRG_1195:g.48140T>C), *5 (LRG_1195:g.48144C>G), *6 (LRG_1195:g.16126del), *8 (LRG_1195:g. 9152G>A), *11 (LRG_1195:g. 48067C>T), *12 (LRG_1195:g.55863C>T), *13 (LRG_1195:g.8801T>C), *15 (LRG_1195:g.14625C>A), *25 (LRG_1195:g.9056_9065del), *27 (LRG_1195:g. 9152G>T); CYP2D6 *2 (LRG_303:g.7870C>T; 9200G>C), *3 (LRG_303:g.7569del), *4 (LRG_303:g.[5119C>T; 6866G>A; 9200G>C]), *5 (del(CYP2D6)), *6 (LRG_303:g.6727del), *7 (LRG_303:g.7955A>C), *8 (LRG_303:g.[6778G>T; 7870C>T; 9200G>C), *9 (LRG_303:g. 7635_7637del), *10 (LRG_303:g.[5119C>T; 9200G>C]), *11 (LRG_303:g.[9200G>C ;590G>C]), *12 (LRG_303:g.[5143G>A; 7870C>T; 9200G>C]), *114 (LRG_303:g.[5119C>T; 6778G>A ; 7870C>T; 9200G>C]), *14 (LRG_303:g.[6778G>A ;7870C>T; 9200G>C]),*15 (LRG_303:g.5156dup), *17 (LRG_303:g.[6041C>T; 7870C>T; 9200G>C], *18 (NC_000022.11:g.42126666_42126667insAGTGGGCAC), *19 (LRG_303:g.[7559_7562del; 9200G>C;]), *20 (LRG_303:g.[6996dup; 9200G>C]), *29 (LRG_303:g.[7870C>T;8203G>A;9200G>C], *36 (NC_000022.10:g.[42526694G>A ;42522624_42522669con42536337_42536382]), *41(LRG_303:g.[7870C>T; 8008G>A; 9200G>C]), *69 (LRG_303:g.[5119C>T; 8008G>A; 9200G>C]); CYP3A4 *2 (NG_008421.1:g.20826T>C), *17 (NG_008421.1:g.20728T>C), *22 (NG_008421.1:g.20493C>T); ADRA2A - rs1800544 (LRG_545:g.4714G>A); CES1A1 - rs71647871 (NG_012057.1:g.14506G>A); HLA-A*31:01 - rs1061235 (NG_029217.2:g.8057A>T); HLA-B*15:02 - rs144012689 (NG_023187.1:g.7210A>T); HTR2A - rs6311 (LRG_1008:g.4692G>T); SLC6A4 - NG_011747.2:g.3628_3670del; UGT2B15 - rs1902023 (NG_052676.1:g.5411T>G) and UGT1A4 - rs2011425 (NG_002601.2:g.134219T>A or NG_002601.2:g.134219T>G).

SPEAK TO OUR SPECIALISTS

As part of our clinical service, we have a team of clinical experts available to answer any questions you may have about this report or about pharmacogenomics in general. If you have any such queries, please contact us at clinical@mydna.life

EXAMPLE

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