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MMG ID	MMGSAMPLE	TEST ID	MMGSAMPLEMMPSAMPLE
CUSTOMER NAME	SAMPLE REPORT	SAMPLE TYPE	Buccal Swab
GENDER	Female	SAMPLE COLLECTION DATE	12/5/2016
DOB	--	SAMPLE RECEIPT DATE	12/5/2016
ETHNICITY	Not specified	REPORT DATE	2/6/2018
TEST: MedicaMap™			

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ABOUT THE REPORT

READING YOUR REPORT

DRUG RESPONSE BASED ON METABOLIZER STATUS

Single base variations in certain genes result in varied response to drugs. Bioconversion (drug to active metabolite) and drug clearance depends on gene expression and thereby, enzyme activity.

- Some genetic variations can result in reduced enzyme production and slower rate of drug metabolism. Carriers of these variations are termed "slow metabolizers" or "poor metabolizers". A reduced rate of drug metabolism may lead to elevated plasma drug concentrations and potential adverse effects, known as drug-induced toxicity.
- Some genetic variations can result in elevated enzyme activity and rapid drug metabolism. Carriers of these variations are termed "rapid metabolizers" or "ultrarapid metabolizers". An elevated rate of drug metabolism may lead to subtherapeutic plasma drug concentrations and reduced clinical response, which results in ineffective therapy.
- For some drugs, Guidelines from the Food and Drug Administration (FDA) and the Clinical Pharmacogenetics Implementation Consortium (CPIC) have recommended genetic testing in individuals prior to initiating therapy, for identification of ultrarapid and poor metabolizers. This may be followed by alternate drug evaluation, if necessary**.

**Final patient recommendations to be determined by physician only.

LEGENDS

Appears in the Snapshot. We have tested your DNA for efficacy for the drug.

Appears in the Snapshot. We have tested your DNA for possible adverse reactions for the drug


Appears in the detailed report. Indicates favorable response or low toxicity for the drug.


Appears in the detailed report. Indicates medium response or slightly high toxicity for the drug.


Appears in the detailed report. Indicates poor response or high toxicity for the drug.

ABOUT US

Mapmygenome is a molecular diagnostics company for people who are proactive about their health. Their personal genomics products provide insights into the genetic basis of individuals' health, including traits, lifestyle, drug responses, inherited conditions, and diseases. By combining genetic report and health history with genetic counseling, Mapmygenome provides actionable steps for individuals and their physicians towards a healthier life.

WHAT IS MEDICAMAP

When it comes to medication, doctors agree that one DRUG DOES NOT SUIT ALL. A treatment option that works wonders for one patient may not suit another. As a result, there are times when different therapies may be tested before arriving at the perfect treatment option. Oftentimes, this could impact treatment time and expense.
Recent advances in genetics indicate that the answer to this puzzle of drug action on individuals is determined by their own DNA. With 16+ years of expertise in genomics, Mapmygenome has designed MedicaMap™, a DNA based test to assess drug efficacy and performance. With this test, a physician can prescribe suitable treatment options and dosage that works for the patient.

WHAT DO WE DO WITH YOUR DNA

When we receive your sample, the first thing we do is to isolate and extract your DNA. The extracted DNA is your genetic component and is used by us to identify potentially hazardous markers, which have proven association with health conditions we cover. The markers we look for in your DNA are called SNPs or single nucleotide polymorphisms and these are selected by our scientists after stringent scrutiny of their association with a given health condition.

WHAT IS GENETIC INFORMATION?

Genome is the genetic content or hereditary information of an organism, which is made up of DNA in humans and other higher organisms. DNA is made up of four bases Adenine, Thymine, Guanine, and Cytosine, designated by four letters A, T, G, C, respectively. Although the genome of all humans is almost the same, a minor difference exists among individuals. This difference, which is called genetic variation is responsible for unique phenotype (appearance, e.g., color of skin/eyes, type of hair (curly, smooth), etc.) and difference in the health of each individual. In most of the cases, this difference or variation is passed on to the next generation (inheritance), which confers disease susceptibility in the offspring.

SINGLE NUCLEOTIDE POLYMORPHISM (SNP)

Single nucleotide polymorphism or SNP is a type of genetic variation, where in a single letter difference occurs in the DNA sequence of an individual when compared to others.

Example: Sequence 1: ----AGCCTAATGGGC----
Sequence 2: ----AGCCTAAGGGGC----

Here, in the given example, the first sequence differs from the second sequence only by a single letter (nucleotide T/ G). This single letter variation affects many phenotypic traits, disease susceptibility/resistance, response to drugs, chemicals, radiation, etc.

SNP GENOTYPING

Genotype is the genetic makeup of an organism, and genotyping (process by which the genotype sequence is decoded) is done to understand difference in the genetic makeup between different individuals. SNP genotyping helps to analyze the SNPs present in an individual.

SIGNATURES

REVIEWED BY

Dr. Arun Shastry	Dr. Sandhya Kiran	Dr. Shivaji Jhadav
Head of Medical Operations	Principal Data Scientist	Head of Lab Operations

REFERENCES

- Chua, Eng Wee, and Martin A. Kennedy. "Current state and future prospects of direct-to-consumer pharmacogenetics." *Frontiers in pharmacology* 3 (2012): 152.
- Scott, Stuart A., Katrin Sangkuhl, Alan R. Shuldiner, Jean-Sébastien Hulot, Caroline F. Thorn, Russ B. Altman, and Teri E. Klein. "PharmGKB summary: very important pharmacogene information for cytochrome P450, family 2, subfamily C, polypeptide 19." *Pharmacogenetics and genomics* 22, no. 2 (2012): 159.
- Drozda, Katarzyna, Daniel J. Müller, and Jeffrey R. Bishop. "Pharmacogenomic testing for neuropsychiatric drugs: current status of drug labeling, guidelines for using genetic information, and test options." *Pharmacotherapy: The Journal of Human Pharmacology and Drug Therapy* 34, no. 2 (2014): 166-184.
- Cavallari, Larisa H., Hyunyoung Jeong, and Adam Bress. "Role of cytochrome P450 genotype in the steps toward personalized drug therapy." *Pharmgenomics Pers Med* 4 (2011): 123-136.
- Haga, Susanne B., Rachel Mills, and Jivan Moaddeb. "Pharmacogenetic information for patients on drug labels." *Pharmacogenomics and personalized medicine* 7 (2014): 297.
- Smith, Teri, Susan Sharp, Ann M. Manzardo, and Merlin G. Butler. "Pharmacogenetics informed decision making in adolescent psychiatric treatment: A clinical case report." *International journal of molecular sciences* 16, no. 3 (2015): 4416-4428.

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DISCLAIMER

Mapmygenome does not prescribe or suggest any kind of medication to its customers. The "Drug Responses" here refer to your genetic predisposition to the drugs mentioned in the report. This section is for a physician's reference. This report is based on your genetic response alone. It does not take into account other factors including but not limited to interactions with food/supplements/other medications consumed, timing of the medication, presence of any illness or health conditions, fitness for medication, etc. Decisions pertaining to medication and/or dosage should done in consultation with your doctor.

SNAPSHOT

CARDIOLOGY

STATINS

SIMVASTATIN	TOXICITY	Baseline risk for drug induced toxicity.

ANTIPLATELETS

CLOPIDOGREL	EFFICACY TOXICITY	Ultrarapid metabolizer. Typical likelihood for positive clinical outcome, on standard dose.

ANTICOAGULANTS/BLOOD THINNERS

WARFARIN	EFFICACY TOXICITY	Baseline risk for drug sensitivity. The genetic markers that have been evaluated do not increase risk for warfarin-induced bleeding.

DIABETOLOGY

SULPHONYL UREA ANTIDIABETICS

GLIMEPERIDE	TOXICITY	Baseline risk for drug-induced hemolysis.

PSYCHIATRY

TRICYCLIC ANTIDEPRESSANTS

AMITRIPTYLINE	EFFICACY TOXICITY	Normal metabolizer. The genetic markers that have been evaluated do not increase risk for drug-induced side effects or toxicity.

ANTIPSYCHOTICS

RISPERIDONE	EFFICACY TOXICITY	Normal metabolizer. The genetic markers that have been evaluated do not increase risk for drug-induced side effects or toxicity.

PERPHENAZINE	EFFICACY TOXICITY	Normal metabolizer. The genetic markers that have been evaluated do not increase risk for drug-induced side effects or toxicity.
THIORIDAZINE	EFFICACY TOXICITY	Normal metabolizer. The genetic markers that have been evaluated do not increase risk for drug-induced side effects or toxicity.

ANXIOLYTICS

DIAZEPAM	TOXICITY	Normal metabolizer. Baseline risk for drug-induced side effects.

OPIOID ANTAGONISTS

NALTREXONE	EFFICACY	Slightly high likelihood for positive drug response

NEUROLOGY

ANTICONVULSANTS

PHENYTOIN	TOXICITY	Baseline risk for drug-induced toxicity.

ANTIEPILEPTICS

VALPROIC ACID	TOXICITY	Baseline risk for drug-induced toxicity. The genetic markers that have been evaluated do not increase risk for drug-induced toxicity.

GENERAL MEDICINE

NON STEROIDAL INFLAMMATORY DRUGS (NSAID)

CELECOXIB	EFFICACY TOXICITY	Baseline risk for drug sensitivity. The genetic markers that have been evaluated do not increase risk for drug-induced side effects or toxicity.

OPIOIDS

TRAMADOL	EFFICACY TOXICITY	Normal metabolizer. The genetic markers that have been evaluated do not increase risk for drug-induced side effects or toxicity.

LAXATIVE

PEG-3350	TOXICITY	Baseline risk for drug-induced hemolysis.

ONCOLOGY

PLATINUM-BASED CHEMOTHERAPEUTICS

CISPLATIN	TOXICITY	High enzyme activity. Baseline risk for drug induced toxicity.

KINASE INHIBITOR CHEMOTHERAPEUTICS

DABRAFENIB	TOXICITY	Baseline risk for drug-induced hemolysis.
LAPATINIB	TOXICITY	Baseline risk for drug-induced toxicity. The genetic markers that have been evaluated do not increase risk for drug-induced toxicity.

IMMUNOSUPPRESSANTS

THIOPURINES	TOXICITY	High enzyme activity. Baseline risk for drug induced toxicity.

IMMUNOLOGY

IMMUNOSUPPRESSANTS

THIOPURINES	TOXICITY	High enzyme activity. Baseline risk for drug induced toxicity.

GASTROENTEROLOGY

PROTON PUMP INHIBITORS

OMEPRAZOLE	EFFICACY	Ultrarapid metabolizer. The genetic markers that have been evaluated increase the likelihood for therapy failure. Higher dose or alternate drug may be required

INFECTIOUS DISEASE

ANTIVIRALS

ABACAVIR	TOXICITY	Baseline risk for drug induced toxicity.
EFAVIRENZ	TOXICITY	Baseline risk for drug-induced toxicity. The genetic markers that have been evaluated do not increase risk for drug-induced toxicity.

RIBAVIRIN	EFFICACY	Slightly low likelihood for for positive drug response

ANTIBACTERIALS

FLUCLOXACILLIN	TOXICITY	Baseline risk for drug induced liver toxicity.
DAPSONE	TOXICITY	Baseline risk for drug-induced hemolysis.
RIFAMPIN	EFFICACY TOXICITY	Slow acetylator, high risk for drug-induced toxicity. Reduced dose may be required.

OTHERS

ELIGLUSTAT	EFFICACY TOXICITY	Normal metabolizer. The genetic markers that have been evaluated do not increase risk for drug-induced side effects or toxicity.
DEXTROMETHORPHAN & QUINIDINE	EFFICACY TOXICITY	Normal metabolizer. The genetic markers that have been evaluated do not increase risk for drug-induced side effects or toxicity.

CARDIOLOGY

MEDICATIONS WITH RECOMMENDATIONS BASED ON CPIC GUIDELINES

SIMVASTATIN

DRUG TYPE	CARDIOLOGY : STATINS
COMMON BRANDS	ZOCOR
GENE	SLCO1B1
GENOTYPE	AA
INFERENCE	Baseline risk for drug induced toxicity. 
GENETIC INTERPRETATION	This indicates that you may not be genetically susceptible for developing drug induced toxicity and you may be given the regular therapy. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	Genetic elements significantly determine the response of patients to the drug. People with specific marker in their genotype are unable to metabolize the drug efficiently. Hence, the drug is accumulated in the muscles leading to muscle toxicity or myopathy. SLCO1B1 gene encodes the protein OATP1B1, which is associated with hepatic uptake of the drug. Variation in gene sequence of SLCO1B1 impairs the normal function of the encoded protein, altering the plasma concentration of statins and increasing related muscle damage.
CPIC RECOMMENDATIONS **	CPIC guidelines recommend standard dose of simvastatin for individuals who do not carry the SLCO1B1 variant allele.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.
**For medical practitioners only.

ADDITIONAL INFORMATION

PRESCRIBED AS	A lipid lowering drug recommended for prevention of coronary heart disease.
DRUG INFORMATION	Statins are lipid lowering drugs recommended for the prevention of primary and secondary cardiovascular disease. Concomitant administration of the drug along with regular diet and exercise have found to reduce levels of LDL cholesterol (Low Density Lipoprotein) or bad cholesterol and also increase the level of HDL cholesterol (High Density Lipoprotein) or good cholesterol in the body.

ADVERSE EFFECTS	Statins have wide therapeutic index and are safe in general. Some of these adverse reactions could be non-serious and reversible, while rare cases could also be fatal. Most common adverse effects include muscle toxicity or myopathy and rare cases of rhabdomyolysis (degeneration of muscles with acute kidney injury).

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

CLOPIDOGREL

DRUG TYPE	CARDIOLOGY : ANTIPLATELETS
COMMON BRANDS	PLAVIX, TORPLATT, THINRIN, THEMIGRIL, STROMIX, PSYGREL, PREVA, STARCLOP, PLATLOC, PLAGRIL, PLAGERINE, NOPLAQ, ORAWIS, NOKLOT, NUGREL, CLOPID, CLOPILET,CERUVIN, CLAVIX, APTOGREL
GENE	CYP2C19
GENOTYPE	*1/*17
INFERENCE	Ultrarapid metabolizer. Typical likelihood for positive clinical outcome, on standard dose. 
GENETIC INTERPRETATION	This indicates that the drug will be metabolized quickly and standard dose may be adequate. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	Genetic factors significantly determine response of the patient to the drug. Clopidogrel undergoes bioactivation within the body, to form an active metabolite which interacts with blood platelets. Several enzymes are involved in this transformation. Variation in genetic sequence of these enzymes affects the enzyme function.This alters pharmacokinetics of the drug, leading to ineffective response in some people or can even lead to major adverse cardiovascular events. The hepatic CYP2C19 is one such enzyme involved in bioactivation of the drug. Several variants of CYP2C19 genetic sequence have been reported to correlate with drug efficacy in patients. Some variants impair the functional domain of the enzyme thereby inhibiting the interaction with platelets. On the other hand, variants reported in the regulatory region of the gene are known to enhance the function of the enzyme.
CPIC RECOMMENDATIONS **	In case of CYP2C19 ultrarapid metabolizers, CPIC guidelines recommend standard therapy and dosing for clopidogrel.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.
**For medical practitioners only.

ADDITIONAL INFORMATION

PRESCRIBED AS	An anti-platelet drug, inhibiting platelet aggregation and preventing blood clot formation in the body.
DRUG INFORMATION	Clopidogrel is an anti-platelet drug that inhibits platelet aggregation and thereby prevents blood clot formation in the body. It hinders narrowing of blood vessels and maintains easy flow of blood in the body. It decreases the risk of heart disorders and strokes primarily in patients who have incurred a cardiovascular attack.

ADVERSE EFFECTS	Fatal adverse effects from the therapy are very rare. Most frequent mild reactions include nausea, diarrhea, fever, bleeding or bruising and skin rashes. Occasional side effects may include bleeding in stomach and brain or variations in blood pressure levels. Major adverse cardiovascular events and high risks of stent thrombosis are reported as few rare life threatening events associated with the therapy.

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

WARFARIN

DRUG TYPE	CARDIOLOGY : ANTICOAGULANTS
COMMON BRANDS	COUMADIN
GENE	CYP2C9
GENOTYPE	*1/*1
INFERENCE	Baseline risk for drug sensitivity. The genetic markers that have been evaluated do not increase risk for warfarin-induced bleeding. 
GENETIC INTERPRETATION	This indicates that you may not be genetically sensitive for the drug therapy and you may be given the regular warfarin dose. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	Genetic factors constitute a primary component that determine the optimal dosage of warfarin therapy. Liver condition and drug-drug interaction are very crucial factors in deciding the dosage along with other preliminary factors like age, sex and race of an individual. CYP2C9 and CYP4F2 enzymes catalyze warfarin drug metabolism in the liver. Variation in gene sequence of the two enzymes impairs the enzyme function. Hence, the regular drug metabolism is hampered. VKORC1 protein, the target molecule for warfarin, is associated with the conversion of vitamin K epoxide to vitamin K. Variation in regulatory region of VKORC1 significantly lowers the protein production, thereby affecting the regular pharmacodynamics of the drug.
CPIC RECOMMENDATIONS **	CPIC guidelines recommend starting the therapy on standard drug dose of warfarin.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.

**For medical practitioners only.

ADDITIONAL INFORMATION

PRESCRIBED AS	An anticoagulant drug to treat heart attack and stroke.
DRUG INFORMATION	Warfarin is the most widely recommended anticoagulant (blood thinner) used to treat thrombotic disorders (formation of thrombus or blood clot in blood vessel). It inhibits the formation of clotting factors thus reducing blood clots in arteries and veins, of the body. It is generally prescribed in conditions of arrhythmia, myocardial infarction and stroke.

ADVERSE EFFECTS	The medication is safe in general; adverse events are reported occasionally. Most common side effects include nausea, vomiting, diarrhea, fatigue, loss of appetite, skin rashes, wheezing and difficulty in breathing, and abdominal pain. Medical cases of fatal bleeding or bruising are reported. Blood in urine and stool, nosebleeds and bleeding gums are associated with overdose of the drug.

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

DIABETOLOGY

NOVEL PHARMACOGENIC ASSOCIATIONS

GLIMEPERIDE

DRUG TYPE	DIABETOLOGY : SULFONYLUREA ANTIDIABETICS
COMMON BRANDS	GLISTA, GLUCONORM-PG, GLUCORYL, GLYPRIDE, GLYFIX, GRIDE, MYPRIDE, SULFOGLIM, TRIGULIN, ZORYL, GLIMPID, GLIMSITE, GLIMULIN, GLIMY, GLAMOR, GLADOR, GLIMCIP, GLIMER, DIBIGLIM, EUGLIM, FLEXIGLIM, AMARYL, BETAGLIM, BLISTO, CAPRIL, DIAGLIM, AVAGLIM
GENE	G6PD
GENOTYPE	GG_AA_GG_GG
INFERENCE	Baseline risk for drug-induced hemolysis. 
GENETIC INTERPRETATION	According to your genotypes, the genetic markers that have been evaluated do not increase risk for drug-induced hemolysis. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	G6PD (Glucose-6 Phosphate Dehydrogenase) deficiency, or favism, is an inherited condition caused by mutations in the G6PD gene on chromosome X. The G6PD enzyme maintains the level of NADPH in red blood cells, for protection against oxidative stress. In case of G6PD deficiency, the red blood cells are depleted of NADPH which triggers a series of chemical reactions that oxidize hemoglobin to a denatured form of the protein. This results in hemolysis, i.e., destruction of red blood cells, both in intra- and extravascular domains. Individuals carrying G6PD mutations are at risk for drug-induced AHA (acute hemolytic anaemia) and may need to monitored regularly, via complete blood count tests. The U.S Food and Drug Administration (FDA) has issued a warning regarding the potential hazard of administering certain drugs such as sulfonylureas (eg., glimeperide), antidepressants, antimalarials, etc.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.
Note: Hemizygous males and homozygous females may display more severe symptoms of G6PD deficiency, when compared with heterozygous females. Drug response may vary.

ADDITIONAL INFORMATION

PRESCRIBED AS	Antihyperglycemic agent used in the treatment of Type 2 Diabetes.
DRUG INFORMATION	Glimeperide is a second-generation sulfonylurea which acts as an insulin secretagogue. The drug binds to the potassium-channel receptors and depolarizes the cell membrane. This results in increased calcium ion influx, and subsequent secretion of insulin by the pancreatic cells.

ADVERSE EFFECTS	Hypoglycemia (less common when compared with glipizide and gluburide); Coma; Seizures; Beta-cell impairment

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

PSYCHIATRY

MEDICATIONS WITH RECOMMENDATIONS BASED ON CPIC GUIDELINES

AMITRIPTYLINE

DRUG TYPE	PSYCHIATRY: TRICYCLIC ANTIDEPRESSANTS
COMMON BRANDS	AMIT, AMITOR, ABITRIP, AMITRIP, ELIWIL, LIBOTRYP, ELAVIL, GOLDEP, RELIDEP
GENE	CYP2D6
GENOTYPE	II_GG_II_GG_GG
INFERENCE	Normal metabolizer. The genetic markers that have been evaluated do not increase risk for drug-induced side effects or toxicity. 
GENETIC INTERPRETATION	According to your genotypes, the drug may be metabolized normally and standard dose could be adequate. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	Tricyclic Antidepressants (TCAs) are metabolized by the CYP2D6 and CYP2C19 enzymes of the cytochrome p450 family of proteins. Poor metabolizers (reduced cytochrome enzyme activity) are at risk for adverse effects, due to elevated plasma drug concentrations. Ultra metabolizers (greater cytochrome enzyme activity) may experience failure in treatment (due to subtherapeutic drug concentrations). Genetic analysis of the CYP2D6 and CYP2C19 markers is used to determine an individual's metabolizer status and thereby, preferred range of dosing. However, there is limited data on combinatorial phenotypes (from CYP2D6 and CYP2C19 genotypes). Intermediate (1-13% of patients) and poor metabolizers (1-10% of patients) may be prescribed a lowered dose of the TCA. In some cases, an alternate antidepressant (not metabolized by CYP2D6 or CYP2C19) may be prescribed by the physician. In case of no alternative, stringent monitoring of patient's response to the TCA is essential, after a reduced dose of the same drug.
CPIC RECOMMENDATIONS**	In case of CYP2D6 normal metabolizers, CPIC guidelines recommend starting the therapy on standard drug dose.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.
Note: CYP2D6 *3, *4, *6, *10 and *41 alleles are included in the analysis
**For medical practitioners only.

ADDITIONAL INFORMATION

PRESCRIBED AS	Antidepressants prescribed for pain management during migraine (interval therapy), depression, obsessive-compulsive disorder (OCD).

DRUG INFORMATION	Amitriptyline is a tertiary amine Tricyclic Antidepressant. It targets serotonin and norepinephrine receptors in the presynaptic terminal, and inhibits chemical reuptake (dopamine levels are usually not affected by TCAs). This mechanism is responsible for modulating pain in migraine prophylaxis, and the management of psychiatric symptoms such as manic depression.
ADVERSE EFFECTS	TCAs are more likely to cause side effects, when compared with other classes of antidepressants. Their natural ability to bind to chemical agents like cholinergic, adrenergic and histamine receptors results in blocked neurotransmission. Adverse effects include Dizziness, postural hypotension, Glaucoma, and Tachyarrhythmia. TCAs have been reported to cause severe toxicity and near-lethal effects, in incidents of drug overdosing and poisoning.

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

NOVEL PHARMACOGENIC ASSOCIATIONS

NALTREXONE

DRUG TYPE	PSYCHIATRY : OPIOID ANTAGONISTS
COMMON BRANDS	NALTREX, NODICT, NALCON, NALTIMA, REVIA, CONTRAVE, VIVITROL, VIVITREX
GENE	OPRM1
GENOTYPE	AG
INFERENCE	Slightly high likelihood for positive drug response 
GENETIC INTERPRETATION	According to your genotypes, this drug may be activated to a greater extent and therapy could be more effective on you. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	A certain polymorphism in the Âµ-opioid receptor (OPRM1) gene enhances an individual's likelihood of successful therapy with naltrexone. Presence of the variant is associated with reduced cravings, lesser withdrawal symptoms and an increased period of abstinence. Asians have a greater frequency for this gene variant, when compared with other populations. The same variant is linked with alcoholism risk.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.

ADDITIONAL INFORMATION

PRESCRIBED AS	Opioid antagonist for the management of substance abuse disorders, e.g., alcoholism, narcotic addiction, etc.

DRUG INFORMATION	Naltrexone is primarily an opioid antagonist which helps reduce an individual's addiction to alcohol or opioids, by minimizing the reward sensation experienced when consuming these substances. The drug targets mu-opioid receptors, effectively blocking their association with regular agonists. Thus, the body does not respond to the opioid substance, due to competitive binding and reduced availability of opioid receptors in the brain. In the absence of euphoria, individuals have reduced cravings, and thus are less dependant on the consumption of narcotics/alcohol.
ADVERSE EFFECTS	Vomiting and stomach cramps; allergic reaction such as hives; anxiety.

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

RISPERIDONE

DRUG TYPE	PSYCHIATRY : ANTIPSYCHOTICS
COMMON BRANDS	RIZE, RESPIDON, RIDON, RISDONE, RISINA, RISNIA, RISPERDAL, RISPOND, ZISPER, ROZIDAL, RAGRACE, PERIDON, DON PLUS, GENREST, KRISP, R-DON, RISET, SIZODON, SPERIDON
GENE	CYP2D6
GENOTYPE	II_GG_II_GG_GG
INFERENCE	Normal metabolizer. The genetic markers that have been evaluated do not increase risk for drug-induced side effects or toxicity. 
GENETIC INTERPRETATION	According to your genotypes, the drug may be metabolized normally and standard dose could be adequate. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	The cytochrome p450 2D6 (CYP2D6) genotype is a strong predictor of potential antipsychotic-induced extrapyramidal symptoms such as tardive dyskinesia. Individuals who carry one or more non-functional CYP2D6 alleles (intermediate and poor metabolizers) metabolize the drug less efficiently, and may require a reduced drug dose. Individuals who carry CYP2D6 gene duplications (ultra metabolizers) rapidly metabolize the drug and may require an alternate drug (not metabolized by CYP2D6), due to treatment failure. A few variants in the COMT, DRD2 genes have also been studied for their role in antipsychotic response. However, these findings need to be validated by larger studies. The current FDA guidelines for antipsychotic drugs are based on CYP2D6 genotype.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.
Note: CYP2D6 *3, *4, *6, *10 and *41 alleles are included in the analysis

ADDITIONAL INFORMATION

PRESCRIBED AS	Risperidone is used to treat schizophrenia and symptoms of bipolar disorder. It is also used in autistic children to treat symptoms of irritability.

DRUG INFORMATION	Risperidone is a second-generation antipsychotic (atypical antipsychotic), which acts as serotonin and dopamine antagonist. Dopamine activity is higher in individuals on atypical antipsychotics. A study of drug effects in elderly patients administered with antipsychotics proved that atypical antipsychotic drugs are associated with a lower risk for certain side effects, due to a slightly peculiar mechanism. This is because dopamine neurotransmission is not compromised, which reduces the probability of motor and cognitive impairment.
ADVERSE EFFECTS	Commonly reported side effects include: agitation, akathisia, anxiety, constipation, dizziness, drowsiness, dystonia, extrapyramidal reaction, nausea, rhinitis, and weight gain. Other side effects include: abdominal pain, sialorrhea, skin rash, tachycardia, and xeroderma.

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

PERPHENAZINE

DRUG TYPE	PSYCHIATRY : ANTIPSYCHOTICS
COMMON BRANDS	TRILAFON, ETRAFON, DECENTAN
GENE	CYP2D6
GENOTYPE	II_GG_II_GG_GG
INFERENCE	Normal metabolizer. The genetic markers that have been evaluated do not increase risk for drug-induced side effects or toxicity. 
GENETIC INTERPRETATION	According to your genotypes, the drug may be metabolized normally and standard dose could be adequate. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	The cytochrome p450 2D6 (CYP2D6) genotype is a strong predictor of potential antipsychotic-induced extrapyramidal symptoms such as tardive dyskinesia. Individuals who carry one or more non-functional CYP2D6 alleles (intermediate and poor metabolizers) metabolize the drug less efficiently, and may require a reduced drug dose. Individuals who carry CYP2D6 gene duplications (ultra metabolizers) rapidly metabolize the drug and may require an alternate drug (not metabolized by CYP2D6), due to treatment failure. A few variants in the COMT, DRD2 genes have also been studied for their role in antipsychotic response. However, these findings need to be validated by larger studies. The current FDA guidelines for antipsychotic drugs are based on CYP2D6 genotype.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.
Note: CYP2D6 *3, *4, *6, *10 and *41 alleles are included in the analysis

ADDITIONAL INFORMATION

PRESCRIBED AS	Perphenazine is used to treat schizophrenia. It is also used to control severe nausea and vomiting.

DRUG INFORMATION	Perphenazine is a first-generation antipsychotic (typical antipsychotic) that acts as dopamine antagonist, by blocking the dopamine D2 receptors in the striatum, which receives inputs for the rest of the basal ganglia, via neurotransmission.
ADVERSE EFFECTS	Common adverse effects include: Extrapyramidal reactions (e.g., Parkinson-like symptoms, dystonia, akathisia, tardive dyskinesia), drowsiness, muscular weakness, dry mouth, blurred vision, weight gain, skin reactions, amenorrhea, galactorrhea.

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

THIORIDAZINE

DRUG TYPE	PSYCHIATRY : ANTIPSYCHOTICS
COMMON BRANDS	RIDAZIN, MELLERIL, THIOZID, THIORIL, ZENERIL
GENE	CYP2D6
GENOTYPE	II_GG_II_GG_GG
INFERENCE	Normal metabolizer. The genetic markers that have been evaluated do not increase risk for drug-induced side effects or toxicity. 
GENETIC INTERPRETATION	According to your genotypes, the drug may be metabolized normally and standard dose could be adequate. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	The cytochrome p450 2D6 (CYP2D6) genotype is a strong predictor of potential antipsychotic-induced extrapyramidal symptoms such as tardive dyskinesia. Individuals who carry one or more non-functional CYP2D6 alleles (intermediate and poor metabolizers) metabolize the drug less efficiently, and may require a reduced drug dose. Individuals who carry CYP2D6 gene duplications (ultra metabolizers) rapidly metabolize the drug and may require an alternate drug (not metabolized by CYP2D6), due to treatment failure. A few variants in the COMT, DRD2 genes have also been studied for their role in antipsychotic response. However, these findings need to be validated by larger studies. The current FDA guidelines for antipsychotic drugs are based on CYP2D6 genotype.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.

ADDITIONAL INFORMATION

PRESCRIBED AS	Thioridazine is used for treating schizophrenia in patients who have not shown improvement with or are unable to take other medicines.
DRUG INFORMATION	Thioridazine is a first-generation antipsychotic (typical antipsychotic) that acts as dopamine antagonist, by blocking the dopamine D2 receptors in the striatum, which receives inputs for the rest of the basal ganglia, via neurotransmission.

ADVERSE EFFECTS	Commonly reported adverse effects include: syncope, akathisia, constipation, dizziness, drowsiness, epithelial keratopathy, increased serum prolactin, nasal congestion, retinitis pigmentosa, sedation, star-shaped cataract, urinary retention, visual disturbance, hypohidrosis, and xerostomia.

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

DIAZEPAM

DRUG TYPE	PSYCHIATRY : ANXIOLYTICS
COMMON BRANDS	VALIUM
GENE	CYP2C19
GENOTYPE	*1/*17
INFERENCE	Normal metabolizer. Baseline risk for drug-induced side effects. 
GENETIC INTERPRETATION	According to your genotypes, you do not have the variants for poor drug metabolism and hence may not be at risk for adverse effects, on standard dose. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	Diazepam is primarily metabolized via the cytochrome P450 pathway, by the CYP2C19 enzyme. Genetic variation in CYP2C19 affects the in vivo metabolism of the drug, which alters the plasma concentrations of the drug and therapeutic outcome. An individual can be classified into extensive metabolizer (EM), intermediate metabolizer (IM) or poor metabolizer (PM), based on his/her CYP2C19 genotype. Intermediate and poor metabolizers may display reduced enzyme activity, and an increased exposure to the drug, due to greater concentration in the plasma. Such individuals may require a reduced dose of the drug, to avoid side effects. One study showed that when CYP2C19 poor metabolizers were administered diazepam to relieve preoperative anxiety, they emerged from general anesthesia after a longer period of time, when compared with extensive metabolizers. CYP2C19 poor metabolizers may also be at risk for clobazam-induced side effects.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.

ADDITIONAL INFORMATION

PRESCRIBED AS	Major depressive disorder, muscle spasms/seizures.
DRUG INFORMATION	Diazepam is a benzodiazepine drug, which exerts anxiolytic and antiepileptic effects. It binds to the gamma-aminobutyric acid type A receptor complex (GABA), at a specific site between the alpha and gamma subunits. This results in hyperpolarization, due to increased inflow of chloride ions in the neurons. Therapeutic effect is achieved by producing a calming effect in the CNS (Central Nervous System), which can alleviate symptoms such as seizures.
ADVERSE EFFECTS	Common side effects include dry mouth, rashes (hives), nausea, vomiting, dizziness and diarrhoea. Reduced sexual drive has also been reported.

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

NEUROLOGY

MEDICATIONS WITH RECOMMENDATIONS BASED ON CPIC GUIDELINES

PHENYTOIN

DRUG TYPE	NEUROLOGY : ANTICONVULSANTS
COMMON BRANDS	BARBITOIN, EPICURE, EPIPHEN, DILATIN, EPICENT, EPILEPTIN, EPISOL, EPITAB, EPTOIN, EPILAN, FENTOIN, NEPTOIN, PHENYKEM, PHETOIN, POLYTOIN, SOLPHEN, STOIN TAB, EQUIL, PHENBARB, PHENYTAL, POLYTOIN, CONVUL, DANTEN, DANTOINE, DIFETOIN, HYDANTOIN, LEPITOIN, SANEPIL, TOIN
GENE	FLOT1
GENOTYPE	HLA-B*15:02 Negative
INFERENCE	Baseline risk for drug-induced toxicity. 
GENETIC INTERPRETATION	According to your genotype, the genetic markers that have been evaluated do not increase risk for drug-induced toxicity. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	Individuals who carry the HLA-B*1502 allele are at risk for Severe Cutaneous Adverse Reaction (SCAR) syndrome and may require reduced doses of phenytoin, due to its potential for toxicity. HLA-B gene encodes for heavy chain of MHC class-1 molecule, presenting antigenic peptides to body's immune system. Variation in the HLA complex at specific loci HLA-B*1502 leads to inappropriate presentation of antigens to T-Cell receptors, thus triggering an adverse reaction, in the body. The risk allele frequency of HLA-B varies significantly across geographical distribution. The HLA-B*1502 ancestry has been significantly reported across borders of Asia, including South Asians, primarily Indians. The frequency of risk allele in Indians is around 2-4% or even higher. Southeast Asian countries such as Malaysia, Indonesia, Philippines, Taiwan and parts of China have the highest frequency rate of up to 12%. Genetic pre-screening of HLA variant is recommended by the United States FDA before starting the drug therapy in patients. This involves identifying the corresponding tag SNPs for HLA variants. Patients testing positive for the risk allele are not given the drug, unless the benefit of therapy significantly outweighs the risk of adverse reaction.
CPIC RECOMMENDATIONS**	According to CPIC guidelines, standard dosing of phenytoin may be administered, in case of a negative genetic test for HLA-B*15:02 markers.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.
**For medical practitioners only.

ADDITIONAL INFORMATION

PRESCRIBED AS	Anticonvulsant, antiarrhythmic agent used for the treatment of generalized and complex (psychomotor, temporal lobe) seizures.
DRUG INFORMATION	Phenytoin is a hydantoin derivative which preferentially binds SCN2A (sodium voltage gated channel alpha subunit 2), for its antiepileptic effect. By increasing sodium ion efflux, the drug regulates electrical conductance and helps control seizure mechanisms in the motor cortex.
ADVERSE EFFECTS	Gingival hyperplasia; Coma; Loss of muscle control; Severe Cutaneous Adverse Reaction (SCAR) syndrome due to genetic mutations

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

NOVEL PHARMACOGENIC ASSOCIATIONS

VALPROIC ACID

DRUG TYPE	DEPAKENE, DEPAKOTE, STAVZOR, DEVELPIN, DIVUNA, DEVOT, EPILEX, EPSOVAL, PROVALEX, NAVALIN, TORVATE, VALCOT, VALPARIN, VALPORATE, VALTEC, ENCORATE, EPIVAL, MANOVAL, VALKID, VALPORIL, VALTRIL, ZORAT, CONVULEX, SPRINKLE
COMMON BRANDS	DEPAKENE, DEPAKOTE, STAVZOR, DEVELPIN, DIVUNA, DEVOT, EPILEX, EPSOVAL, PROVALEX, NAVALIN, TORVATE, VALCOT, VALPARIN, VALPORATE, VALTEC, ENCORATE, EPIVAL, MANOVAL, VALKID, VALPORIL, VALTRIL, ZORAT, CONVULEX, SPRINKLE
GENE	POLG
GENOTYPE	GG_GG
INFERENCE	Baseline risk for drug-induced toxicity. The genetic markers that have been evaluated do not increase risk for drug-induced toxicity. 
GENETIC INTERPRETATION	According to your genotype, you do not have an elevated risk for drug-induced toxicity. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	The Ala467Thr and Trp748Ser mutations are observed in approximately 2/3 of patients with autosomal recessive POLG-related disorders such as Alpers Huttenlocher Syndrome. Valproic acid is contraindicated in patients with known urea cycle disorders (UCDs) and in patients with mutations in POLG gene. POLG gene mutations are associated with hereditary neurometabolic syndromes such as Alpers Huttenlocher Syndrome, and those patients are at an increased risk of liver failure. Individuals who carry the gene mutations may need reduced dose or close monitoring during therapy (if alternate drug is not available).

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.

ADDITIONAL INFORMATION

PRESCRIBED AS	Anticonvulsant used in the treatment of epilepsy, bipolar disorder, mania, migraine prophylaxis.

DRUG INFORMATION	Valproic acid is a fatty acid derivative which increases gamma-aminobutyric acid levels in the brain. It also alters the properties of voltage dependent sodium channels. According to some studies, valproic acid may be also be used in controlling HIV infection, due to its ability to inhibit histone deacetylase.
ADVERSE EFFECTS	Diarrhoea; Abdominal cramps; Nausea; Dizziness; Tremors; Irregular menses; Pruritus; Toxic epidermal necrolysis

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

GENERAL MEDICINE

NOVEL PHARMACOGENIC ASSOCIATIONS

CELECOXIB

DRUG TYPE	GENERAL MEDICINE : NON STEROIDAL ANTI INFLAMMATORY DRUGS
COMMON BRANDS	COXIB, COLCIBRA, ICEL, J FLEX, REVIBRA, ZECOXIB, ZYCEL, COBIX, CELIB, CELEXAX, CELETOP, CELCOX, CELACT, CE, CELEMAX, SIONARA, CELEBREX
GENE	CYP2C9
GENOTYPE	*1/*1
INFERENCE	Baseline risk for drug sensitivity. The genetic markers that have been evaluated do not increase risk for drug-induced side effects or toxicity. 
GENETIC INTERPRETATION	This indicates that you may not be genetically sensitive for the drug therapy and standard dose may be effective. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	Genetic variation in CYP2C9 affects the in vivo metabolism of NSAIDs, which alters the plasma concentrations of the drug and therapeutic outcome. An individual can be classified into extensive metabolizer(EM), intermediate metabolizer(IM) or poor metabolizer (PM), based on his/her CYP2C9 genotype. Intermediate and poor metabolizers may display reduced enzyme activity, and an increased exposure to the drug. This can lead to Upper GastroIntestinal Bleeding (UGIB). Poor metabolizers (individuals who carry two risk variants) may be at 2-fold greater risk for NSAID-induced UGIB, when compared with extensive metabolizers. Such individuals may require a reduced dose of the drug.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.

ADDITIONAL INFORMATION

PRESCRIBED AS	Non Steroidal Anti Inflammatory Drug (NSAIDs) used for pain management in rheumatoid arthritis, osteoarthritis, ankylosing spondylitis, menstrual syndromes (dysmenorrhea), and in the treatment of juvenile rheumatoid arthritis, in children >2 years of age.
DRUG INFORMATION	Celecoxib is a selective cyclooxygenase (COX)-2 inhibitor, which is used as an analgesic and antipyretic, because of its anti-inflammatory role. The drug compound blocks COX-2 enzyme activity, which results in reduced prostaglandin synthesis. This mechanism is responsible for alleviating symptoms triggered by the excess cytokines produced during inflammatory response, joint pain, fever, etc. This drug does not affect COX-1 activity, and hence projects lesser risk for side effects, unlike opioid analgesics.

ADVERSE EFFECTS	Diarrhoea, abdominal pain, headache, fatigue, elevated blood pressure, fluid retention - especially in the hands and feet (e.g., swelling of the ankles), urinary infection, difficulty in swallowing, severe gastrointestinal bleeding (in case of genetic factors)

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

TRAMADOL

DRUG TYPE	GENERAL MEDICINE : OPIOIDS
COMMON BRANDS	ACMEDOL, CALMPAIN, DOLOTRAM, DOMADOL, PAINADOL, POSTADOL, RAMAX, TRAMACIP, TRAMAZAC, TRAMBAX, TRANDOL, TRANZAT, TRUMP, ULTRAM, ZAMADOL, VICTADOL, ZYTRAM, ACUPAIN, OPI OT, TRAMA, TRAMP TAB, RALIVIA, ZYDOL
GENE	CYP2D6
GENOTYPE	II_GG_II_GG_GG
INFERENCE	Normal metabolizer. The genetic markers that have been evaluated do not increase risk for drug-induced side effects or toxicity. 
GENETIC INTERPRETATION	According to your genotypes, the drug may be metabolized normally and standard dose could be adequate. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	Genetic variation in CYP2D6 affects the in vivo bioactivation of tramadol (and many other opioid analgesics), which affects pain relief and therapeutic outcome. An individual can be classified into ultra metabolizer (UM), extensive metabolizer (EM), intermediate metabolizer (IM), or poor metabolizer (PM), based on the CYP2D6 genotype. Intermediate and poor metabolizers may experience insufficient pain relief. Such individuals may require alternate therapy, via a non-opioid analgesic such as morphine itself.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.
Note: CYP2D6 *3, *4, *6, *10 and *41 alleles are included in the analysis

ADDITIONAL INFORMATION

PRESCRIBED AS	Synthetic opioid used in the management of moderate to severe postoperative pain, dental pain, musculoskeletal injury, and as an adjuvant for osteoarthritis.
DRUG INFORMATION	Tramadol is a synthetic analogue of codeine, with a different mechanism of action. The drug acts centrally, as an opioid analgesic via a dual mechanism - it activates mu-opioid receptors (lower affinity than that of codeine) and additionally, inhibits serotonin reuptake. This blocks the transmission of pain sensations in the spinal cord.
ADVERSE EFFECTS	Drowsiness; lightheadedness; nausea, vomiting, stomach cramps; sedation

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

PEG3350

DRUG TYPE	LAXATIVE
COMMON BRANDS	MOVIPREP

GENE	G6PD
GENOTYPE	GG_AA_GG_GG
INFERENCE	Baseline risk for drug-induced hemolysis. 
GENETIC INTERPRETATION	According to your genotypes, the genetic markers that have been evaluated do not increase risk for drug-induced hemolysis. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	G6PD (Glucose-6 Phosphate Dehydrogenase) deficiency, or favism, is an inherited condition caused by mutations in the G6PD gene on chromosome X. The G6PD enzyme maintains the level of NADPH in red blood cells, for protection against oxidative stress. In case of G6PD deficiency, the red blood cells are depleted of NADPH which triggers a series of chemical reactions that oxidize hemoglobin to a denatured form of the protein. This results in hemolysis, i.e., destruction of red blood cells, both in intra- and extravascular domains. Individuals carrying G6PD mutations are at risk for drug-induced AHA (acute hemolytic anaemia) and may need to monitored regularly, via complete blood count tests. The U.S Food and Drug Administration (FDA) has issued a warning regarding the potential hazard of administering certain drugs such as moviprep, antidepressants, antimalarials, sulfonylureas, etc.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.

Note: Hemizygous males and homozygous females may display more severe symptoms of G6PD deficiency, when compared with heterozygous females. Drug response may vary.

ADDITIONAL INFORMATION

PRESCRIBED AS	Laxative which is used for colon cleansing prior to medical procedures such as colonoscopy.
DRUG INFORMATION	The combination of PEG-3350, sodium sulphate, sodium chloride, potassium chloride, sodium ascorbate and ascorbic acid (Vitamin C) is used as a colon cleanser and sold under the name Moviprep. The drug solution helps in gastric emptying, to get rid of fecal debris. It also carries electrolytes to replenish lost minerals.
ADVERSE EFFECTS	Nausea; Vomiting; Bloating; Stomach cramps

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

ONCOLOGY

MEDICATIONS WITH RECOMMENDATIONS BASED ON CPIC GUIDELINES

THIOPURINES

DRUG TYPE	ONCOLOGY : IMMUNOSUPPRESSANTS
COMMON BRANDS	PURINETHOL
GENE	TPMT
GENOTYPE	*1/*1
INFERENCE	High enzyme activity. Baseline risk for drug induced toxicity. 
GENETIC INTERPRETATION	This indicates that you may not be genetically susceptible to drug toxicity. Hence the therapy may be recommended for you. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	Thiopurines are administered as pro-drugs and further require biological activation to exert cytotoxic action. Azathioprine is reduced to 6MP, non-enzymatically. Both 6MP and 6TG are sequentially metabolized to form the final active cytotoxic compound ThioGuanine Nucleotide (TGN). Several enzymes catalyze the process. The cytotoxic TGN then is incorporated into DNA and /or RNA, causing strand breaks and strand cross-links, destroying cancer cells. However, excess cytotoxic activity of the drug is regulated by the enzyme Thiopurine S-MethylTransferase (TPMT). The enzyme catalyzes deactivation of the drug through S- methylation reaction. Genetic variations of TPMT loci drastically affect the enzyme activity. As a result, the drug is poorly metabolized. If given the normal dose of drug in such people, it leads to enhanced cytotoxicity due to accumulation of unmetabolized drug in the body. TPMT deficiency is an autosomal recessive trait and primarily determines inter-individual differences in drug response. Its association with drug toxicity has gained notable significance. Like many other new therapeutic drugs, prolonged treatment course of thiopurines is associated with drug induced liver injury (Hepatotoxicity). Furthermore, the drug causes characteristic myelosuppression in TPMT deficient people. There are occasional cases of serious and fatal myelosuppression caused by the drug. Around 1% of population has the drug susceptible homozygous risk genotype and around 7% of people inherit the heterozygous genotype. Rest of the fraction constitutes normal wild type gene.
CPIC RECOMMENDATIONS**	CPIC guidelines recommend starting the therapy on standard drug dose, with 2 weeks between dose adjustments.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.
**For medical practitioners only.

ADDITIONAL INFORMATION

PRESCRIBED AS	A chemotherapeutic drug in treatment of cancers or as immunosuppressant drug in treating auto-immune disorders.
DRUG INFORMATION	They are a class of cytotoxic drugs prescribed to treat several health conditions. They function as antimetabolites for cancers (leukemia, lymphoma) and immunosuppressants for auto immune diseases (rheumatoid arthritis) or inflammatory diseases (Crohn's disease, ulcerative colitis). They are also widely used in transplantation medicine (kidney). Biochemically the drug is a purine analog and is more reactive than regular purine bases. It is incorporated into DNA and /or RNA causing strand breaks and cross-links. Azathioprine, 6-mercatopurine, 6-thioguanine are examples of thiopurine drugs.
ADVERSE EFFECTS	The adverse reactions of the therapy and its severity may vary from person to person. Some common and mild side effects include nausea, vomiting, diarrhea, skin reactions, hair loss, loss of appetite, flu-like symptoms, fever, and chills. Occasional life threatening adverse events are reported in susceptible patients. Drug induced hepatotoxicity (liver toxicity) and myelosuppression (bone marrow suppression) are few of the kind observed and reported. Bone marrow suppression leads to significantly decreased count of blood cells. This causes anaemia and weakens immunity in patients further, triggering several other infectious diseases.

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

NOVEL PHARMACOGENIC ASSOCIATIONS

CISPLATIN

DRUG TYPE	ONCOLOGY : PLATINUM-BASED CHEMOTHERAPEUTICS
COMMON BRANDS	TEVAPLATIN, CISTRIZ, PLATIN, NEOPLAT, PLATICIS, KEMOPLAT, ONCOPLATIN, CISPLATIN, CISPLAN, PLATIKEM, BLASTOLEM, PLATINOL, CADIPLAT, CARBOKEM, CARBOPLAN, CYTOCARB, CYTOPLATIN, UNIPLATIN, PAMIFECT, PLASTIN, ABIPLATIN
GENE	TPMT
GENOTYPE	*1/*1
INFERENCE	High enzyme activity. Baseline risk for drug induced toxicity. 
GENETIC INTERPRETATION	This indicates that you may not be genetically susceptible to drug toxicity. Hence the therapy may be recommended for you. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.

GENETIC SIGNIFICANCE	Cisplatin-induced ototoxicity has been reported in children and adults on chemotherapy. Studies have deduced that in the presence of thiopurine S-methyltransferase (TPMT) mutations, the products of drug-DNA crosslinking (cisplatin-purines) may not be inactivated and hence, buildup in the cells. This leads to hearing loss, or ototoxicity. The U.S Food and Drug Administration (FDA) has acknowledged the possibility of TPMT-deficient individuals being at risk for drug-induced toxicity. However, more substantial evidence is required for a direct correlation between drug dose, genotype and clinical outcome.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.

ADDITIONAL INFORMATION

PRESCRIBED AS	Anticancer drug in the chemotherapy for solid tumours for a wide range of cancers including ovarian, cervical, testicular, lung, head, neck and bladder cancer. Specialist drug for pediatric cancers such as neuroblastoma, osteosarcoma and hepatosarcoma.
DRUG INFORMATION	Cisplatin is a platinum-based chemotherapy drug which exerts antineoplastic activity. The drug binds to the nuclear DNA of cancerous cells, to form stable adducts (DNA-platination). Thus, its crosslinking mechanism disrupts the progression of cancer by arresting the cell cycle and division, which leads to apoptosis, i.e., programmed cell death.
ADVERSE EFFECTS	Temporary hair loss; ringing in ears, reduced hearing; nausea and vomiting (persistent); loss of appetite; irreversible hearing loss, i.e., ototoxicity due to excessive free radicals.

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

DABRAFENIB

DRUG TYPE	ONCOLOGY : KINASE INHIBITOR CHEMOTHERAPEUTICS
COMMON BRANDS	TAFINLAR
GENE	G6PD
GENOTYPE	GG_AA_GG_GG
INFERENCE	Baseline risk for drug-induced hemolysis. 
GENETIC INTERPRETATION	According to your genotypes, the genetic markers that have been evaluated do not increase risk for drug-induced hemolysis. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	Dabrafenib can trigger hemolysis in melanoma patients who are positive for glucose-6-phosphate dehydrogenase (G6PD) mutations. These individuals are at risk for drug-induced AHA (acute hemolytic anaemia) and may need to monitored regularly, via complete blood count tests. The U.S Food and Drug Administration (FDA) has issued a warning regarding the potential hazard of administering certain drugs such as antidepressants, antimalarials, dabrafenib, etc.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.

Note: Hemizygous males and homozygous females may display more severe symptoms of G6PD deficiency, when compared with heterozygous females. Drug response may vary.

ADDITIONAL INFORMATION

PRESCRIBED AS	Anticancer drug used in the treatment of metastatic melanoma, for BRAF V600E-mutation positive cases.
DRUG INFORMATION	Dabrafenib is a protein kinase inhibitor which targets the tumour-specific mutation BRAF V600E, seen in around eighty percent of melanoma patients. BRAF is an protooncogene which is a key regulator of the RAS/MAPK pathway. This pathway carries out important events such as cellular division, differentiation, migration and apoptosis. Mutant cells display uncontrolled cellular proliferation, i.e., cancer. Dabrafenib targets the MAPK pathway, through selective inhibition of the BRAF mutations in the melanoma cells. Thus, it effectively reduces the size of metastatic tumours.
ADVERSE EFFECTS	Dabrafenib adverse effects appear here

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

LAPATINIB

DRUG TYPE	ONCOLOGY : KINASE INHIBITOR CHEMOTHERAPEUTICS
COMMON BRANDS	TYLERB, TYVERB, TYCERB
GENE	HLA-DRB1
GENOTYPE	HLA-DRB1*0701 Negative
INFERENCE	Baseline risk for drug-induced toxicity. The genetic markers that have been evaluated do not increase risk for drug-induced toxicity. 
GENETIC INTERPRETATION	According to your genotype, you do not have an elevated risk for drug-induced toxicity. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	In vitro studies and genomic association studies have identified that the MHC Class II allele HLA-DRB1*07:01 is a strong risk factor for lapatinib-induced liver injury. The United States Food and Drug Administration (FDA) has printed a warning about the potential hazard of drug sensitivity in individuals who carry the HLA-DRB1*07:01 allele.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.

ADDITIONAL INFORMATION

PRESCRIBED AS	Anticancer agent used in combination with other drugs for the treatment of HER-2 positive metastatic breast cancer.
DRUG INFORMATION	Lapatinib is a 4-anilinoquinazoline kinase inhibitor which effectively blocks the action of both the epidermal growth factor receptor (HER1/EGFR/ERBB1) and human epidermal growth factor receptor type 2 (HER2/ERBB2). Tumor cell growth is stopped through this dual mechanism.

ADVERSE EFFECTS	Hair loss; Hand-foot syndrome; Nausea, diarrhoea; Liver toxicity; Neutropenia; Anaemia

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

GASTROENTEROLOGY

NOVEL PHARMACOGENIC ASSOCIATIONS

OMEPRAZOLE

DRUG TYPE	GASTROENTEROLOGY : PROTON PUMP INHIBITORS
COMMON BRANDS	OMEZ, OMEPREN, OMIZAC, PROCID, PROTOLOC, SKYZOLE, ULZOL, ULCATON, ZOLSEC, ZECID, ZOLOP, ZOSEC, ORAZ, OMEPRAZ, NOGACID, OCID, LOMAC, COZEP, ACICHEK, AXFIRE, BIOCID, ACITEC, ACIZOLE, C-PRAZ, CAPCID, COZ, GASIPRA, GASOC, PRILOSEC, LORESS, M-ZOLE, LUPOME, MYSET-O, OLIT, OMALCER, OMECER, OMECIP, OMEE, ZOLCER, RESEC, ROMECID, PIRAZOLE
GENE	CYP2C19
GENOTYPE	*1/*17
INFERENCE	Ultrarapid metabolizer. The genetic markers that have been evaluated increase the likelihood for therapy failure. Higher dose or alternate drug may be required 
GENETIC INTERPRETATION	According to your genotype, the drug may be metabolized to a greater extent, and clinical response may be low, due to subtherapeutic drug concentrations. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	Omeprazole is metabolized by the CYP2C19 enzyme of the cytochrome p450 family of proteins. Ultrarapid metabolizers (greater cytochrome enzyme activity) may experience failure in treatment (due to subtherapeutic drug concentrations). Intermediate and poor metabolizers (reduced cytochrome enzyme activity) display greater plasma drug concentrations and may experience successful therapy.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.

ADDITIONAL INFORMATION

PRESCRIBED AS	Proton pump inhibitor used in the treatment of heartburn, acid reflux, symptoms of Gastro Esophageal Reflux Disease (GERD) and erosive esophagitis.
DRUG INFORMATION	Omeprazole stops the production of acid in parietal cells, by blocking the H+/K+ATPase proton pump, via irreversible binding to the H+/K+ATPase.
ADVERSE EFFECTS	Generally safe, with minimal adversities. Common side effects include abdominal pain, diarrhoea, flatulence, nausea and vomiting, pharyngitis and poor nutrient absorption (in case of prolonged use).

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

INFECTIOUS DISEASE

MEDICATIONS WITH RECOMMENDATIONS BASED ON CPIC GUIDELINES

ABACAVIR

DRUG TYPE	INFECTIOUS DISEASE : ANTIVIRALS
COMMON BRANDS	ZIAGEN
GENE	HLA-B*5701
GENOTYPE	AA
INFERENCE	Baseline risk for drug induced toxicity. 
GENETIC INTERPRETATION	This indicates that you may not be genetically susceptible for developing drug induced toxicity and you may be given the regular therapy. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	Genetic factor of an individual significantly determines the drug response. HLA-B gene that encodes for the heavy chain of MHC class-1 molecule plays a crucial role in presenting the antigenic peptides to the body's immune system. Variation in gene sequence of HLA-B triggers severe HSR in the mutants (HLA-B*5701) of the gene. Southwest Asians, particularly Indians have the highest prevalence rate of HLA-B*5701 of up to 20% of population, whereas Africans have the least prevalence rate. Up to 8% of patients taking abacavir experience hypersensitive reaction if genetic pre- screening is not performed. It is important to understand that HIV infection by itself triggers a wide range of symptoms and additionally the patient may be prescribed a combination therapy involving multiple drugs. Hence, the root cause of HSR may be obscured. Therefore, genetic pre-screening of HLA-B*5701 variant is recommended by the United States Food and Drug Administration (FDA) before starting or re-starting the drug therapy in patients. This involves identifying the corresponding tag marker for HLA-B*5701 variant, i.e., rs2395029 of HCP5.
CPIC RECOMMENDATIONS**	CPIC guidelines recommend standard dose of abacavir for individuals who do not carry the HLA *57:01 alleles.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.
**For medical practitioners only.

ADDITIONAL INFORMATION

PRESCRIBED AS	An antiviral drug given in combination with other antiretroviral drugs for the treatment of human immunodeficiency viral (HIV) infection.

DRUG INFORMATION	The drug is a synthetic nucleoside and is phosphorylated to form an analog of guanine. It inhibits the viral DNA synthesis and thereby combats the viral replication in human body. Abacavir is generally prescribed in combination with other drugs to treat HIV.
ADVERSE EFFECTS	The drug by large is safe. However, in some people it may trigger severe Hyper Sensitive Reaction (HSR), which is a multi-organ syndrome, characterized by fever, rashes on skin, headache, nausea, cough, sore throat, vomiting, diarrhea, breathing difficulties and abdominal pain. Gastrointestinal symptoms are reported in significant number of cases. Lactic acidosis in blood and abnormalities in liver and kidney are also reported.

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

RIBAVIRIN

DRUG TYPE	INFECTIOUS DISEASES : ANTIVIRALS
COMMON BRANDS	RIBAVIN, REBETOL, VIRAZIDE, RIBASPHERE, COPEGUS
GENE	IFNL3
GENOTYPE	AG
INFERENCE	Slightly low likelihood for for positive drug response 
GENETIC INTERPRETATION	Likely to have a reduced Sustained Virologic Response (SVR) and moderate drug efficacy.
GENETIC SIGNIFICANCE	A genetic variant near the interferon-lambda-3 (IFNL3) gene affects the Sustained Virological Response (SVR) achieved during antiviral therapy. The IFNL3 gene (also known as IL28B) has been studied in HCV patients of different ethnicities, and patient IFNL3 genotype has been proved to be a strong predictor of therapy duration and outcome. Individuals who carry the favourable IFNL3 variant are good responders and are likely to achieve SVR in a shorter time, when compared with non-carriers.
CPIC RECOMMENDATIONS**	According to CPIC guidelines, individuals who carry the AG or AA genotypes for rs12979860 (IFNL3) are less favourable candidates for ribavirin-containing regimes, when compared with GG carriers. There is a 60% chance for SVR after 24-48 weeks of treatment.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.
**For medical practitioners only.

ADDITIONAL INFORMATION

PRESCRIBED AS	Antiviral drugs for the treatment of HCV (hepatitis-C virus)-induced liver disease.
DRUG INFORMATION	Sofosbuvir is a nucleoside inhibitor which targets the HCV NS5B polymerase to stop viral replication. This drug, along with peginterferon and ribavirin (Triple Therapy), is one of the most successful treatment regimes, for all HCV genotypes, even those with drug resistance mutations.
ADVERSE EFFECTS	Common adverse effects include anaemia, headache and fatigue, nausea, diarrhoea, pruritus, rash. There may also be symptoms similar to flu.

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

NOVEL PHARMACOGENIC ASSOCIATIONS

EFAVIRENZ

DRUG TYPE	INFECTIOUS DISEASE : NON NUCLEOSIDE REVERSE TRANSCRIPTASE INHIBITORS (NNRTIs)
COMMON BRANDS	SUSTIVA, VIRADAY, VIROLIS, VONAVIR, ZIDOLAM, TRUSTIVA, TRIODAY, ODIVIR, TENOLA, LAMIVIR, LAZID, DUOVIR, EMDUO, CYTOCOM, EFAVIR, EFFERVEN, EFCURE, ESTIVA, VIRANZ, VIROCMB-E, STOCRIN, ZIDOLAM, REVENZ
GENE	CYP2B6
GENOTYPE	AA_CC
INFERENCE	Baseline risk for drug-induced toxicity. The genetic markers that have been evaluated do not increase risk for drug-induced toxicity. 
GENETIC INTERPRETATION	This indicates that you may not be genetically susceptible for developing drug induced toxicity and you may be given the regular therapy. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	Efavirenz is converted by the CYP2B6 enzyme to its main metabolite, 8-hydroxy efavirenz. Genetic variation in CYP2B6 significantly affects the rate of drug metabolism. The CYP2B6*6 haplotype (combined genotype from 2 non-synonymous SNPs) is linked with reduced CYP2B6 activity and drug clearance, and thereby, elevated efavirenz concentrations in the plasma (AUC), thus increasing the risk for drug toxicity. CYP2B6 poor metabolizers are at greater risk for insufficient clearance of the drug, which leads to neurotoxicity.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.

ADDITIONAL INFORMATION

PRESCRIBED AS	Antiretroviral agent used in combination therapy for Human Immunodeficiency Virus (HIV) infection.
DRUG INFORMATION	Efavirenz is a non nucleoside reverse transcriptase inhibitor which has virustatic activity, when metabolized to its active form. By blocking reverse transcriptase function and DNA synthesis, the activated drug plays a vital role in stopping further production of new virions. However, current guidelines recommend the use of efavirenz only in combination with other nucleoside inhibitors, and not as an independent drug.
ADVERSE EFFECTS	Neuropsychiatric manifestations - sleep disorders, hallucinations; Skin rash; Hepatitis; Dyslipidemia

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

DAPSONE

DRUG TYPE	INFECTIOUS DISEASE : ANTIBACTERIALS
COMMON BRANDS	DAPSONE, ACNESONE, ACZONE, UDOLAC, TARIMYL, SULFONA, AVLOSULFON
GENE	G6PD
GENOTYPE	GG_AA_GG_GG
INFERENCE	Baseline risk for drug-induced hemolysis. 
GENETIC INTERPRETATION	According to your genotypes, the genetic markers that have been evaluated do not increase risk for drug-induced hemolysis. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	G6PD (Glucose-6 Phosphate Dehydrogenase) deficiency, or favism, is an inherited condition caused by mutations in the G6PD gene on chromosome X. The G6PD enzyme maintains the level of NADPH in red blood cells, for protection against oxidative stress. In case of G6PD deficiency, the red blood cells are depleted of NADPH which triggers a series of chemical reactions that oxidize hemoglobin to a denatured form of the protein. This results in hemolysis, i.e., destruction of red blood cells, both in intra- and extravascular domains. Individuals carrying G6PD mutations are at risk for drug-induced AHA (acute hemolytic anaemia) and may need to monitored regularly, via complete blood count tests. The U.S Food and Drug Administration (FDA) has issued a warning regarding the potential hazard of administering certain drugs such as dapsone, sulfonylureas, antidepressants, antimalarials, etc.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.

Note: Hemizygous males and homozygous females may display more severe symptoms of G6PD deficiency, when compared with heterozygous females. Drug response may vary.

ADDITIONAL INFORMATION

PRESCRIBED AS	Antimicrobial used in the treatment of leprosy, dermatitis herpetiformis, dermatoses, acne vulgaris, malaria and prevention of AIDS recurrence.
DRUG INFORMATION	Dapsone is a synthetic sulfone which has antimycobacterial and antiprotozoal activity. It is one of the principal agents for the treatment of leprosy. It is also effective against bacteria such as streptococci, staphylococci and pneumococci. By mimicking sulfonamides and competitively binding to dihydropteroate synthetase (enzyme), the drug stops the synthesis of dihydrofolate by the microbes, thereby suppressing their growth. Topical dapsone is used in treating acne and other skin infections. The drug plays a dual role by exerting anti inflammatory effect.
ADVERSE EFFECTS	Abdominal pain, nausea, vomiting; Eruptions on the skin, hives; Neuropathy (less common); Drug-induced acute hemolytic anaemia due to genetic defects, i.e., G6PD deficiency

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

RIFAMPIN

DRUG TYPE	INFECTIOUS DISEASE : ANTIBACTERIALS
COMMON BRANDS	3 FD, 4 D, 4 D PLUS, AKURIT, ANACOX, ANTICOX, CAVICIN, CAVITER, MYCOCOX, MYCODOT, RIFACOM, RIFATER, RIMSTAR, RINIZID, TETRACOX, TIBIKIT, TRICOX, ACRIFA FORTE, CAVIRIP-1000, EUFAZID, GOCOX-3, MYCURIT-Z, RCINEX-Z, STANEX-3, TER-3
GENE	NAT2
GENOTYPE	GG_GG
INFERENCE	Slow acetylator, high risk for drug-induced toxicity. Reduced dose may be required. 
GENETIC INTERPRETATION	According to your genotypes, the drug may be metabolized very slowly and result in increased plasma concentrations. This can cause adverse reactions, due to toxicity. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	N-acetyl transferase 2 (NAT2) is an important enzyme involved in xenobiotic- and drug-related metabolic reactions. Individuals carrying at least two slow-acetylator variants in the NAT2 gene are classified slow acetylators, and may be exposed to elevated plasma drug concentrations, due to reduced drug clearance. Some studies have reported a link between slow acetylators and drug-induced adverse effects such as toxicity. On the other hand, fast or rapid acetylators are more likely to require higher drug doses due to possible therapy failure, due to rapid metabolism and subtherapeutic plasma concentrations, but this needs further validation.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.

ADDITIONAL INFORMATION

PRESCRIBED AS	Combination medicine used in the treatment of Mycobacterium Tuberculosis (TB) infection.
DRUG INFORMATION	Rifampin, isoniazid and pyrazinamide is a combination medicine used for treating TB. Rifampin is a bactericidal drug which blocks DNA-dependant RNA polymerase enzyme and subsequent RNA synthesis, thus killing the bacteria. Isoniazid exerts bactericidal effect on rapidly growing bacteria, and bacteriostatic effect on slow-growing bacteria. Upon activated by bacterial enzymes in vivo, the drug compound blocks the production of mycolic acid, which is an essential component of the bacterial cell wall. Pyrazinamide is a nicotinamide analog which inhibits synthesis of bacterial fatty acids, by blocking fatty acid saturase enzyme activity. These three compounds are used in combination
ADVERSE EFFECTS	Dark-coloured urine; Nausea, vomiting; Yellowing of eyes and skin due to liver problems

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

FLUCLOXACILLIN

DRUG TYPE	INFECTIOUS DISEASE : ANTIBIOTIC
COMMON BRANDS	FLOXAPEN
GENE	HLA-B*5701

GENOTYPE	AA
INFERENCE	Baseline risk for drug induced liver toxicity. 
GENETIC INTERPRETATION	This indicates that you may not be genetically susceptible to develop drug induced toxicity and you may be given the regular therapy. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	Genes involved in the metabolism of the drug in connection to its adverse reaction are not well known. However, MHC loci may share a crucial role in determining the drug response of an individual. Several genes like TNF, HSPA1L, HLA and ST6GAL1 may correlate with drug induced liver injury. The MHC loci HLA-B*5701 has a greater significance of association with drug toxicity. HLA-B*5701 is commonly detected by screening for its tag marker rs2395029. One out of 500-1000 people with HLA-B*5701 marker may have liver toxicity when treated with the drug. However, cases of liver toxicity with HLA-B*5701 are hardly seen in Asian countries.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.

ADDITIONAL INFORMATION

PRESCRIBED AS	An antibiotic drug for treating bacterial infections.
DRUG INFORMATION	Flucloxacillin is a penicillin class antibiotic commonly preferred to fight staphylococcal and other gram positive bacterial infections (streptococci, pneumococci). It is also effective against some strains of gram negative aerobic or anaerobic bacteria. It is generally prescribed to treat bacterial infections of the skin, urinary tract, respiratory tract, enteritis and other tissues.
ADVERSE EFFECTS	Stomach ache and diarrhoea are commonly reported mild reversible side effects. Uncommon effects could be skin rashes, jaundice and bruising of mucous membranes. Occasional cases of severe liver damage is associated with prolonged or repeated course of treatment. This may lead to notable yellowing of skin and eyes.

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

OTHERS

NOVEL PHARMACOGENIC ASSOCIATIONS

ELIGLUSTAT

DRUG TYPE	OTHERS
COMMON BRANDS	CERDELGA
GENE	CYP2D6
GENOTYPE	II_GG_II_GG_GG
INFERENCE	Normal metabolizer. The genetic markers that have been evaluated do not increase risk for drug-induced side effects or toxicity. 
GENETIC INTERPRETATION	According to your genotypes, the drug may be metabolized normally and standard dose could be adequate. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	The drug's efficacy and potential for causing toxicity are determined by the rate of metabolism and clearance of the active drug compound. Since CYP2D6 activity is decreased in the presence of certain genetic variants (some of the most important ones are *3,*4,*5,*6,*10 and *41), individual response to CERDELGA may vary. Poor CYP2D6 metabolizers (individuals who carry at least two no-activity alleles) are exposed to greater plasma drug concentrations, and are at risk for for drug-induced toxicity. The FDA label for this drug recommends a lower dose of the drug (84 mg once a day) for poor metabolizers, to avoid adverse effects.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.
Note: CYP2D6 *3, *4, *6, *10 and *41 alleles are included in the analysis

ADDITIONAL INFORMATION

PRESCRIBED AS	Enzyme inhibitor used in the substrate reduction therapy of Gaucher disease type I.
DRUG INFORMATION	Eliglustat is a ceramide analog and selectively inhibits the UDP-glucosylceramide synthase enzyme. This prevents the excess accumulation of glucosylceramide, the lipid substrate which builds up in Gaucher disease. Glucosylceramide is the main product of the enzymatic processing of UDP-glucose and ceramide. Eliglustat tartrate is the FDA approved drug for the treatment of the metabolic disease, Gaucher disease type 1.
ADVERSE EFFECTS	Diarrhoea, nausea; Pain in the back and extremities; Headache; Abdominal pain

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.

DEXTROMETHORPHAN & QUINIDINE

DRUG TYPE	Dextromethorphan hydrobromide and quinidine sulfate
COMMON BRANDS	NUEDEXTA
GENE	CYP2D6
GENOTYPE	II_GG_II_GG_GG
INFERENCE	Normal metabolizer. The genetic markers that have been evaluated do not increase risk for drug-induced side effects or toxicity. 
GENETIC INTERPRETATION	According to your genotypes, the drug may be metabolized normally and standard dose could be adequate. However, your physician shall decide on the drug and the dose depending on other clinical factors or medications you might be taking.
GENETIC SIGNIFICANCE	The drug's efficacy and potential for causing toxicity are determined by the rate of metabolism and clearance of the active drug compound. Since CYP2D6 activity is decreased in the presence of certain genetic variants (some of the most important ones are *3,*4,*5,*6,*10 and *41), individual response to NUEDEXTA may vary. Poor CYP2D6 metabolizers (individuals who carry at least two no-activity alleles) are exposed to greater plasma drug concentrations, and are at risk for for drug-induced toxicity. The FDA label for this drug mentions the benefit of genetic testing prior to beginning treatment, to avoid adverse effects in poor metabolizers.

Note: MedicaMap looks for the most common and already-proven genetic variants associated with the therapy and does not screen for rare markers affecting the drug response.
Note: CYP2D6 *3, *4, *6, *10 and *41 alleles are included in the analysis

ADDITIONAL INFORMATION

PRESCRIBED AS	Fixed-dose combination of Dextromethorphan hydrobromide and quinidine sulfate, used in the treatment of pseudobulbar affect (PBA) secondary to neurological disorders such as Alzheimer's, amyotrophic lateral sclerosis, Parkinson's, multiple sclerosis and brain trauma.
DRUG INFORMATION	The United States Food and Drug Administration (FDA) approved the fixed-dose combination of dextromethorphan hydrobromide and quinidine sulfate, for the treatment of PBA. Dextromethorphan acts as a weak sigma-1 receptor agonist and is also inhibits the reuptake of serotonin and norepinephrine in the CNS. Since dextromethorphan is metabolized by CYP2D6, it may not reach optimal bioavailability for successful therapy, when administered alone. Hence, it is administered in combination with quinidine, a CYP2D6 inhibitor.
ADVERSE EFFECTS	Muscle spasticity, spasms; pain in the abdomen; diarrhoea, vomiting; headache; dizziness. Contraindicated in individuals taking monoamine oxidase inhibitors (MAOIs).

Note: Mapmygenome recommends you to discuss possible adverse effects with your treating clinician.