

RightMed® Oncology Medication Report

The RightMed Oncology Report is a specialty report. It contains a set of medications selected and classified by OneOme for use in the treatment of oncology patients and their supportive care needs.

Patient and report summary

Patient name: Sample Patient
Patient date of birth: 1949-05-20
Original report date: 2025-09-10

Ordering provider: Sample Doctor Ordering facility: LIMSTITUTION Product type: Oncology Medication

Report type: Original

Major gene-drug interactions	2 Chemotherapeutic agents	1 Supportive care medications
Moderate gene-drug interactions	O Chemotherapeutic agents	4 Supportive care medications

Report legend

Based on this patient's genetic profile, medications are reported according to genotype-predicted interactions described below.



Major gene-drug interaction

Major genotype-drug interaction identified that affects the metabolism of the medication and/or indicates an elevated risk of adverse reaction or loss of efficacy.



Moderate gene-drug interaction

Moderate genotype-drug interaction identified that affects the metabolism of the medication and/or indicates an elevated risk of adverse reaction or loss of efficacy.



Minimal gene-drug interaction

Minimal genotype-drug interaction identified that does not significantly affect medication metabolism or predict an elevated risk of adverse reaction or loss of efficacy.

lcon legend

Some medications are reported with icons to indicate that specific clinical annotations and/or dosing guidelines provided by FDA, CPIC, or other professional associations are available in Vantage.

*	FDA evidence	This medication is listed on the FDA Table of Pharmacogenetic Associations. Not all genetic variants or genetic variant-inferred phenotypes may be accounted for. Further information can be found at: https://www.fda.gov/medical-devices/precision-medicine/table-pharmacogenetic-associations.
+	Increased exposure	Total exposure to active compound(s) may be increased. Monitor for adverse effects.
_	Decreased exposure	Total exposure to active compound(s) may be decreased. Monitor for lack of therapeutic response.
*	Difficult to predict	Total exposure to active compound(s) is difficult to predict. Monitor patient response.
ì	Reduced response	Response to medication may be lowered due to genetic changes impacting mechanisms other than exposure (e.g. receptor function).
	Additional testing	According to FDA labeling, additional laboratory testing may be indicated.
	Professional guideline	Medication has professional guidelines associated with this patient's genetic test results. Avoidance, dose adjustment, or heightened monitoring may be indicated.



Genotype-predicted interactions for chemotherapeutic agents

Chemotherapy

(1) Major gene-drug interaction

- Moderate gene-drug interaction
- Minimal gene-drug interaction

- Capecitabine (Xeloda®) * 1, 5, 17, 21, 38, 62,
 65
- Belinostat (Beleodaq®) 1, 65
- Fluorouracil * 🚉 1, 5, 17, 21, 38, 62, 65

Mercaptopurine (Purixan®) ■ 1, 3, 17, 44,
 55, 65

Irinotecan (Camptosar®) 1, 18, 26, 54, 65

■ Thioguanine (Tabloid®) 📭 1, 3, 17, 44, 55, 65

Kinase inhibitors (KIs) and monoclonal antibodies (mAbs)

(1) Major gene-drug interaction

Moderate gene-drug interaction

- Minimal gene-drug interaction
- Gefitinib (Iressa®) 1, 17, 65
- Nilotinib (Tasigna®) 1, 4, 65
- Pazopanib (Votrient®) 1, 49, 65

Genotype-predicted interactions for supportive care medications

Gastrointestinal management (nausea/vomiting, appetite, gastritis, GERD)

Major gene-drug interaction

- Moderate gene-drug interaction
- Dexlansoprazole (Dexilant®) 🚉 1, 41, 65
- Lansoprazole (Prevacid®) 📔 1, 17, 41, 65
- Omeprazole (Prilosec®) 1, 17, 19, 41, 47, 65
- Pantoprazole (Protonix®) 🚉 1, 17, 41, 65
- Minimal gene-drug interaction
- Dronabinol (Marinol®, Syndros®) 1, 65
- Metoclopramide (Reglan®) / 1, 7, 65
- Ondansetron (Zofran®) 1, 8, 31, 66

Pain management (opioid therapy)

Major gene-drug interaction

Moderate gene-drug interaction

- Minimal gene-drug interaction
- Codeine [1] 1, 15, 17, 42, 46, 65
- Tramadol (Ultram®) 👔 1, 15, 17, 46, 65

Neuropathy and non-opioid pain management

Major gene-drug interaction

↑ Moderate gene-drug interaction

Minimal gene-drug interaction

- Amitriptyline (Elavil®) * 1, 2, 17, 23, 24, 67
- Celecoxib (Celebrex®) 🚉 1, 63, 65
- Clomipramine (Anafranil®) 🚉 1, 17, 23, 65
- Desipramine (Norpramin®) 🛅 1, 23, 65
- Doxepin (Silenor®) * 1, 23
- Flurbiprofen (Ansaid®) 🚉 1, 63, 65
- Ibuprofen (Advil®, Motrin®) 🗱 🖺 1, 33, 63
- Imipramine (Tofranil®) 🗱 🚉 1, 17, 23, 58, 59
- Meloxicam (Mobic®) 🚉 1, 63, 65



Neuropathy and non-opioid pain management (cont.)

(1) Major gene-drug interaction

Moderate gene-drug interaction

- Minimal gene-drug interaction
- Nortriptyline (Pamelor®) 📳 1, 17, 23, 65
- Piroxicam (Feldene®) 🗱 🖺 1, 51, 52, 63
- Trimipramine (Surmontil®) 🗱 📭 1, 23, 36, 37

Mental health (antidepressants, anxiolytics)

Major gene-drug interaction

Moderate gene-drug interaction

- Minimal gene-drug interaction
- Citalopram (Celexa®) 👔 1, 10, 11, 17, 65
- Escitalopram (Lexapro®) 🚉 1, 10, 11, 17, 65
- Fluoxetine (Prozac®) 📳 1, 10, 11, 43
- Fluvoxamine 1, 10, 11, 17, 65
- Paroxetine (Paxil®) 📭 1, 10, 11, 17, 65
- Sertraline (Zoloft®) 🚉 1, 10, 11
- Venlafaxine (Effexor®) 1, 10, 17, 50, 60, 61, 65
- Vortioxetine (Trintellix®) 🚉 1, 10, 65

Neuropsychiatry (anticonvulsants, smoking cessation, sleep medication)

Major gene-drug interaction

1, 6, 17, 53, 65

Moderate gene-drug interaction

- Minimal gene-drug interaction
- Brivaracetam (Briviact®) 1, 65
- Clobazam (Onfi®) 1, 65, 68
- Fosphenytoin (Cerebyx®) 🚉 1, 34, 65
- Phenytoin (Dilantin®) 🚉 1, 34, 45, 65
- Quetiapine (Seroquel®) 🚉 1, 9

Antimicrobial (antibiotics, antifungals)

Carbamazepine (Carbatrol®, Tegretol®) *

Major gene-drug interaction

↑ Moderate gene-drug interaction

- Minimal gene-drug interaction
- Voriconazole (Vfend®) 📲 1, 13, 17, 48, 65

Antithrombosis and cardiovascular management (hypertension, hyperlipidemia)

Major gene-drug interaction

↑ Moderate gene-drug interaction

- ✓ Minimal gene-drug interaction
- Atorvastatin (Lipitor®) 1, 14, 17, 65
- Clopidogrel (Plavix®) 🚉 1, 17, 40
- Fluvastatin (Lescol®) 🚉 1, 14, 17
- Lovastatin (Mevacor®) [1, 14
- Pitavastatin (Livalo®) 🚉 1, 14 ■ Pravastatin (Pravachol®) 🚉 1, 14
- Rosuvastatin (Crestor®) 1, 14, 65
- Simvastatin (Zocor®) 🚉 1, 14, 17, 39, 65



Antithrombosis and cardiovascular management (hypertension, hyperlipidemia) (cont.) Major gene-drug interaction Moderate gene-drug interaction Warfarin (Coumadin®, Jantoven®) * 1, 12, 29, 30 Other Major gene-drug interaction Moderate gene-drug interaction Moderate gene-drug interaction Allopurinol (Aloprim®, Zyloprim®) * 16, 22, 27, 35, 56 Tamsulosin (Flomax®) 1, 65

Genotype-derived classification of medications is provided as a service by OneOme and is intended solely for use by a medical professional who has reviewed and understands all sections within this report, including possible limitations of the services provided by OneOme. The relationships between the drugs and pharmacogenes annotated in this report are supported by scientific evidence that meets OneOme's criteria for inclusion. The order in which drugs are listed does not have any clinical or medical implications. Commonly used trade names for medications are listed for reference only. The list may not be inclusive of all trade names available and does not indicate preference or recommendation by OneOme of one medication product over another. For more information on these medications, for a list of additional medications curated but not annotated by OneOme, or to evaluate possible drug-to-drug interactions, please consult Vantage, which is accessible through the provider portal at portal.oneome.com.



Gene and phenotype summary

Gene	Genotype		Phenotype summary / Metabolic status
CYP1A2	*1A/*1A	PAN IM NM RM UN	Normal metabolizer Fully functional enzyme activity is likely based on the genotype results. Normal metabolism of the medication affected by this gene is predicted.
CYP2B6	*1/*1	PM IM NM RM UN	Normal metabolizer Fully functional enzyme activity is likely based on the genotype results. Normal metabolism of the medication affected by this gene is predicted.
CYP2C9	*1/*1	PM IM NM RM UN	Normal metabolizer Fully functional enzyme activity is likely based on the genotype results. Normal metabolism of the medication affected by this gene is predicted.
CYP2C19	*1/*1	PM IM NM RM UN	Normal metabolizer Fully functional enzyme activity is likely based on the genotype results. Normal metabolism of the medication affected by this gene is predicted.
CYP2C Cluster	rs12777823 GG		Normal CYP2C rs12777823 homozygous wild-type genotype consistent with normal clearance of a certain medication, independent of the impact of CYP2C9*2 and *3. CYP2C rs12777823, together with CYP4F2, CYP2C9, and VKORC1, may affect treatment management of a certain medication.
CYP2D6	*1/*10	PM IM NM RM UM	Normal metabolizer Fully functional enzyme activity is likely based on the genotype results. Normal metabolism of the medication affected by this gene is predicted.
СҮРЗА4	*1/*1	PAN IM NM RM UM	Normal metabolizer Fully functional enzyme activity is likely based on the genotype results. Normal metabolism of the medication affected by this gene is predicted.
СҮРЗА5	*3/*3	PRA IM NM RM UN	Poor metabolizer This CYP3A5 genotype is associated with the phenotype most prevalent in studies used to define standard dosing guidelines.



Gene and phenotype summary (cont.)

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CYP4F2	*1/*1	⊘	Normal activity Genotype consistent with normal activity of the CYP4F2 enzyme, which catalyzes the metabolism of vitamin K, in counterpoint to the activity of VKORC1. CYP4F2, together with CYP2C9, VKORC1, and a variant in CYP2C Cluster, may affect treatment management of a certain medication.
СОМТ	rs4680 AA	\triangle	Low activity The AA (Met/Met) genotype is associated with lower COMT activity than the GG (Val/Val) or GA (Val/Met) genotypes.
DPYD	*1/HapB3		Intermediate metabolizer DPD activity score= 1.5. This genotype and activity score is consistent with an intermediate metabolizer phenotype. Decreased DPD enzyme activity is associated with an increased risk for severe or fatal drug toxicity when treated with fluoropyrimidine drugs.
F2	rs1799963 GG	②	Normal risk Normal risk of thrombosis associated with Factor II (prothrombin). Other genetic and clinical factors contribute to the risk for thrombosis.
F5	rs6025 GG	②	Normal risk Normal risk of thrombosis associated with Factor V. Other genetic and clinical factors contribute to the risk for thrombosis.
GRIK4	rs1954787 TT	\triangle	Altered receptor function Homozygous variant glutamate ionotropic receptor kainate type subunit 4 (GRIK4) genotype is consistent with altered receptor function.
HLA-A	Positive for *31:01		Increased risk Positive for presence of the HLA-A*31:01 allele. Increased risk of hypersensitivity induced by a certain medication, and possibly others of structural similarity. Hypersensitivity and severe cutaneous reactions may occur regardless of the presence of the HLA-A*31:01 allele, in particular the presence of the HLA-B*15:02 allele is associated with severe cutaneous reactions induced by certain medications.
HLA-B	Negative	⊘	Normal risk Negative for presence of the HLA-B*15:02, HLA-B*57:01, and HLA-B*58:01 alleles. Normal risk of hypersensitivity, severe cutaneous reactions, and severe hepatotoxicity induced by certain medications. Hypersensitivity, severe cutaneous reactions, and severe hepatotoxicity may occur regardless of the presence of HLA-B*15:02, HLA-B*57:01, or HLA-B*58:01 alleles. In particular, the presence of the HLA-A*31:01 allele is associated with hypersensitivity reactions induced by a certain

medication, and possibly other medications of structural similarity.



Gene and phenotype summary (cont.)

HTR2A	rs7997012 TT	②	Variant absent This genotype is the wildtype genotype associated with intron 2 of the HTR2A gene.
NUDT15	*1/*1	⊘	Normal metabolizer NUDT15 genotype is consistent with normal enzyme activity and is not associated with an increased risk of thiopurine-induced toxicities. Toxicities with thiopurines can occur due to impaired TPMT activity independently from the NUDT15 activity.
OPRM1	rs1799971 AA	⊘	Variant absent The AA genotype (or Asn/Asn isoform) is the wildtype genotype associated with the mu-1 opioid receptor.
SLC6A4	L/L (La/La)	⊘	Variant present The SLC6A4 5-HTTLPR (L= long, S= short) and rs25531 variants are located in the promoter region of this gene and may influence transcriptional activity.
SLCO1B1	*1/*1	②	Normal function SLCO1B1 genotype consistent with normal function of the OATP1B1 transporter.
TPMT	*1/*1	⊘	Normal metabolizer TPMT genotype is consistent with a normal metabolizer phenotype and is not associated with an increased risk of thiopurine-induced toxicities. Toxicities with thiopurines can occur due to impaired NUDT15 activity independently from the TPMT activity.
UGT1A1	*1/*1	②	Normal metabolizer Genotype consistent with fully functional UGT1A1 enzyme activity, or a normal metabolizer phenotype.
VKORC1	rs9923231 GG	⊘	Normal activity Genotype consistent with normal expression of the vitamin K epoxide reductase gene.

CYP phenotype abbreviations

PM	Poor metabolizer
IM	Intermediate metabolizer
NM	Normal metabolizer
RM	Rapid metabolizer
UM	Ultrarapid metabolizer



Test information

Specimen ID: 6423878267926 Clinical testing performed by: Reported by: Ellie Jhun in None

Specimen type: Buccal swab
OneOme
CLIA: 24D2109855
Collection date: 2025-09-01
Receive date: 2025-09-10
Minneapolis, Minnesota 55413, United States
NY PFI: 9226

Test results

The following analytical results were interpreted by OneOme to produce the pharmacogenomic interpretations and annotations described in the *Gene and phenotype summary*. Method-specific analytical limitations or inferred haplotypes may limit the ability to produce a definitive phenotype interpretation. See *Methodology and limitations* and/or the *Report and laboratory comments* sections for additional information.

CYP1A2 *1A	/*1A		rs5030656	NM_000106.5:c.841_843delAAG	AAGAAG
rs762551	NM_000761.4:c9-154C>A	CC	rs35742686 rs72549353	NM_000106.5:c.775delA NM_000106.5:c.765_768delAACT	AA AACTAACT
rs2069514	NG_008431.2:g.28338G>A	GG	rs5030655	NM_000106.5:c.454delT	TT
rs2069526	NM_000761.4:c10+103T>G	TT	rs774671100	NM_000106.5:c.137_138insT	
rs12720461	NM_000761.4:c10+113C>T	CC	rs1080985	NM_000106.5:c1584C>G	CC
rs35694136	NM_000761.4:c1635delT	TT	rs59421388	NM_000106.5:c.1012G>A	GG
	····· <u>-</u> 5557511 1161 1555 4 611		rs28371725	NM_000106.5:c.985+39G>A	GG
CYP2B6 *1/*	*1		rs72549346	NM_000106.5:c.1088_1089insGT	
			rs5030865	NM_000106.5:c.505G>[A,T]	GG
s3745274	NM_000767.4:c.516G>T	GG	135050005	14M_000100.3.c.3030 [A,1]	00
s2279343	NM_000767.4:c.785A>G	AA	CYP3A4 *1/	*1	
s34223104	NM_000767.5:c82T>C	TT		<u> </u>	
s3211371	NM_000767.4:c.1459C>T	CC	rs2740574	NM_017460.5:c392G>A	AA
s36079186	NM_000767.5:c.593T>C	TT	rs35599367	NM_017460.5:c.522-191C>T	CC
s28399499	NM_000767.4:c.983T>C	TT			
			CYP3A5 *3/	[*] 3	
CYP2C9 *1/*	*1		770740	NIA 000777 4 040 0070 4	
s7900194	NM_000771.3:c.449G>A	GG	rs776746	NM_000777.4:c.219-237G>A	GG
s1799853	NM_000771.3:c.449G>A NM_000771.3:c.430C>T	CC	rs10264272	NM_000777.4:c.624G>A	GG
s1799855 s1057910			rs41303343	NM_000777.4:c.1035_1036insT	
	NM_000771.3:c.1075A>C	AA	C)/D4E2 *4/8	· A	
s28371686	NM_000771.3:c.1080C>G	CC	CYP4F2 *1/*	· 1	
rs56165452	NM_000771.3:c.1076T>C	TT	rs2108622	NM_001082.4:c.1297G>A	GG
s28371685	NM_000771.3:c.1003C>T	CC	132100022	NM_001002.4.0.123707A	00
s9332131	NM_000771.3:c.817delA	AA	COMT rs46	80 AA	
CYP2C19 *1,	/*1		rs4680	NM_000754.3:c.472G>A	AA
s12248560	NM_000769.2:c806C>T	CC			
s4244285	NM_000769.2:c.681G>A	GG	DPYD *1/Ha	pB3	
s4986893	NM_000769.2:c.636G>A	GG	rs75017182	NM_000110.4:c.1129-5923C>G	CG
s6413438	NM 000769.2:c.680C>T	CC		_	TT
s28399504	 NM_000769.2:c.1A>G	AA	rs55886062	NM_000110.3:c.1679T>G	
	· ···· <u>-</u> - · · · · · · ·		rs67376798	NM_000110.3:c.2846A>T	AA
CYP2C Clust	er rs12777823 GG		rs3918290	NM_000110.3:c.1905+1G>A	GG
			rs115232898	NM_000110.4:c.557A>G	AA
s12777823	NC_000010.10:g.96405502G>A	GG	F2 rs179996	63 GG	
CYP2D6 *1/*	*10		rs1799963	NM_000506.4:c.*97G>A	GG
s28371706	NM_000106.5:c.320C>T	CC			
s267608319	NM_000106.5:c.1319G>A	GG	F5 rs6025 (3 G	
s16947	NM_000106.5:c.886C>T	CC	rs6025	NM_000130.4:c.1601G>A	GG
s79292917	NM_000106.5:c.975G>A	GG	150023	141VI_000130.4.0.1001G/A	GG
s1065852	NM_000106.5:c.100C>T	CT	GRIK4 rs195	54787 TT	
1135840	NM_000106.5:c.1457G>C	GC	OKIK+ 15190	, , ,,,,	
3892097	NM_000106.5:c.506-1G>A	GG	rs1954787	NM_001282470.2:c.83-10039T>C	TT
s769258	NM_000106.5:c.31G>A	GG		_	
s5030862	NM_000106.5:c.124G>A	GG	HLA-A Posi	tive for *31:01	
s201377835	NM_000106.5:c.181-1G>C	GG			
s5030867	NM_000106.5:c.971A>C	AA	HLA00097	NM_002116 (interrogated at exon 2)	Positive
	14111_000100.3.C.371A/C	~~			
s765776661	NM_000106.5:c.1411_1412insTGCCCACTG	GTGCCCACGTGCC	C		



Test results (cont.)

rs144012689	NM_005514.7:c.1012+104A>T	TT	SLCO1B1 *1/*1		
HLA00386	NM_005514 (interrogated at exon 2 and intron 2)	Negative	rs2306283	NM_006446.4:c.388A>G	Not tested
HLA00381	NM_005514 (interrogated at exon 3)	Negative	rs4149056	NM_006446.4:c.521T>C	TT
		· ·	rs4149015	NM_006446.4:c910G>A	Not tested
HTR2A rs79	997012 TT				
7007040	NIM COOCCA A CAA COMTE C		— TPMT *1/*1		
rs7997012	NM_000621.4:c.614-2211T>C	TT	rs1142345	NM_000367.3:c.719A>G	AA
NUDT15 *1/*	*1		rs1800584	NM_000367.3:c.626-1G>A	CC
			rs1800462	NM_000367.3:c.238G>C	GG
rs116855232	NM_018283.3:c.415C>T	CC	rs1800460	NM_000367.3:c.460G>A	GG
OPRM1 rs17	99971 AA		UGT1A1 *1/	*1	
rs1799971	NM_000914.4:c.118A>G	AA	rs4148323	NM_001072.3:c.862-6536G>A	GG
			rs1976391	NM_001072.3:c.862-9697A>G	AA
SLC6A4 L/L	. (La/La)				
rs25531	NM_001045.5:c1936A>G	AA	VKORC1 rs	9923231 GG	
rs774676466	NM_001045.5:c19171875del43	LL	rs7200749	NM_024006.5:c.358C>T	Not tested
			rs9923231	NM_001311311.1:c1639G>A	GG

Electronically signed by:

Ellie Jhun in None 2025-09-10



Methodology and limitations

This test was developed, and its performance characteristics determined by OneOme, LLC, a clinical laboratory located at 807 Broadway Street NE Suite 100, Minneapolis, MN 55413. These tests have not been cleared or approved by the U.S. Food and Drug Administration. The FDA does not require this test to go through premarket FDA review. OneOme is certified under CLIA-88 and accredited by the College of American Pathologists as qualified to perform high-complexity testing. This test is approved for clinical use by the New York State Department of Health. This test should not be regarded as investigational or for research.

Genomic DNA was analyzed by PCR using Thermo Fisher TaqMan® and/or LGC Biosearch BHQ® probe-based methods to interrogate the variant locations listed in the Test results table above. For tests that include CYP2D6, the CYP2D6 copy number status was assessed at sites within the promoter, intron 2, intron 6, and exon 9. The test detects CYP2D6 deletions, duplications/multiplications, and hybrid alleles, but cannot differentiate duplications in the presence of a deletion.

Legacy nomenclature for applicable genes and alleles is used to remain consistent with industry language.

The test does not detect all known and unknown variations in the gene(s) tested, nor does absence of a detectable variant (designated as *1 for genes encoding drug metabolizing enzymes) rule out the presence of other, non-detected variants.

As with other common SNP genotyping techniques, these assays cannot differentiate between the maternal and paternal chromosomes. In cases where observed variants are associated with more than one haplotype, OneOme infers and reports the most likely diplotype based on published allele frequency and/or ethnicity data. Inferences with potential clinical impact are reported in the Report and laboratory comments section.

PCR may be subject to general interference by factors such as reaction inhibitors and low quality or quantity of extracted DNA. When present, these interferents typically yield no result rather than an inaccurate one. Very infrequent variants or polymorphisms occurring in primer- or probe-binding regions may affect testing and could produce an erroneous result or assay failure. Variant locations tested by the assay but not assigned a genotype call are reported as "No Call."

The variant detection methods validated by OneOme provide >99.9% accuracy for the adult population; however, clinical interpretation may be inaccurate for patients who have undergone or are receiving non-autologous blood transfusions, tissue, and/or organ transplant therapies. Although extremely rare, results could also be impacted by other factors not addressed above, such as laboratory error.

Pharmacogenetic correlation is largely based on studies of adult populations. Gene-drug guidance may not be informative in pediatric patients. For patients that may carry a probabilistic risk of disease, patients and providers should consider the benefits of consulting with a trained genetic counseling professional, physician, or pharmacogenomic specialist. For additional support, contact OneOme through the website or by calling 844-663-6635.



OneOme liability disclaimer

The interpretations and clinical annotations provided by OneOme are intended solely for use by a medical professional in the treatment of adult patients and do not constitute medical advice by OneOme. The treating provider remains ultimately responsible for all diagnosis and treatment decisions for the patient. OneOme disclaims liability for any errors, omissions or ambiguities in any translation or interpretation of a report by a third party, including without limitation direct, indirect, incidental, special, consequential or exemplary damages, whether such damages arise in contract, negligence, tort, under statute, in equity, at law or otherwise. Information included in this report is based upon scientific literature, including information from and guidelines published by professional associations (e.g., CPIC, FDA, DPWG), and does not take into account other genetic variants and environmental or social factors that may affect a patient's response. Other factors not included in this report include, but are not limited to, environmental factors (e.g., smoking), health factors (e.g., diet), social and familial factors, various medical conditions, and drug-to-drug interactions. Administration of any medication, including the ones listed in the OneOme reports, requires careful therapeutic monitoring regardless of the phenotype or genotype-predicted interaction reported. As a matter of practice, OneOme will routinely update its pharmacogenomic database as new information becomes available to the scientific community. Genotype-predicted interactions and annotations found on the patient's RightMed Comprehensive Test Report, Vantage Reports, or RightMed specialty reports are therefore dependent on the date of generation and/or the database version used to generate that report. Providers may access these reports with updated annotations using OneOme's latest released version through the provider portal at portal.oneome.com.



References

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