Pharmacogenetics: Another Tool in the Genetic Counselor Toolbox

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Conflicts of Interest

- Gillian Bell, PharmD – employee of Genome Medical, granted options as part of employment

- Rachel Mills, CGC – nothing to disclose

Objectives

- Define pharmacogenetics and related terms
- Utilize available resources to support pharmacogenomic care
- List clinical situations where pharmacogenomic testing is likely to be most useful clinically
- Recognize the roles for genetic counselor in pharmacogenetics services

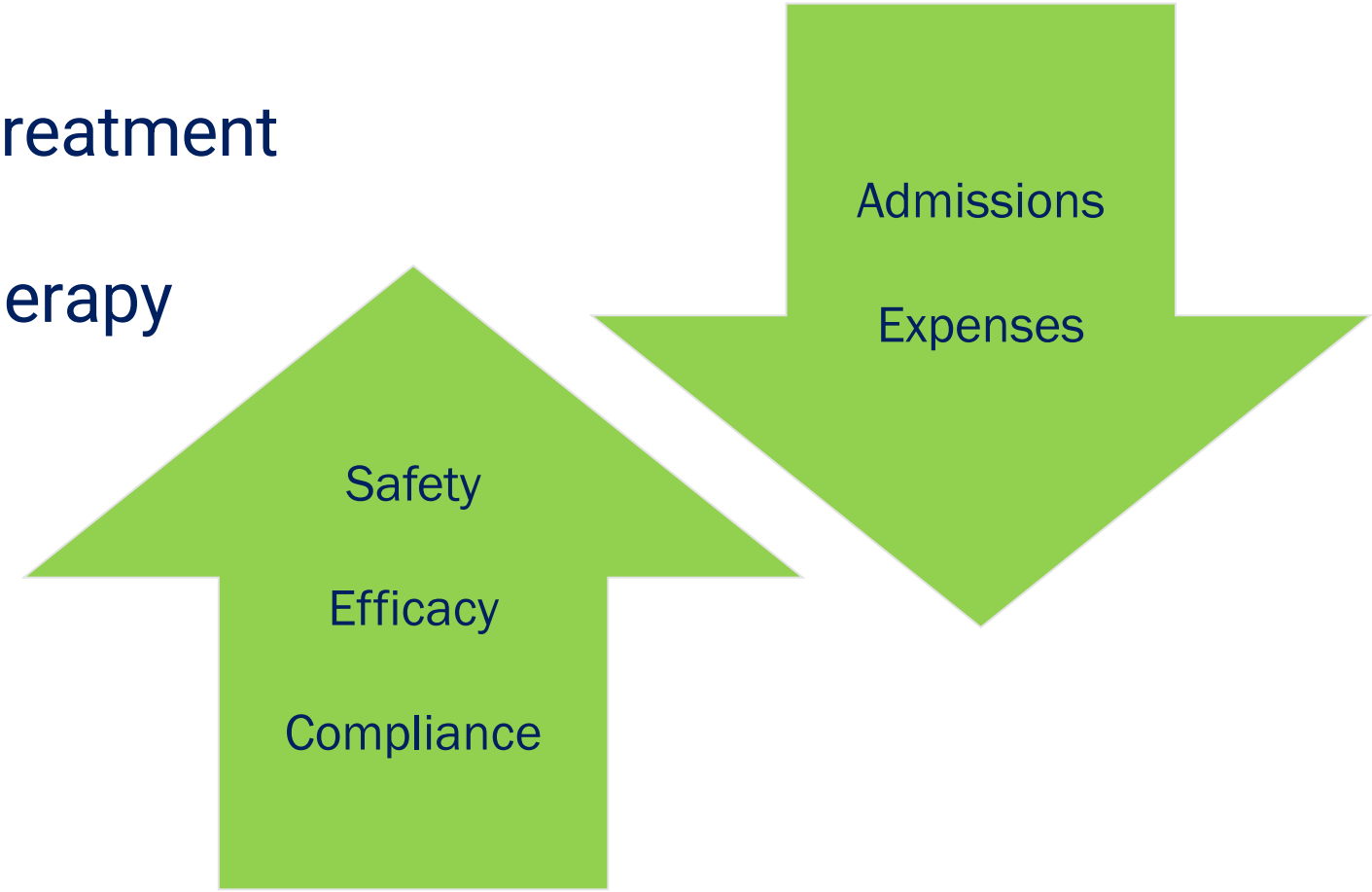
What is pharmacogenetics (PGx)?

- How genetic variations affect drug response
- Pharmacokinetic
  - Drug metabolizing enzymes (*CYP2D6, CYP2C19*)
  - Drug transporters (*SLCO1B1*)
- Pharmacodynamic
  - Drug targets (*VKORC1, OPRM1, SLC6A4*)
- Immune response
  - Hypersensitivity (*HLA-B, HLA-A*)

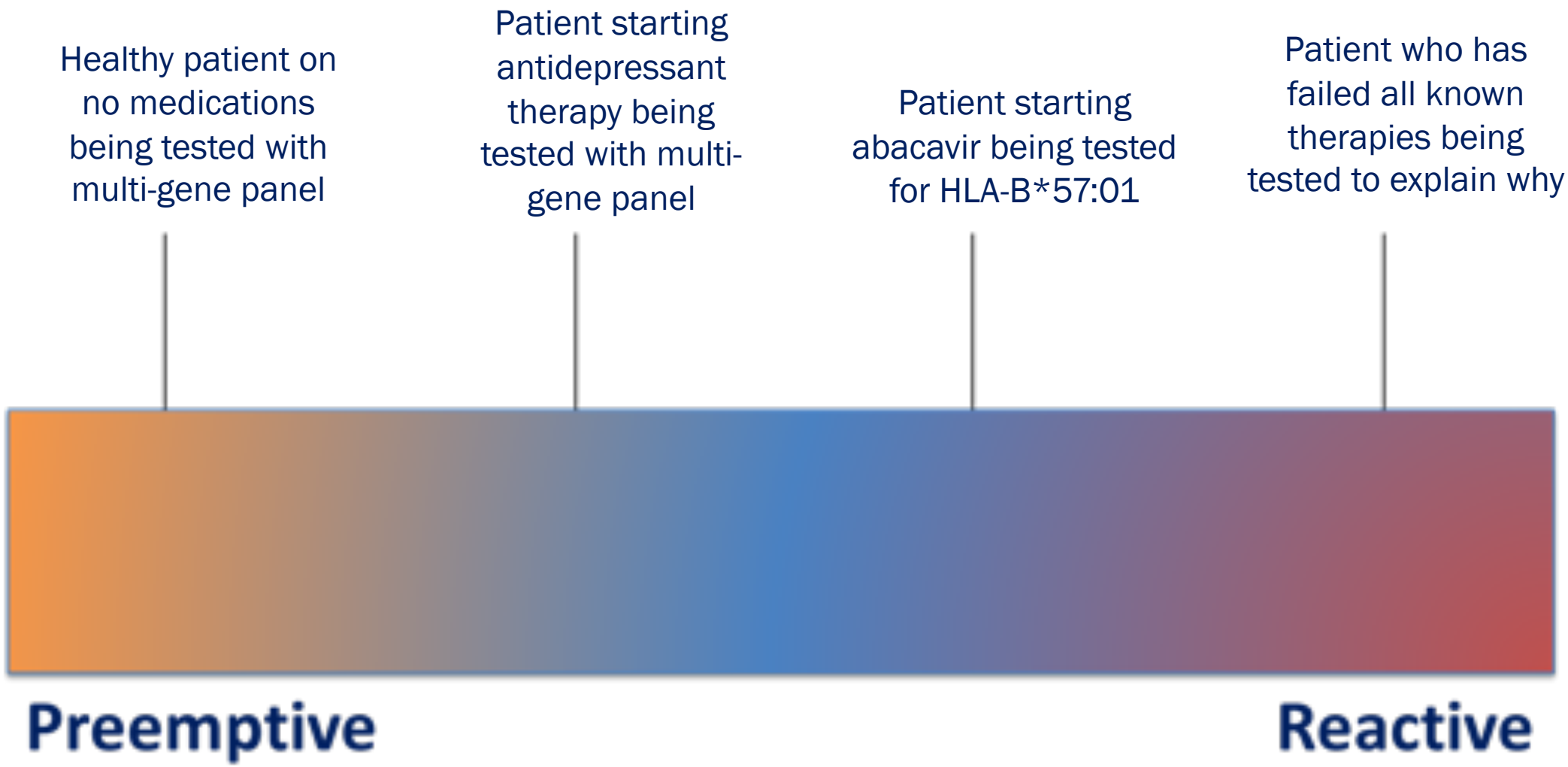
Urs A. Meyer *Nature Reviews Genetics* 5: 669-676, 2004; Crews, et al. *Clin Pharmacol Ther* 2012

PGx helps clinicians choose between therapeutic equals

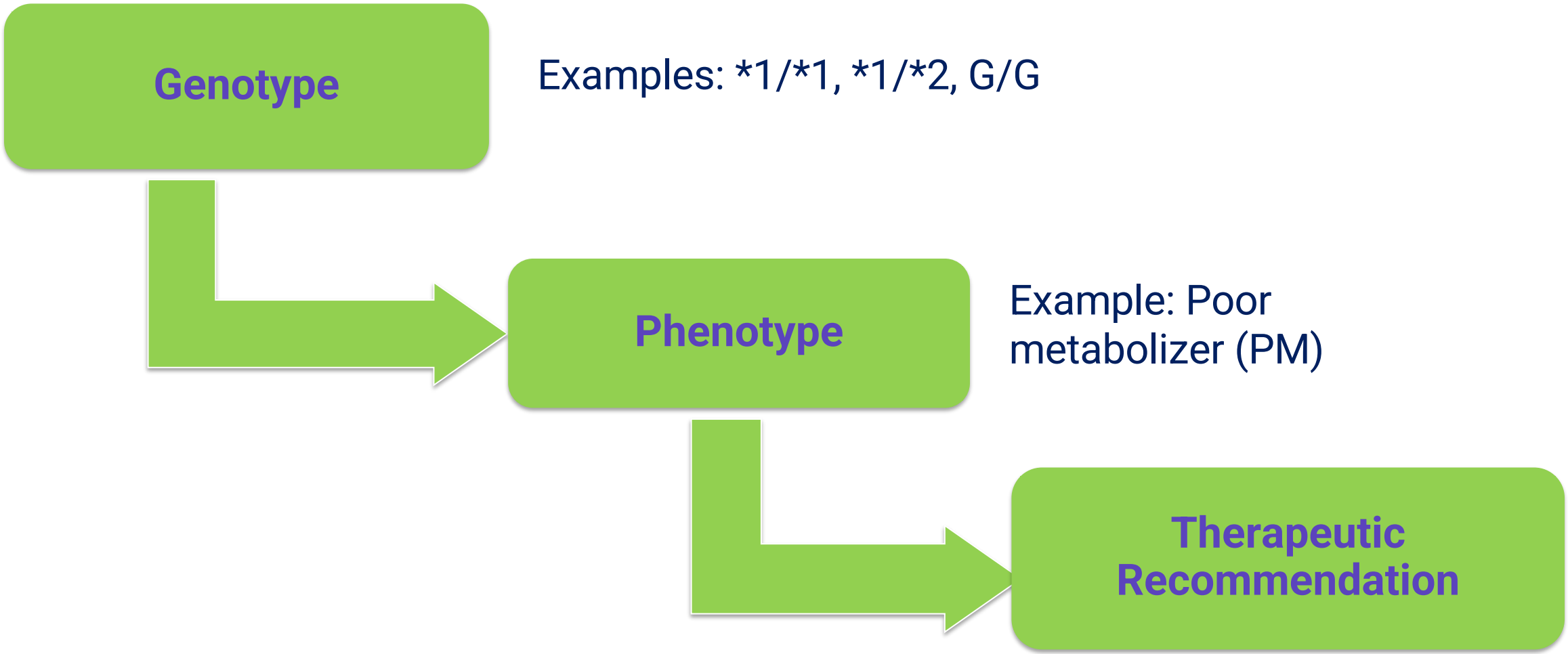
- Safer and more effective drug treatment
- Increased adherence to drug therapy
- Decreased hospitalizations
- Decreased health care costs



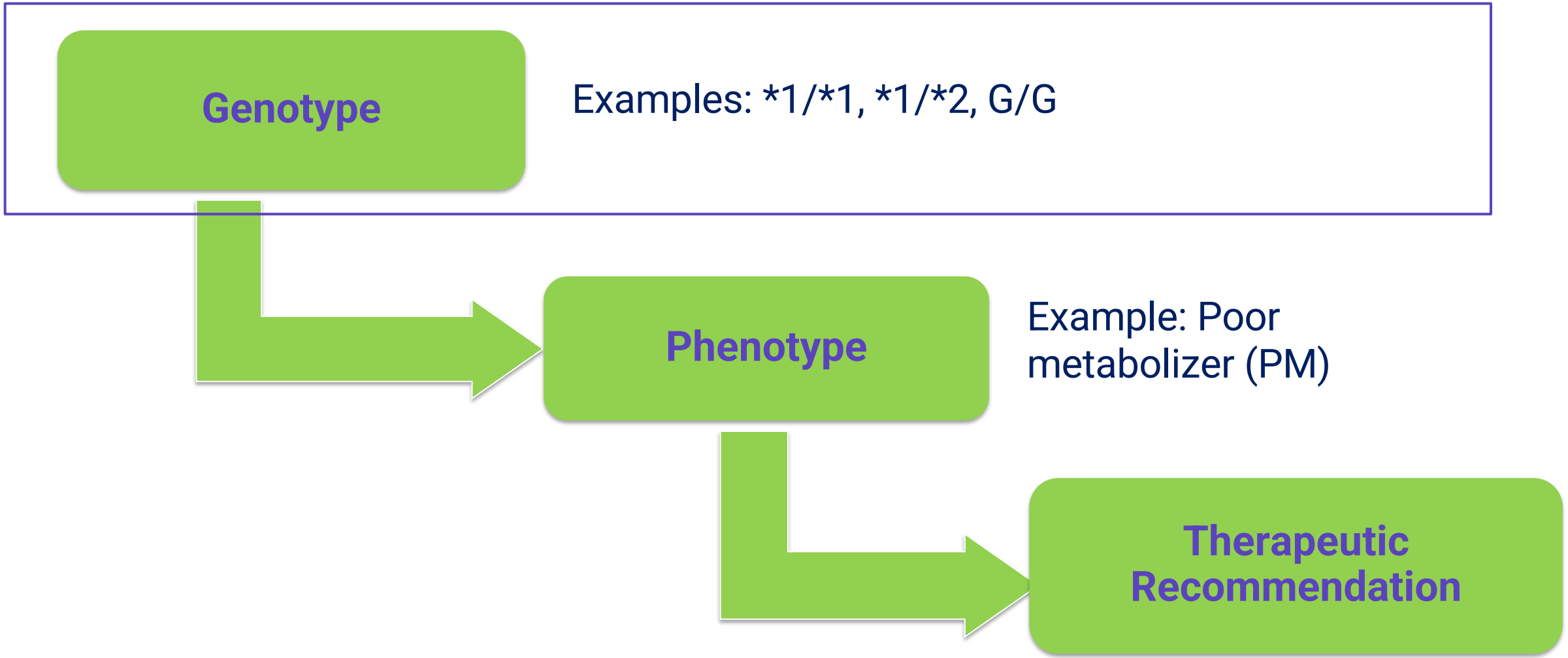
Preemptive vs. Reactive Testing



Interpreting results



Interpreting results



What types of variations?

- Single Nucleotide Polymorphisms (SNPs) – A change in one base pair in a genetic sequence
- InDels – Inserts or deletions of a genetic sequence
  - Can be a few to 100s of base pairs
- Copy number variations – Can have duplications and deletions of genes

Star allele nomenclature can be confusing

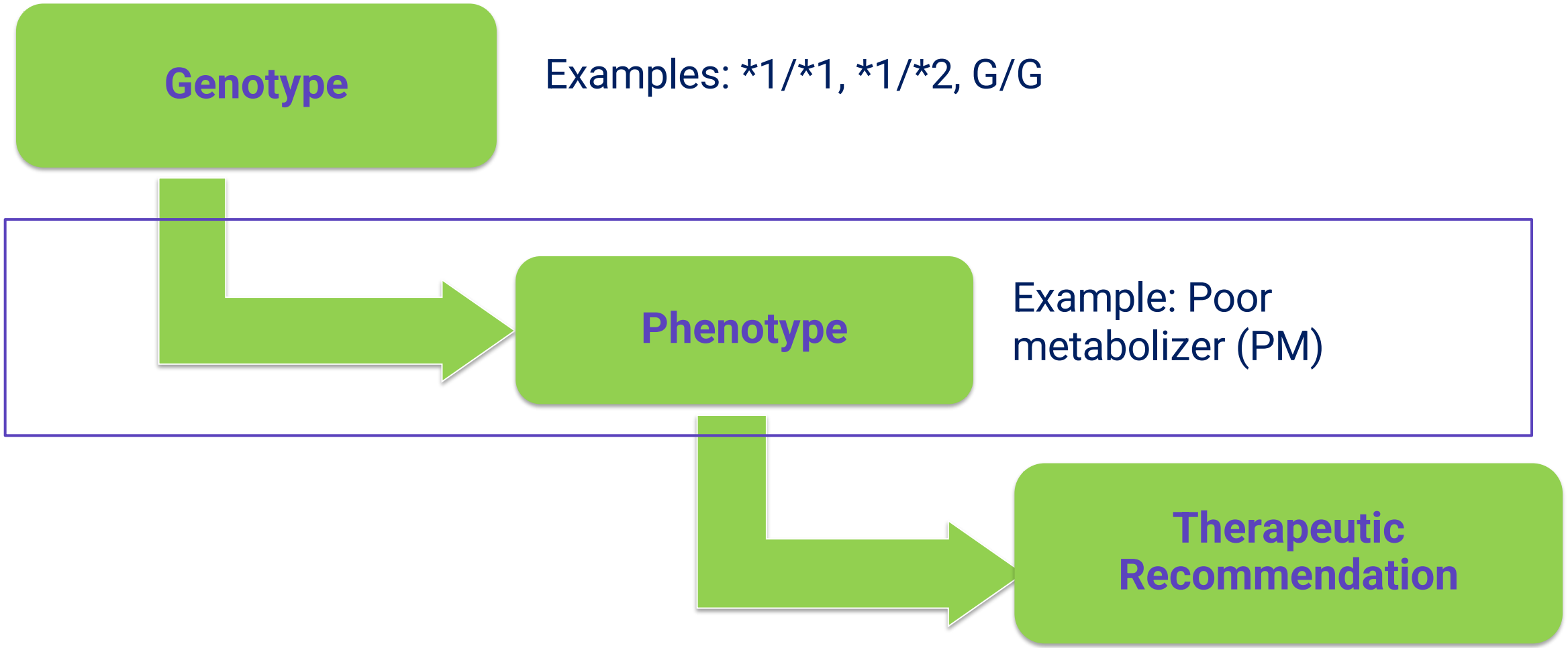
- Used for drug-metabolizing enzymes
- “ Wild-type” or reference allele designated *1
  - Normal activity
- Variants sequentially numbered
  - *CYP2D6* *2 is first variant identified
- Star alleles are not the same across genes!!
  - *CYP2D6**2 = normal function
  - *CYP2C9**2 = decreased function
- Genotype would be combination of alleles (*1/*2)

Other allele nomenclature

- Basic alpha numeric nomenclature
  - Gene Position Allele
  - *OPRM1* A118G
  - *OPRM1* 118 A>G

- Reference SNP (rs) nomenclature
  - The rs naming system is used by the National Center for Biotechnology Information SNP database (dbSNP)
  - Each SNP is assigned a number
  - *OPRM1* A118G = rs1799971

Interpreting results



Phenotype Terms

Drug Metabolizing Enzymes (e.g. CYP enzymes, UGT1A1, TPMT, DPYD)	Ultrarapid Metabolizer
	Rapid Metabolizer
	Normal Metabolizer
	Intermediate Metabolizer
	Poor Metabolizer
Transporters (e.g. SLCO1B1)	Increased Function
	Normal Function
	Decreased Function
	Poor Function
High-risk alleles (e.g. *HLA-B*15:02*)	Positive
	Negative

Worry about drug toxicity!

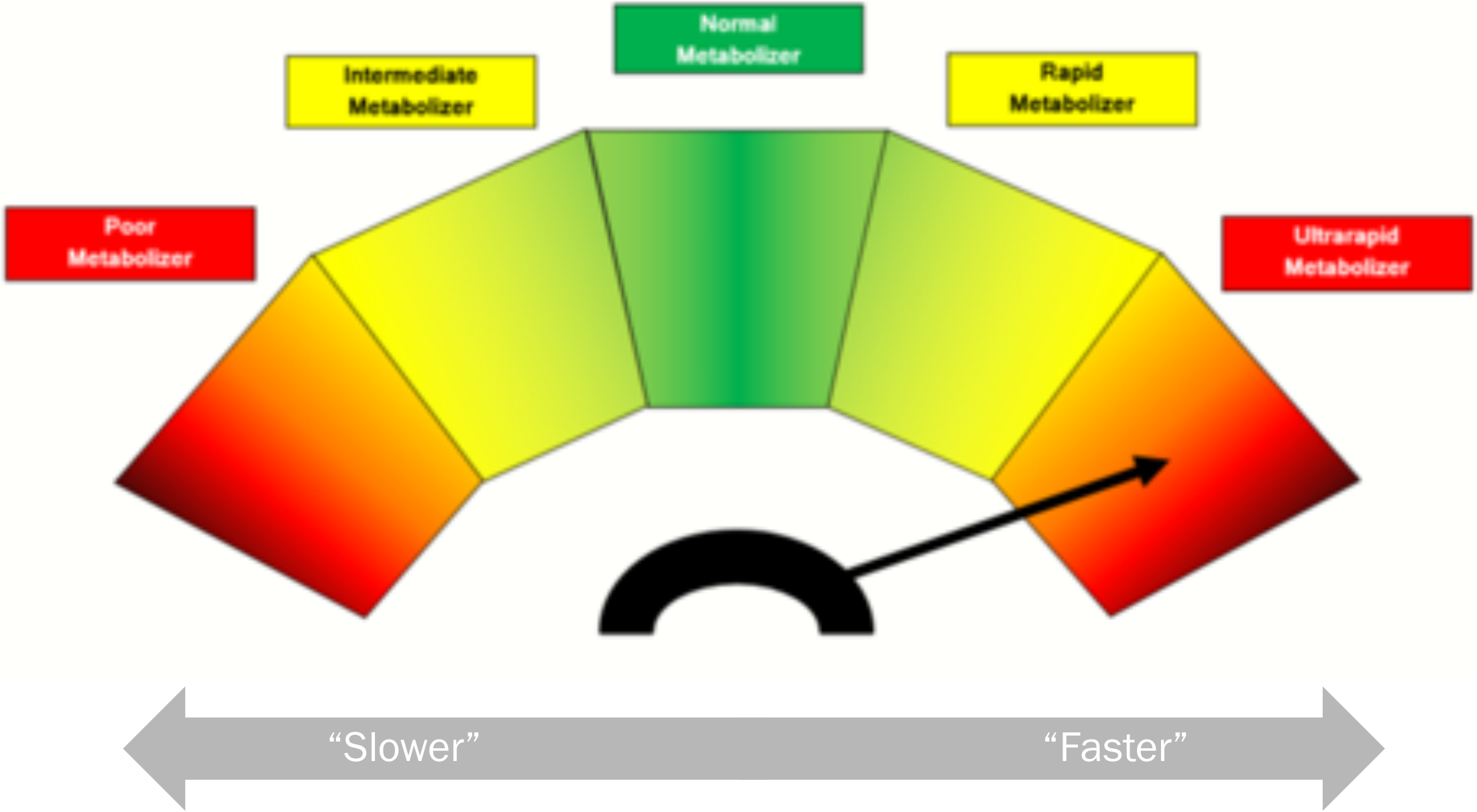
Worry about drug efficacy!

Poor metabolizer - very little to no breakdown, may experience side effects at standard doses

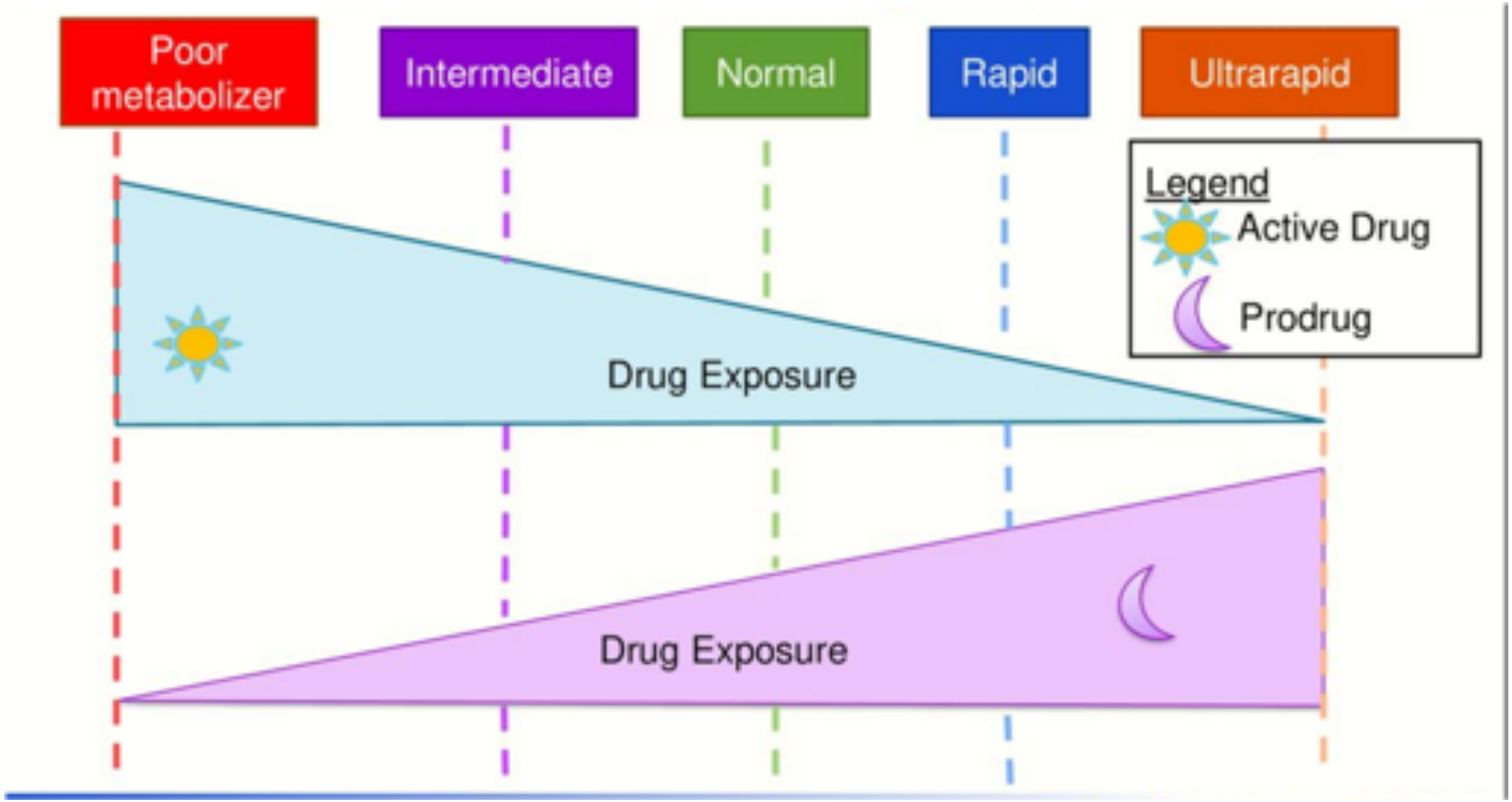
Intermediate metabolizer - breaks down drugs to a lesser extent than NMs. In most cases, should be able to process standard doses of drugs.

Normal metabolizer - breaks down drugs “normally.” Able to process standard doses of drugs.

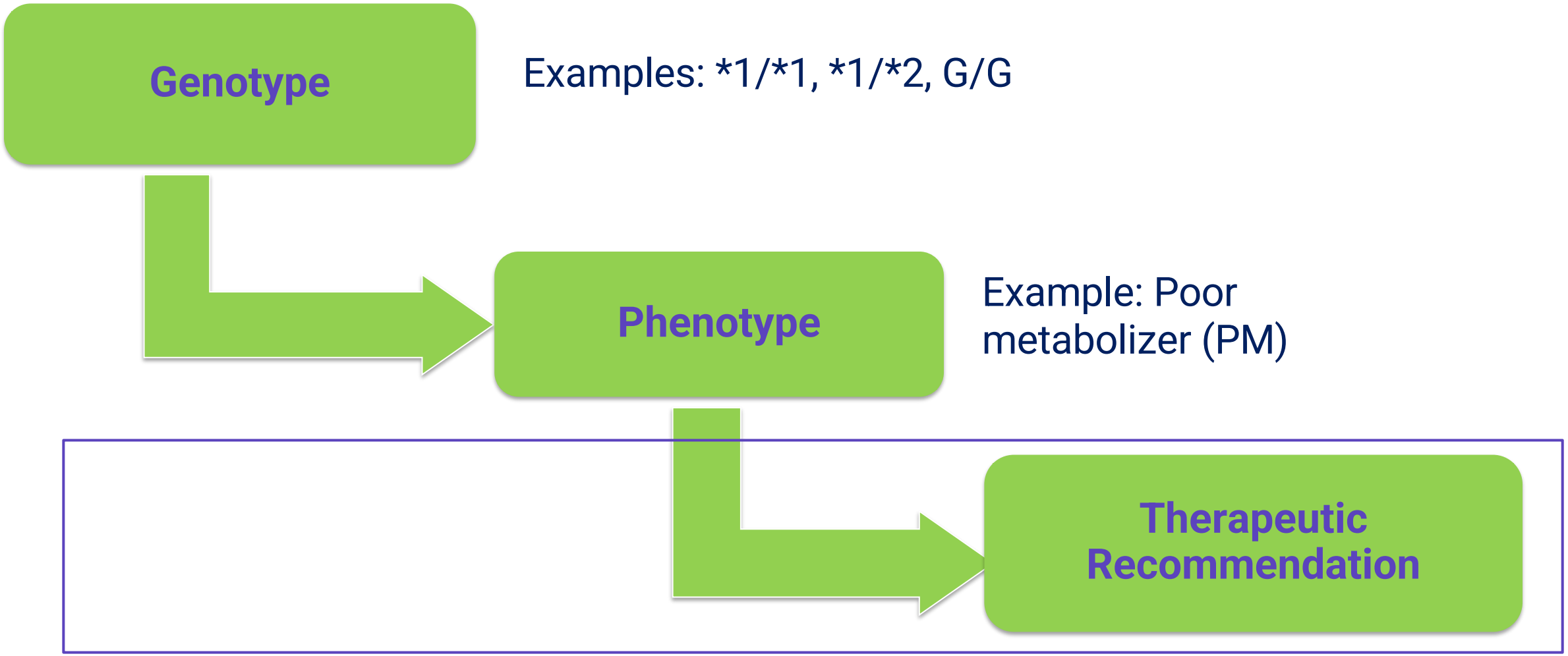
Ultrarapid/rapid metabolizer - breaks down drugs to a greater extent, may not get benefit at standard doses


Normal Metabolizer
Intermediate Metabolizer
Rapid Metabolizer
Poor Metabolizer
Ultrarapid Metabolizer
“Slower”
“Faster”

Active drug vs. Prodrug



Interpreting results



- >400 members
- Clinicians and scientists
- >260 institutions and companies
- 35 countries
- 14 Observers (FDA, professional societies)
- CPIC guidelines are designed to help clinicians understand HOW available genetic test results should be used to optimize drug therapy

CPIC™
Clinical Pharmacogenetics Implementation Consortium

- Have published 25 guidelines and 12 updates; 20 genes and >61 drugs
  - Commonly used drugs include NSAIDs, antidepressants, drugs for reflux and opioid pain drugs

Tables in CPIC guidelines

- Table 1: Genotype to phenotype translation
- Table 2: Prescribing recommendations linked to phenotypes, standard grading for strength of recommendation
- Supplementary material
  - Allele definition table
  - Allele functionality table
  - Allele frequency tables
  - Diplotype-phenotype assignment table
  - Informatics-related tables

CPIC guideline table 1

Table 1b Assignment of CYP2C19 predicted phenotypes

Likely phenotype	Genotypes	Examples of *CYP2C19* diplotypes
Ultrarapid metabolizer (~5–30% of patients)[a]	An individual carrying two increased function alleles or one normal function allele and one increased function allele	*17/*17, *1/*17
Extensive metabolizer (~35–50% of patients)	An individual carrying two normal function alleles	*1/*1
Intermediate metabolizer (~18–45% of patients)	An individual carrying one normal function allele or one increased function allele and one no function allele	*1/*2, *1/*3, *2/*17[a]
Poor metabolizer (~2–15% of patients)	An individual carrying two no function alleles	*2/*2, *2/*3, *3/*3

CPIC guideline table 2

Table 3a Dosing recommendations for citalopram and escitalopram based on CYP2C19 phenotype

Phenotype	Implication	Therapeutic recommendation	Classification of recommendation[a]
CYP2C19 Ultrarapid metabolizer	Increased metabolism when compared to extensive metabolizers. Lower plasma concentrations will increase probability of pharmacotherapy failure.	Consider an alternative drug not predominantly metabolized by CYP2C19.[b]	Moderate
CYP2C19 Extensive metabolizer	Normal metabolism	Initiate therapy with recommended starting dose.	Strong
CYP2C19 Intermediate metabolizer	Reduced metabolism when compared to extensive metabolizers.	Initiate therapy with recommended starting dose.	Strong
CYP2C19 Poor metabolizer	Greatly reduced metabolism when compared to extensive metabolizers. Higher plasma concentrations may increase the probability of side effects.	Consider a 50% reduction[c,d] of recommended starting dose and titrate to response or select alternative drug not predominantly metabolized by CYP2C19.[b]	Moderate

Strength of recommendation

Strength of Recommendation	Definition
Strong	The evidence is high quality and the desirable effects clearly outweigh the undesirable effects
Moderate	There is a close or uncertain balance as to whether the evidence is high quality and the desirable clearly outweigh the undesirable effects
Optional	The desirable effects are closely balanced with undesirable effects, or the evidence is weak or based on extrapolations. There is room for differences in opinion as to the need for the recommended course of action
No recommendation	The desirable effects are closely balanced with undesirable effects, or the evidence is weak or based on extrapolations. There is room for differences in opinion as to the need for the recommended course of action

Caudle KE, et al. *Curr Drug Metab*. 2014;15:209-17

Commonly prescribed meds with CPIC guidelines

Omeprazole (Prilosec)	Fluoxetine (Prozac)	Paroxetine (Paxil)
Simvastatin (Zocor)	Tramadol (Ultram)	Naproxen (Naprosyn)
Hydrocodone (Percocet)	Meloxicam (Mobic)	Ondansetron (Zofran)
Sertraline (Zoloft)	Clopidogrel (Plavix)	Amitriptyline (Elavil)
Pantoprazole (Protonix)	Aspirin	Esomeprazole (Nexium)
Escitalopram (Lexapro)	Warfarin (Coumadin)	Diclofenac (Flector)
Citalopram (Celexa)	Oxycodone	
Ibuprofen (Advil)	Allopurinol (Xyloprim)	

There are >250 FDA approved drugs with pharmacogenomic information in the labeling

- Examples of FDA boxed warnings related to risk for adverse response:
  - **Abacavir (HIV):** Recommend *HLA-B*57:01* screening before use. Do not use if positive.
  - **Carbamazepine (seizure):** Recommend *HLA-B*15:02* screening before use (Asian ancestry). If positive, do not use unless benefits > risks
  - **Clopidogrel (cardio):** Consider use of another platelet P2Y12 inhibitor in CYP2C19 poor metabolizers. Genetic testing available.
  - **Codeine (pain):** respiratory depression and death of children, most cases after tonsillectomy and/or adenoidectomy; some had evidence of ultrarapid metabolism of codeine.
  - **Tramadol (pain):** respiratory depression and death of children, some cases after tonsillectomy and/or adenoidectomy; at least one had evidence of ultrarapid metabolism of tramadol.

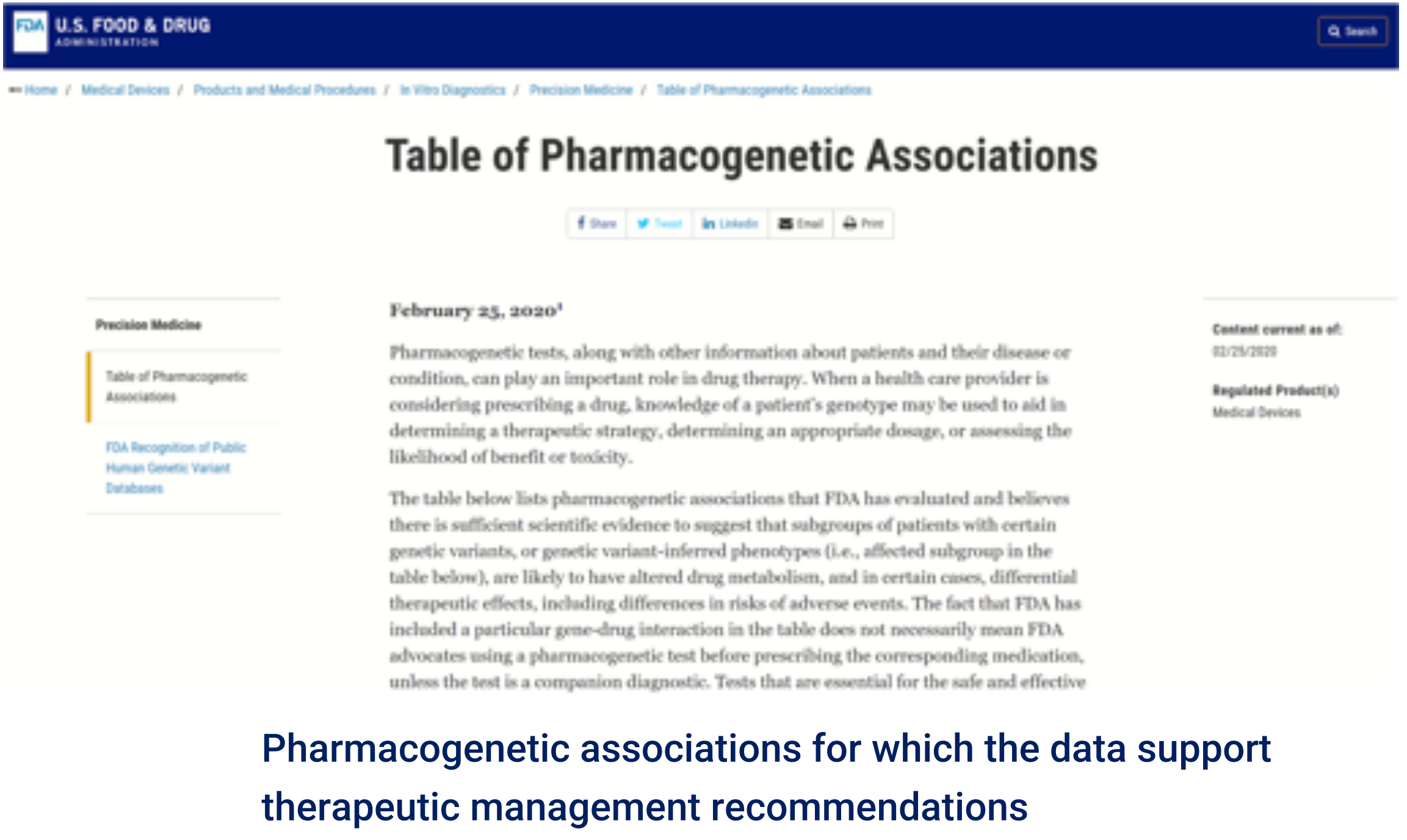


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Table of Pharmacogenetic Associations

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Precision Medicine

Table of Pharmacogenetic Associations

FDA Recognition of Public Human Genetic Variant Databases

February 25, 2020[1]

Pharmacogenetic tests, along with other information about patients and their disease or condition, can play an important role in drug therapy. When a health care provider is considering prescribing a drug, knowledge of a patient's genotype may be used to aid in determining a therapeutic strategy, determining an appropriate dosage, or assessing the likelihood of benefit or toxicity.

The table below lists pharmacogenetic associations that FDA has evaluated and believes there is sufficient scientific evidence to suggest that subgroups of patients with certain genetic variants, or genetic variant-inferred phenotypes (i.e., affected subgroup in the table below), are likely to have altered drug metabolism, and in certain cases, differential therapeutic effects, including differences in risks of adverse events. The fact that FDA has included a particular gene-drug interaction in the table does not necessarily mean FDA advocates using a pharmacogenetic test before prescribing the corresponding medication, unless the test is a companion diagnostic. Tests that are essential for the safe and effective

Content current as of:
02/25/2020

Regulated Product(s)
Medical Devices

Pharmacogenetic associations for which the data support therapeutic management recommendations

Section 1: Pharmacogenetic Associations for which the Data Support Therapeutic Management Recommendations

Drug	Gene	Affected Subgroups+	Description of Gene-Drug Interaction
Abacavir	HLA-B	*57:01 allele positive	Results in higher adverse reaction risk (hypersensitivity reactions). Do not use abacavir in patients positive for HLA-B*57:01.
Amifampridine	NAT2	poor metabolizers	Results in higher systemic concentrations and higher adverse reaction risk. Use lowest recommended starting dosage and monitor for adverse reactions. Refer to FDA labeling for specific dosing recommendations.
Amifampridine Phosphate	NAT2	poor metabolizers	Results in higher systemic concentrations. Use lowest recommended starting dosage (15 mg/day) and monitor for adverse reactions.
Amphetamine	CYP2D6	poor metabolizers	May affect systemic concentrations and adverse reaction risk. Consider lower starting dosage or use alternative agent.
Aripiprazole	CYP2D6	poor metabolizers	Results in higher systemic concentrations and higher adverse reaction risk. Dosage adjustment is recommended. Refer to FDA labeling for specific dosing recommendations.
Aripiprazole Lauroxil	CYP2D6	poor metabolizers	Results in higher systemic concentrations. Dosage adjustment is recommended. Refer to FDA labeling for specific dosing recommendations.
Atomoxetine	CYP2D6	poor metabolizers	Results in higher systemic concentrations and higher adverse reaction risk. Adjust titration interval and increase dosage if tolerated. Refer to FDA labeling for specific dosing recommendations.

PharmGKB

- Pharmacogenomics knowledge resource that provides clinical information, including dosing guidelines and drug labels, potentially clinically-actionable gene-drug associations and genotype-phenotype relationships

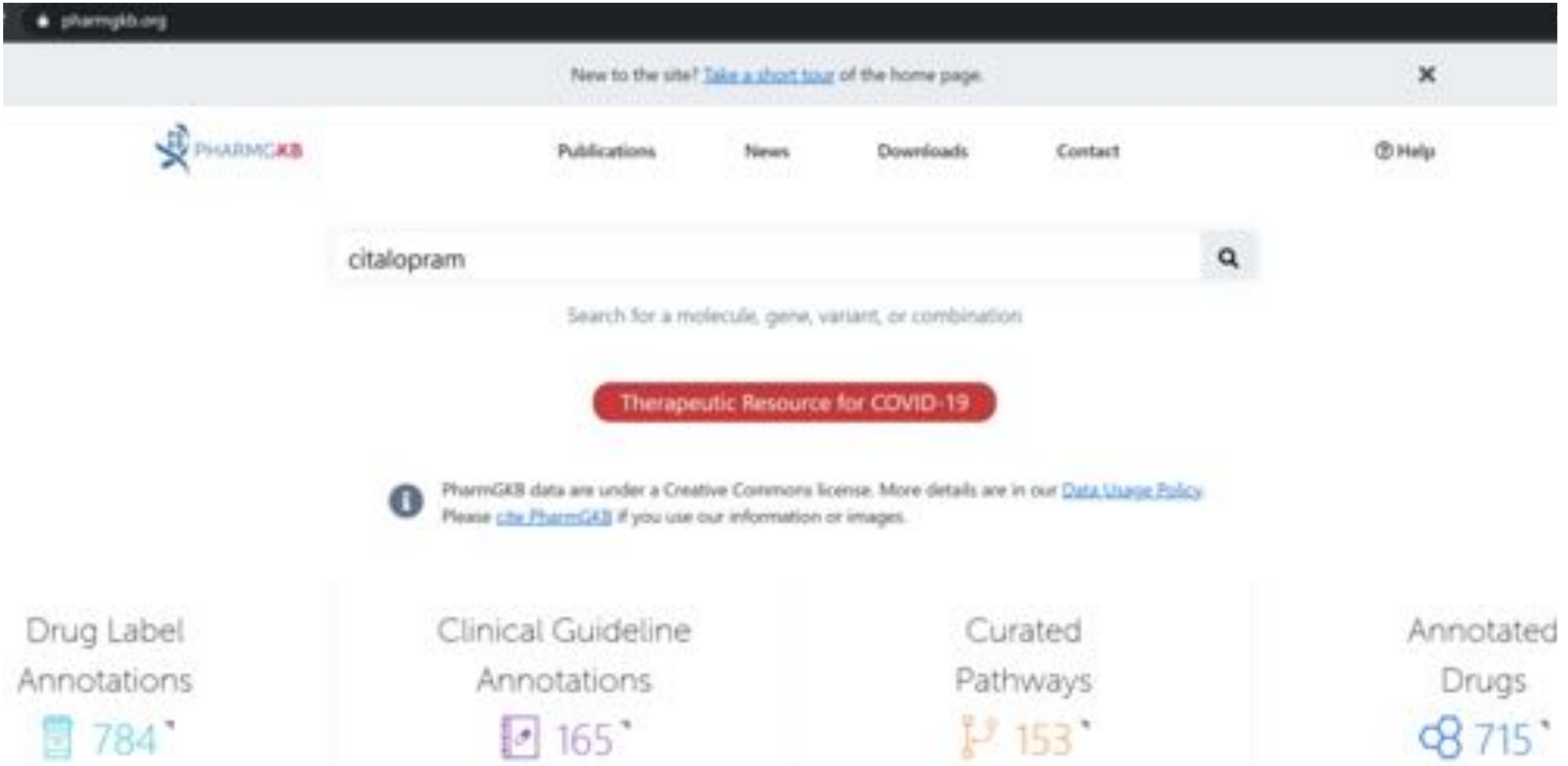
- Level of Evidence
  - Level 1A - CPIC or medical society-endorsed PGx guideline
  - Level 1B - preponderance of evidence shows an association
  - Level 2A - qualifies for level 2B where the variant is within a Very Important Pharmacogene
  - Level 2B - moderate evidence of an association
  - Level 3 - single significant not yet replicated study or multiple studies but lacking clear evidence of an association
  - Level 4 - case report, non-significant study or in vitro, molecular, or functional assay evidence only

https://www.pharmgkb.org/

Annotation for *OPRM1* and opioids - Level 2B

Allele	Phenotype
AA	Patients with the AA genotype may have decreased opioid dose requirements as compared to patients with the AG or GG genotypes. However, some studies did not find an association between this variant and opioid dosing. Other genetic or clinical factors may also affect a patient's opioid dose requirements.
AG	Patients with the AG genotype may have increased opioid dose requirements as compared to patients with the AA genotype, but decreased opioid dose requirements as compared to patients with the GG genotype. However, some studies did not find an association between this variant and opioid dosing. Other genetic or clinical factors may also affect a patient's opioid dose requirements.
GG	Patients with the GG genotype may have increased opioid dose requirements as compared to patients with the AA or AG genotypes. However, some studies did not find an association between this variant and opioid dosing. Other genetic or clinical factors may also affect a patient's opioid dose requirements.

https://www.pharmgkb.org/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Drug Label Annotations
784
Clinical Guideline Annotations
165
Curated Pathways
153
Annotated Drugs
715

Prescribing Info

Clinical Guideline Annotations

	SOURCE	GENES	NAME
Read Now	Clinical Pharmacogenetics Implementation Consortium	CYP2C19	Annotation of CPIC Guideline for citalopram escitalopram and CYP2C19
Read Now	Dutch Pharmacogenetics Working Group	CYP2C19	Annotation of DPWG Guideline for citalopram and CYP2C19
Read Now	Dutch Pharmacogenetics Working Group	CYP2D6	Annotation of DPWG Guideline for citalopram escitalopram and CYP2D6 *No recommendation*

Label Annotations

Annotation of FDA Label for citalopram

Excerpt from the citalopram (CELEXA) drug label:

> In CYP2C19 poor metabolizers, citalopram steady state Cmax and AUC was increased by 68% and 107%, respectively. Celexa 20 mg/day is the maximum recommended dose in CYP2C19 poor metabolizers due to the risk of QT prolongation.

Continue reading Annotation of FDA Label for citalopram

Drug Label Annotations

PharmGKB annotates drug labels containing pharmacogenetic information approved by the US Food and Drug Administration (FDA), European Medicines Agency (EMA), Swiss Agency of Therapeutic Products (Swissmedic), Pharmaceuticals and Medical Devices Agency Japan (PMDA) and Health Canada (Santé Canada) (HCSC).

PharmGKB annotations provide a brief summary of the PGx in the label, an excerpt from the label and a downloadable highlighted label PDF file. The "Prescribing" section of the annotation captures guidance from the label for patients with a particular genotype/metabolizer phenotype, if it exists. The "PGx Level" tag ("Testing required", "Testing Recommended", "Actionable PGx" and "Informative PGx) is the PharmGKB interpretation of the level of action implied in each label. Other tags indicate if the label provides dosing information or states that a drug is either indicated or contraindicated ("Alternate Drug") based on genotype/metabolizer phenotype.

See the legend for more information about drug label sources, which labels are selected for annotation, PGx Levels and the tags described above. We welcome any information regarding drug labels containing PGx information approved by the FDA, EMA, Swissmedic, PMDA, HCSC or other Medicine Agencies around the world - please contact feedback.

	PGX LEVEL	SOURCE	GENES	TITLE
Read Now	Actionable PGx	U.S. Food and Drug Administration	CYP2C19	Annotation of FDA Label for citalopram and CYP2C19
Read Now	Informative PGx	U.S. Food and Drug Administration	CYP2D6	Annotation of FDA Label for citalopram and CYP2D6

Patient Case

- 35 yo female with anxiety and depression
- No other significant past medical history
- Current meds: Multivitamin daily, Vitamin D 50,000 units weekly
- She has tried several antidepressants
  - Citalopram (Celexa) 40mg - didn’t feel like it helped her symptoms
  - Escitalopram (Lexapro) 20mg - didn’t feel like it helped her symptoms
  - Bupropion (Wellbutrin) SR 150mg daily – worsened her anxiety
- Her PCP is considering fluoxetine (Prozac) or venlafaxine (Effexor) for next drug trial

Guidance for dosing based on PGx information

- **CPIC guidelines**
  - SSRIs: citalopram, escitalopram, fluvoxamine, paroxetine
  - TCAs: amitriptyline and nortriptyline
- **Behavioral health medications with PGx information in FDA labeling**
  - SSRIs: citalopram, escitalopram, fluvoxamine
  - TCAs: amitriptyline, clomipramine, desipramine, doxepin, imipramine, nortriptyline, trimipramine
  - Other antidepressants: vortioxetine
  - ADHD: atomoxetine
  - Antipsychotics: aripiprazole, brexpiprazole, perphenazine, thioridazine, clozapine, iloperidone, pimozide

Patient Case

- She decides to pursue PGx testing after discussing with her PCP
- Pertinent results:
  - CYP2C19 Ultrarapid metabolizer
  - CYP2D6 Normal metabolizer
  - CYP2C9 Normal metabolizer
  - CYP1A2 Normal metabolizer
  - SLCO1B1 Normal Function

Patient Case

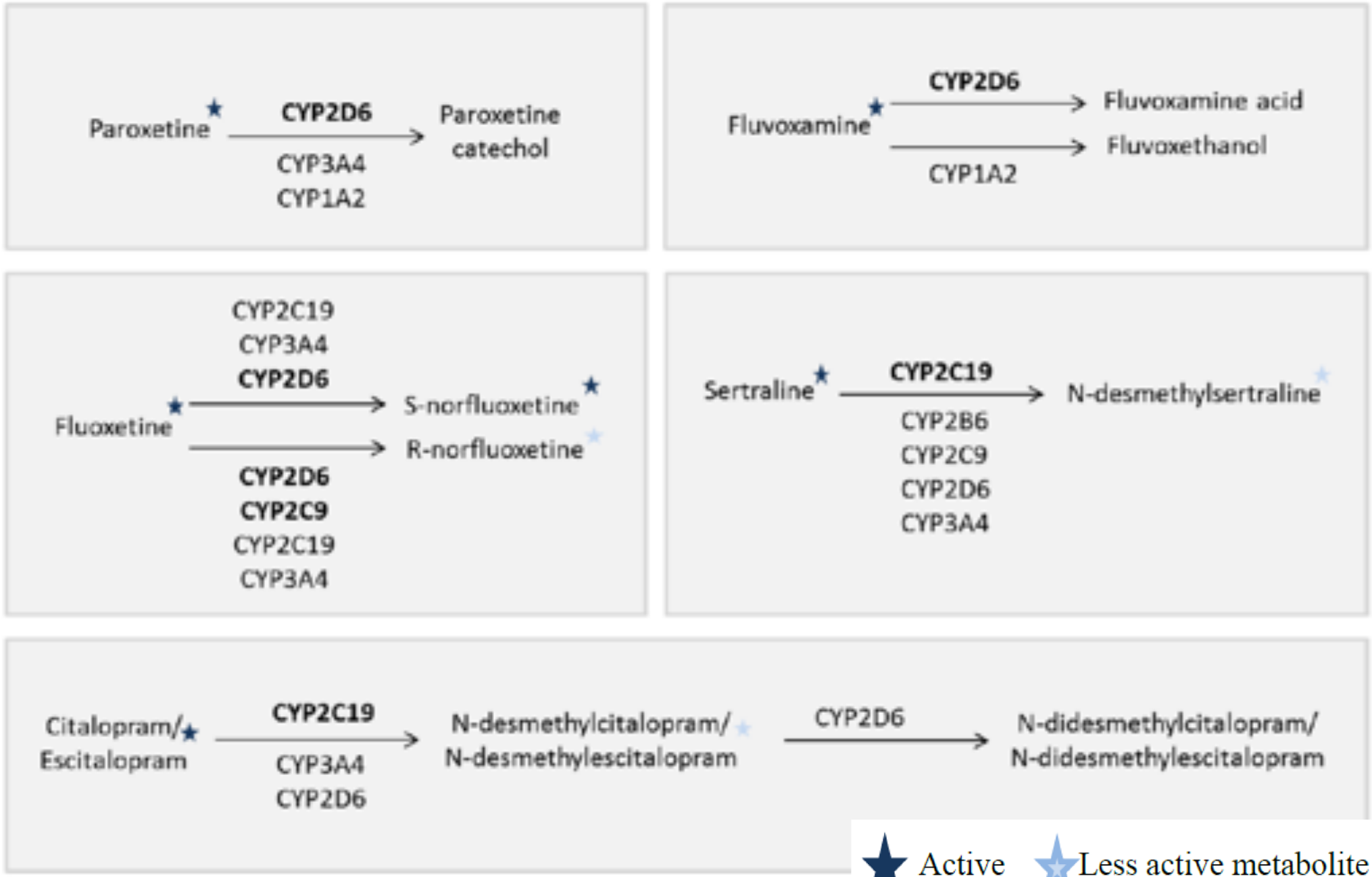
- She decides to pursue PGx testing after discussing with her PCP
- Pertinent results:
  - *CYP2C19 Ultrarapid metabolizer*
  - CYP2D6 Normal metabolizer
  - CYP2C9 Normal metabolizer
  - CYP1A2 Normal metabolizer
  - SLCO1B1 Normal Function

CPIC guideline recommendations

Table 3a Dosing recommendations for citalopram and escitalopram based on CYP2C19 phenotype

Phenotype	Implication	Therapeutic recommendation	Classification of recommendation[a]
CYP2C19 Ultrarapid metabolizer	Increased metabolism when compared to extensive metabolizers. Lower plasma concentrations will increase probability of pharmacotherapy failure.	Consider an alternative drug not predominantly metabolized by CYP2C19.[b]	Moderate
CYP2C19 Extensive metabolizer	Normal metabolism	Initiate therapy with recommended starting dose.	Strong
CYP2C19 Intermediate metabolizer	Reduced metabolism when compared to extensive metabolizers.	Initiate therapy with recommended starting dose.	Strong
CYP2C19 Poor metabolizer	Greatly reduced metabolism when compared to extensive metabolizers. Higher plasma concentrations may increase the probability of side effects.	Consider a 50% reduction[c,d] of recommended starting dose and titrate to response or select alternative drug not predominantly metabolized by CYP2C19.[b]	Moderate

https://cpicpgx.org/content/guideline/publication/SSRI/2015/25974703.pdf


Paroxetine
CYP2D6
CYP3A4
CYP1A2
Paroxetine catechol
Fluvoxamine
CYP2D6
Fluvoxamine acid
Fluvoxethanol
CYP1A2
Fluoxetine
CYP2C19
CYP3A4
CYP2D6
S-norfluoxetine
R-norfluoxetine
CYP2D6
CYP2C9
CYP2C19
CYP3A4
Sertraline
CYP2C19
CYP2B6
CYP2C9
CYP2D6
CYP3A4
N-desmethylsertraline
Citalopram/
Escitalopram
CYP2C19
CYP3A4
CYP2D6
N-desmethylcitalopram/
N-desmethylescitalopram
CYP2D6
N-didesmethylcitalopram/
N-didesmethylescitalopram
Active
Less active metabolite

Recommendations

- Patient is a CYP2C19 ultrarapid metabolizer which is consistent with higher than average CYP2C19 enzymatic activity
- If SSRI use is warranted, consider alternative drugs not predominantly metabolized by CYP2C19 such as fluoxetine (Prozac) and paroxetine (Paxil) (major substrates of CYP2D6). Would need to think through side effects of of these drugs.
- No issue from a metabolism standpoint for fluoxetine (Prozac) or venlafaxine (Effexor)
- Future drugs which could impacted: voriconazole (antifungal) and PPIs (omeprazole, lansoprazole, pantoprazole)

Limitations of using PGx in clinical care

- Not a crystal ball - can’t predict exactly which medication will work
- Can’t predict specific adverse drug reactions
- Genetics only one piece of the puzzle, many other factors affect drug response
  - Diet, disease states, sex, age, weight, drug–drug interactions
- Complex interaction between pharmacokinetic and pharmacodynamic genetic variations
  - Need studies looking at numerous PK/PD variations
  - Drug-drug-gene interactions
- Doesn’t apply to all medications, some have more evidence than others

Take home points

- PGx focuses on how genetic/genomic variations can affect drug response
  - Several different ways - PK/PD/ immune-mediated
- Goal is to optimize drug efficacy and minimize adverse effects
  - Patients have more confidence, fewer drug trials, less ADRs
  - Many caveats, important to set proper expectations if possible
- Increasing amounts of recommendations from several sources
  - FDA, CPIC, PharmGKB
- One piece of a puzzle!

Genetic Counseling for PGx

Rachel Mills, MS, CGC

Take-Home

Counseling for pharmacogenetics is not much different than counseling for any other indication.

Think about a time that you received a referral for an unfamiliar indication.

What did you do?


Resources
& Skillset

Pharmacogenetics

Cancer

Risk information

- Drug response is multi-factorial
  - Genetics
  - Environment
  - Drug-drug interaction
- Highlight how genetic changes may impact drug selection and dosage

- Cancer is multi-factorial
  - Genetics
  - Environmental factors
  - “Two-hit hypothesis”
- Review additional recommended screening and cascade testing

Pharmacogenetics

All specialties

Post-test Counseling

- High rate of consumer-facing testing
- Contracting
  - What is known?
  - Why did you order testing?
- [Re-]setting expectations
  - Drug response is multi-factorial
  - Not all medications are covered by the test or impacted by pharmacogenes
  - Need for clinical grade testing

- Other providers order testing then refer to genetics
- Contracting
  - What is known about the test and referral?
  - Why did you/your provider order testing?
- [Re-]setting expectations
  - Multi-factorial nature of some conditions
  - Need for reordering testing (if incorrect test was ordered)

Pharmacogenetics

Cancer

Scope of Practice

- PGx testing informs what medications or doses to use
  - Which drug/dose are likely to be effective and are unlikely to cause severe side effects
- We do not recommend drug changes to the patient
- We help them understand results and consider questions to ask their prescribing provider about the impact of PGx results on drug/dose selection
- We may provide recommendations to their provider but never directly to the patient

- Genetic testing for hereditary cancer can inform what type of treatment a patient undergoes (i.e. surgery vs. chemo + radiation)
- We do not determine what treatment plan is best for the patient
- We help them understand results and consider questions to ask their oncologist about the impact of genetic testing results on their treatment plan

Pharmacogenetics

Cancer, Prenatal

Following Guidelines

- Guidelines regarding use of PGx results
  - **CPIC** https://cpicpgx.org/guidelines/
  - **FDA** https://www.fda.gov/medical-devices/precision-medicine/table-pharmacogenetic-associations

- Various practice guidelines
  - **NCCN Guidelines** https://www.nccn.org/guidelines/category_1
  - **ACOG Practice Bulletins** related to genetics https://www.acog.org/clinical/clinical-guidance/practice-bulletin

Pharmacogenetics

Sequencing

Prioritizing Results

- Large multi-gene panels
- Consider what is most relevant to the patient
  - What is relevant now
    - Results with CPIC/FDA guidelines that impact current medications
  - What is relevant in the future
    - Results with CPIC/FDA guidelines indicating a change in standard prescribing/dosing
  - What may not be relevant
    - Results in genes that do not have clinical utility/guidelines

- Possibility for multiple results including those that are diagnostic and those that are of uncertain significance
- Consider what is most relevant to the patient
  - What is relevant now
    - Findings related to the indication; results that explain symptoms
  - What is relevant in the future
    - Results in “ACMG 59” genes
  - What may not be relevant
    - VUS

Summary

01. Genetic Risk Information

As many types of genetic tests for cancer, PGx provides risk information. And like cancer, drug response is multi-factorial, impacted by genetics as well as environment.

02. Post-test Counseling

Counseling for PGx often occurs after results are available, like other genetics settings. Counseling strategies focus on contracting, [re-]setting expectations, and identifying next steps.

03. Scope of Practice

GCs are limited by our Scope of Practice. We don't recommend drug or dose changes following PGx testing, just as we don't determine cancer treatment after testing for hereditary cancer.

04. Following Guidelines

Like all areas of genetics care, PGx follows set guidelines related to treatment and care. CPIC & FDA are to PGx as NCCN is to cancer as ACOG is to prenatal.

05. Prioritizing Results

Returning PGx results is similar to returning exome/genome results. You may not discuss every single finding. Instead, you prioritize information that is relevant now, and then discuss what may be relevant in the future.

Resources

- How do I decide what testing platform to use?
  - Vo, T. T., Bell, G. C., Owusu Obeng, A., Hicks, J. K., & Dunnenberger, H. M. (2017). Pharmacogenomics Implementation: Considerations for Selecting a Reference Laboratory. Pharmacotherapy, 37(9), 1014–1022. https://doi.org/10.1002/phar.1985
- Can I get some more details about counseling for PGx with specific gene/drug clinical examples?
  - Zierhut, H. A., Campbell, C. A., Mitchell, A. G., Lemke, A. A., Mills, R., & Bishop, J. R. (2017). Collaborative Counseling Considerations for Pharmacogenomic Tests. *Pharmacotherapy, 37*(9), 990–999. https://doi.org/10.1002/phar.1980
- My physician colleagues are asking me about this – how should they approach PGx testing?
  - Mills, R., Voora, D., Peyser, B., & Haga, S. B. (2013). Delivering pharmacogenetic testing in a primary care setting. *Pharmacogenomics and personalized medicine, 6*, 105–112. https://doi.org/10.2147/PGPM.S50598
- How can I spend another 3.5 hours learning about PGx from genetic counselors?
  - NSGC Online Course: “Integrating PGx Testing into Clinical Practice” https://www.nsgc.org/Education-and-Events/Online-Education-Center/Online-Education-Inventory

Thank you!

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