THE FUTURE PERSPECTIVES AND DEVELOPMENT IN PHARMACOGENETICS

Dr. Zainab G. Al-Jassim
Ph.D. Pharmacology
College of Medicine
Baghdad University

Definitions..

The terms 'Pharmacogenetics' & 'Pharmacogenomics' has conventionally been defined as the study of how a person's genetic make-up affects their response (efficacy and/or safety) to a drug.

This field combines pharmacology (the science of drugs) and genomics (the study of genes and their functions) to develop effective, safe medications that can be prescribed based on a person's genetic makeup.

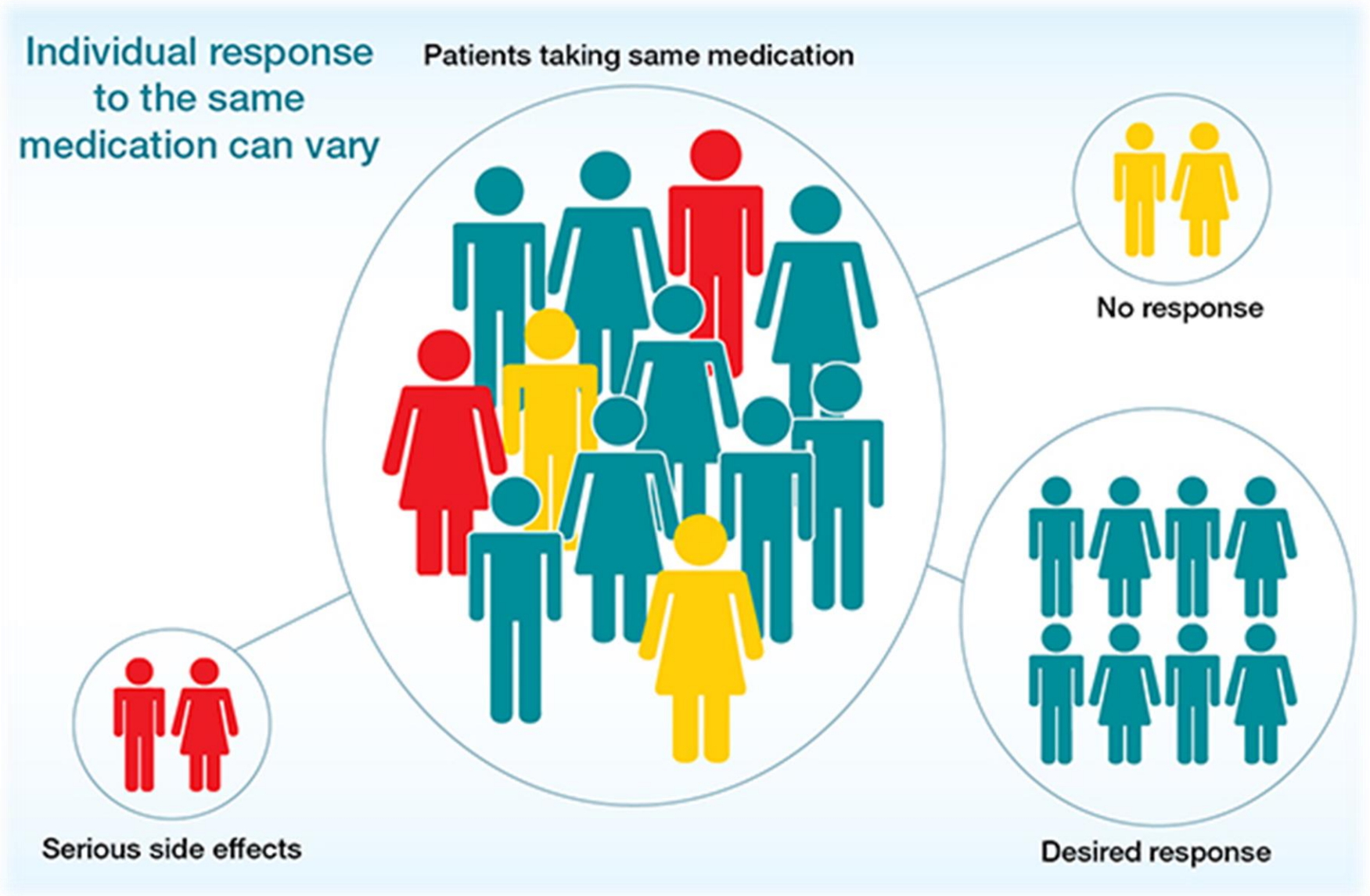
Pharmacogenetics *Vs* Pharmacogenomics

Pharmacogenetics **is the study of how variation in a single gene can impact on variability in the body's response to medicines.**

Pharmacogenomics is the study of how a patient's genome can influence how they respond to medicines.

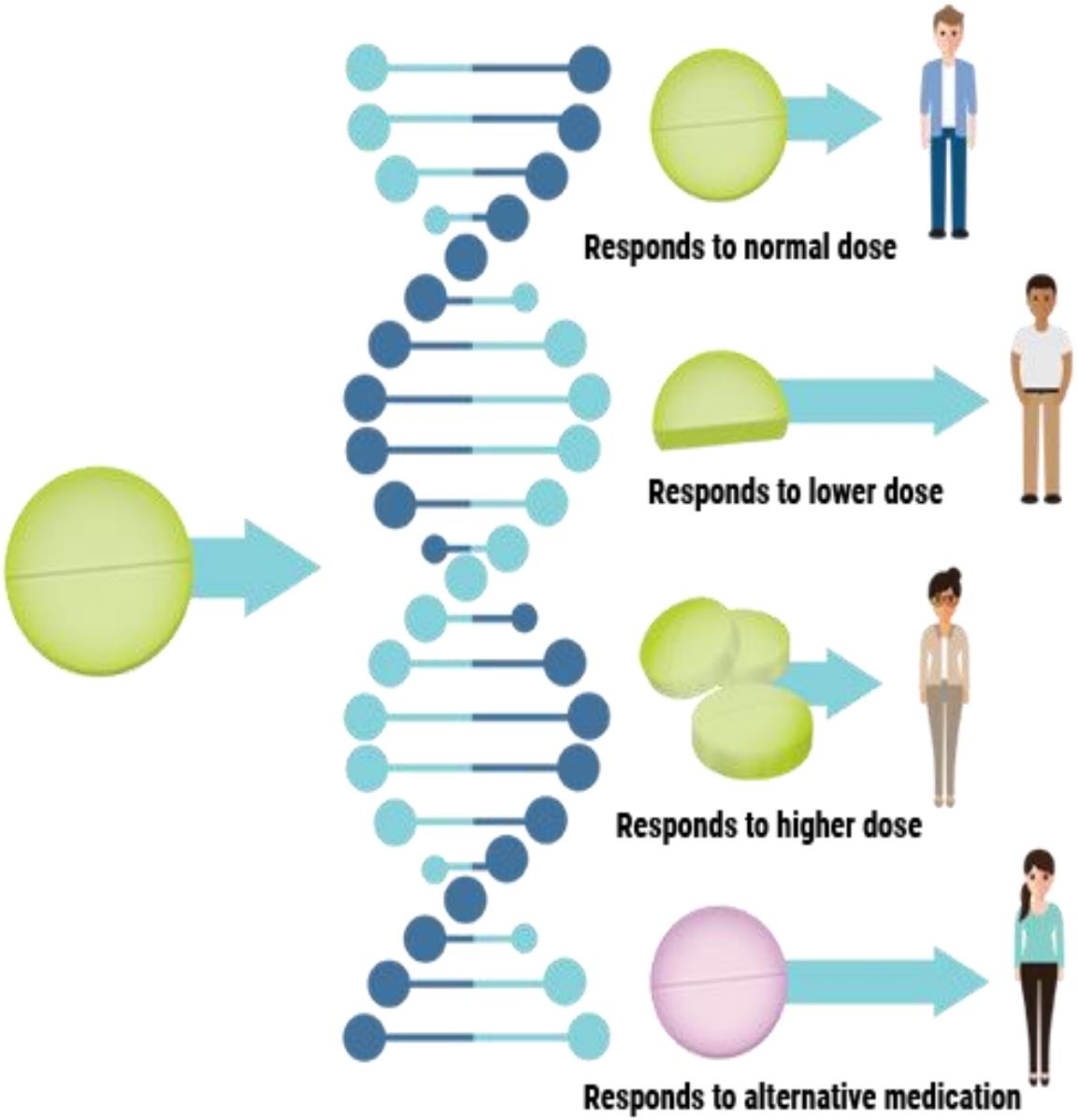
Pharmacogenetics/ Pharmacogenomics

How genes affect the way our body responds to certain medicines..



Differences in some genes that can affect how the body **uses** and **breaks down** medicines can be the reason why some people may benefit from a certain medicine while others may not benefit at all.

Genes can also be the reason why some people have serious **side effects** from a medicine and others have none.



Responses to all the drugs virtually vary between individuals because of some factors:

- Intrinsic factors which means age, health and etc.
- Extrinsic factors which means the diet, lifestyle, the use of concomitant drugs that may affect drug Pharmacokinetic parameters.
- Genes relevant to the drug's Pharmacokinetic and Pharmacodynamic.
- Genes that influence ADRs susceptibility.

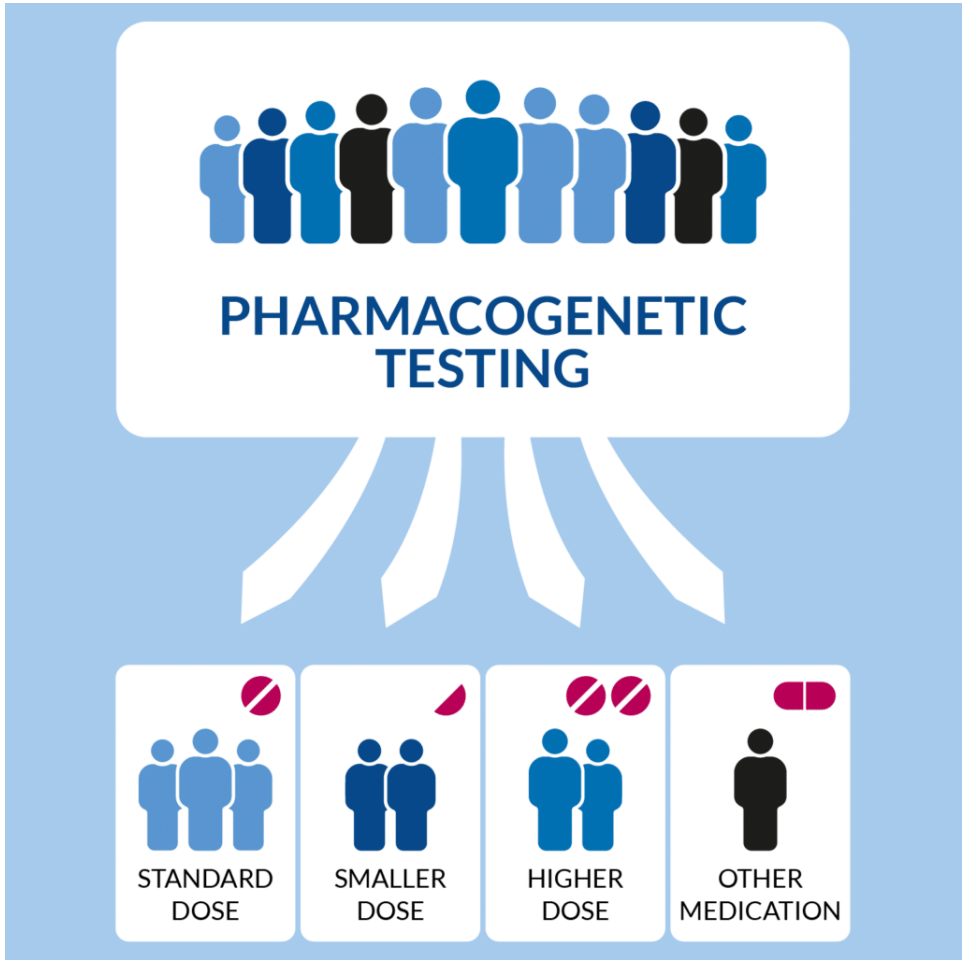
- **Efficacy** rates of different drugs have been reported to vary from ⇨ **25%** to **80%.**

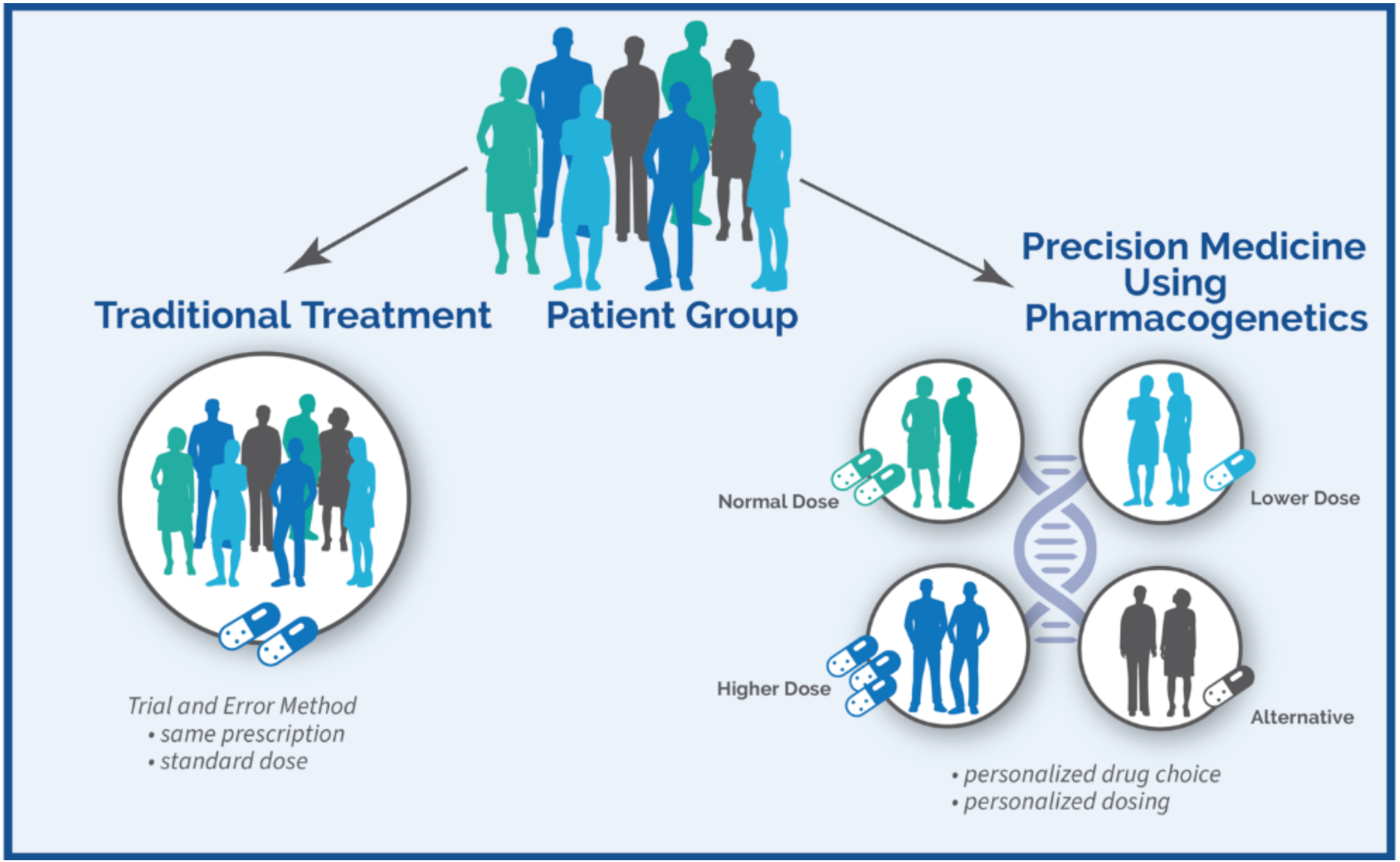
- In 2003, Allen Roses *(clinical neurologist)* famously said: "The vast majority of drugs — more than 90% — only work in 30 or 50% of the people" 😐.

- A more recent analysis showed that for the **ten** highest-grossing drugs in the USA, between 3 and 24 individuals **failed** to show a response.

- In terms of drug safety, adverse drug reactions (ADRs) account for about **6.5%** of hospital admissions in adults, increasing to **>15%** when focusing on people with multimorbidity. Furthermore, ADRs affect about **15%** of people in hospital.

- **From a clinical perspective, the aim of pharmacogenomics is to move away from our current 'one drug fits all' or 'one dose fits all' strategy to a more personalized choice and dose of drug that is relevant for the individual patient's needs.**



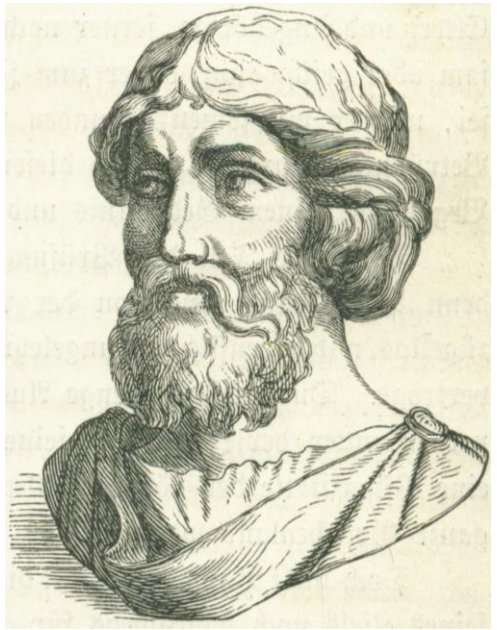

Patient Group
Traditional Treatment
Trial and Error Method
• same prescription
• standard dose
Precision Medicine
Using
Pharmacogenetics
Normal Dose
Lower Dose
Higher Dose
Alternative
• personalized drug choice
• personalized dosing

History ✍

Pythagoras first recognized the concept of Pharmacogenomics around 510 BC. When he made a simple connection among the dangers of the fava bean ingestion with hemolytic anemia and oxidative stress. Then this documentation was later validated and credited to deficiency of G6PD in the 1950s. Friedrich Vogel of Heidelberg first coined the term pharmacogenetic in 1959.

The first FDA approval of a pharmacogenetic test for alleles in CYP2D6 and CYP2C19 was in 2005. These and other discoveries that led to the term pharmacogenomics.

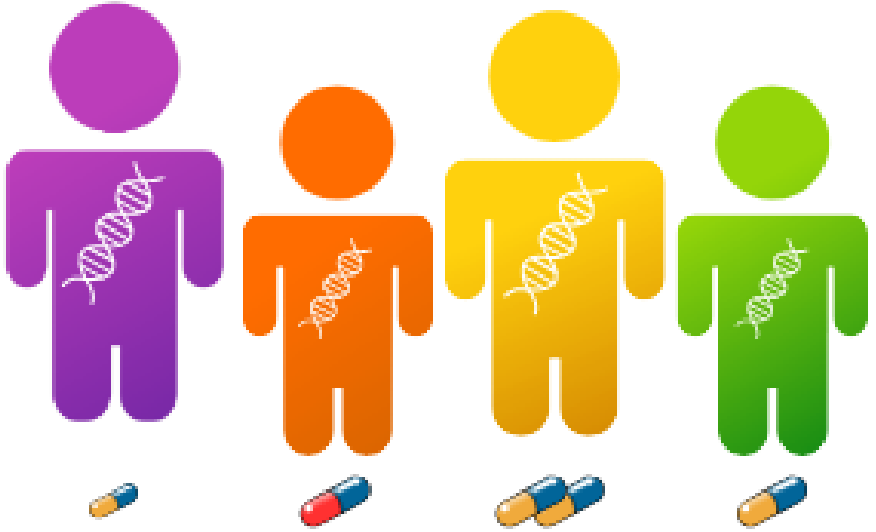
Today, there are cumulative number of genes that the polymorphisms are identified and that are related to variable drug response. Genome-wide analysis is mainly helping to identify previously unpredictable new genes who are associated with disease and drug response.



Genetic Differences in Drug Metabolizing Enzymes

There are numerous known genes which are mostly responsible for alterations in drug metabolism and response.

- **Cytochrome P450s**
- **TPMT**
- **VKORC1**



Cytochrome P450s: ***The most predominant drug-metabolizing enzymes are the Cytochrome P450 enzymes.***

CYP2D6:
CYP2D6 is the most eminent and widely studied CYP gene. This gene is of an excellent interest due to its extremely polymorphic nature, and its participation within the high range of medication metabolisms.

CYP3A4 & CYP3A5:
The CYP3A family most profusely found in the liver. CYP3A4 accounting 29% of the liver content.

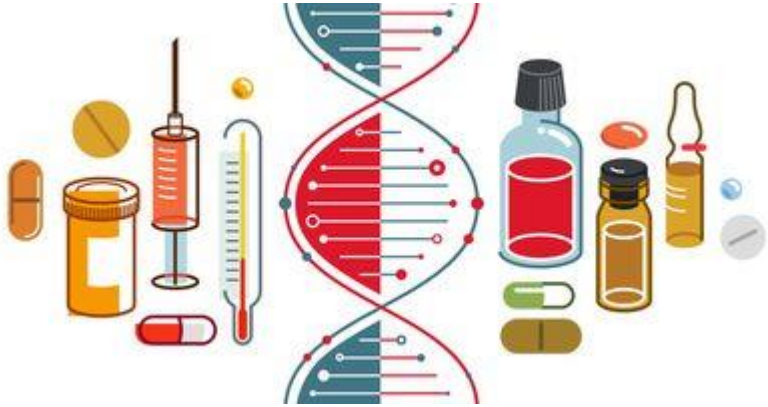
CYP2B6:
CYP2B6 plays a very vital role in the metabolism of drugs as well as the anti-HIV drug efavirenz, the anti-malarial artemisinin, the antidepressants, the anti-cancer drug cyclophosphamide etc. This can be really a polymorphic catalyst with the variant CYP2B6.

CYP2C9:
CYP2C9 constitutes the majority of the CYP2C subfamily, it is involved in the metabolism of about 10% of all the drugs, which also include the medications with narrow therapeutic windows such as warfarin and tolbutamide.

CYP2C19:
CYP2C19 is that the second most generally studied and a well understood gene in pharmacogenomics. For CYP2C19 over 28 genetic variants have been recognized.

TPMT:

Thiopurine methyltransferase (TPMT) catalyzes the S-methylation of thiopurines, thus regulating the balance between cytotoxic thioguanine nucleotide and inactive metabolites in the hematopoietic cells. TPMT is extremely involved in 6-MP metabolism and TMPT activity and genotype is known to affect the risk of toxicity. Extreme levels of 6-MP may cause myelosuppression and myelotoxicity.



VKORC1:

Vitamin K epoxide reductase complex subunit 1 which is responsible for pharmacodynamics of warfarin. VKORC1 along with CYP2C9 are very much useful for recognizing the risk of bleeding during warfarin administration.

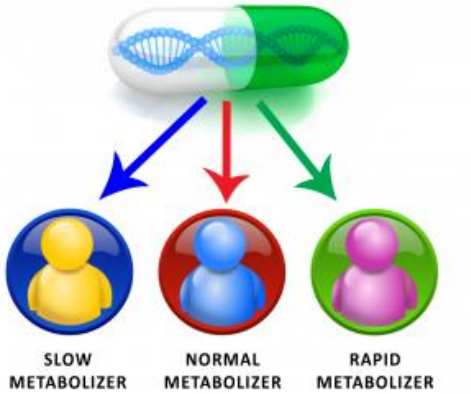
Genotypes of the patient are usually classified into the following predicted phenotypes according to the metabolic rate:

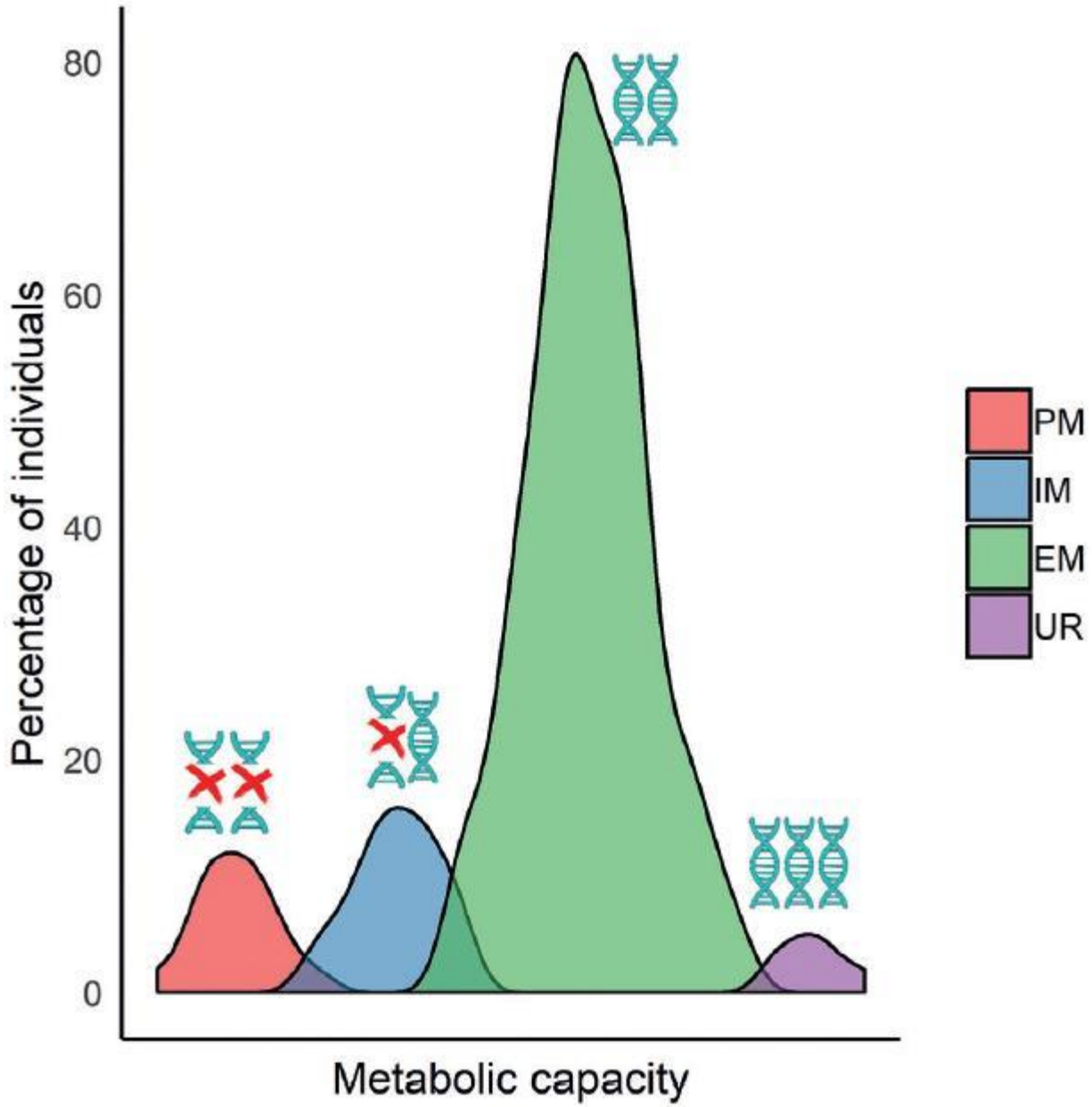
- **UM- Ultra-rapid metabolizer: the patients with substantially increased metabolic activity.**

- **EM- Extensive metabolizer: the normal metabolic activity.**

- **IM- Intermediate metabolizer: the patients with reduced metabolic activity**

- **PM- Poor metabolizer: the patients with little to no functional metabolic activity.**





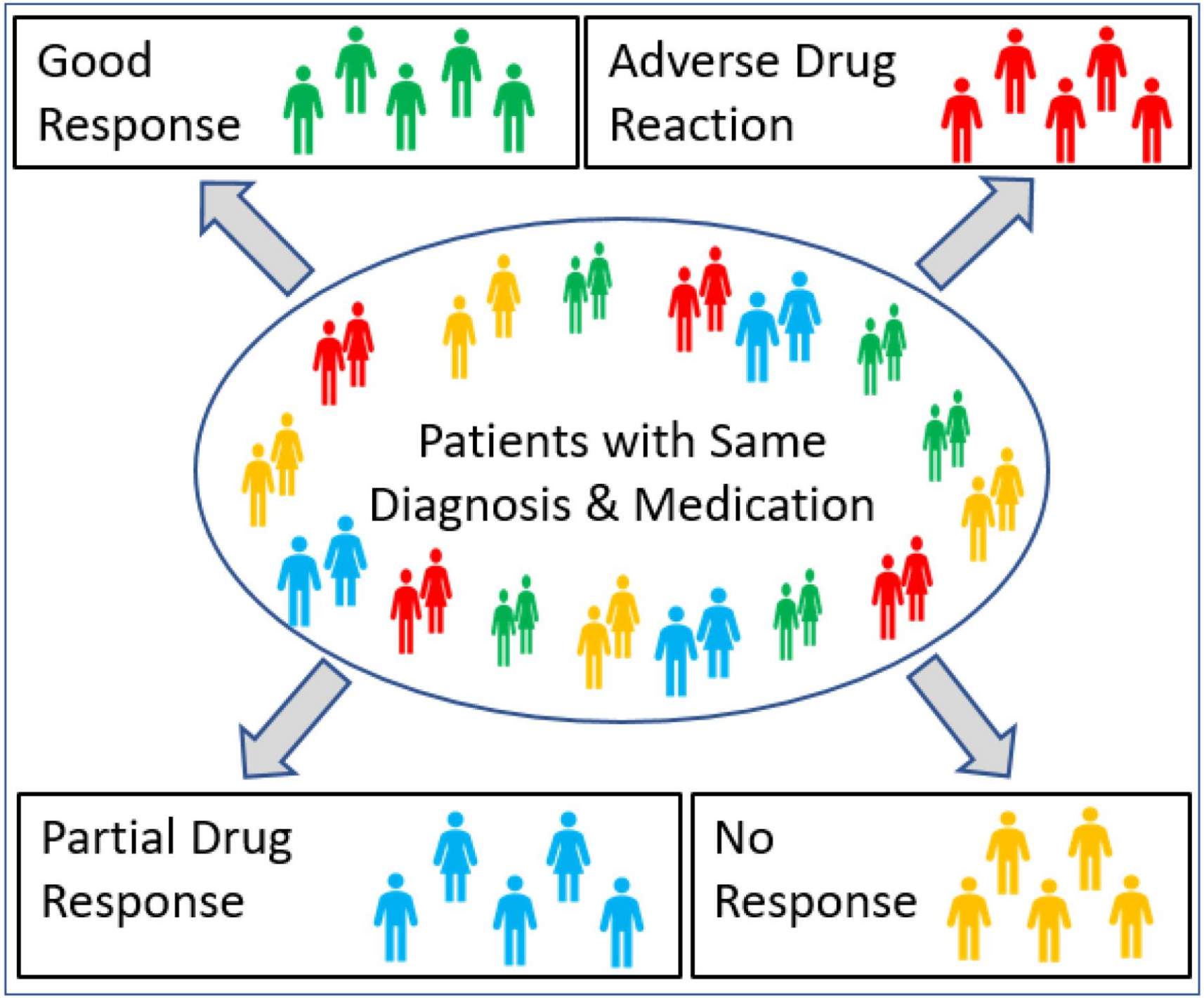
Metabolism capacity (phenotypes) population distribution

https://www.scielo.org.mx/scielo.php?script=sci_arttext&pid=S0034-83762020000500271

Most studied PGx genes classified by their main drug pathway:

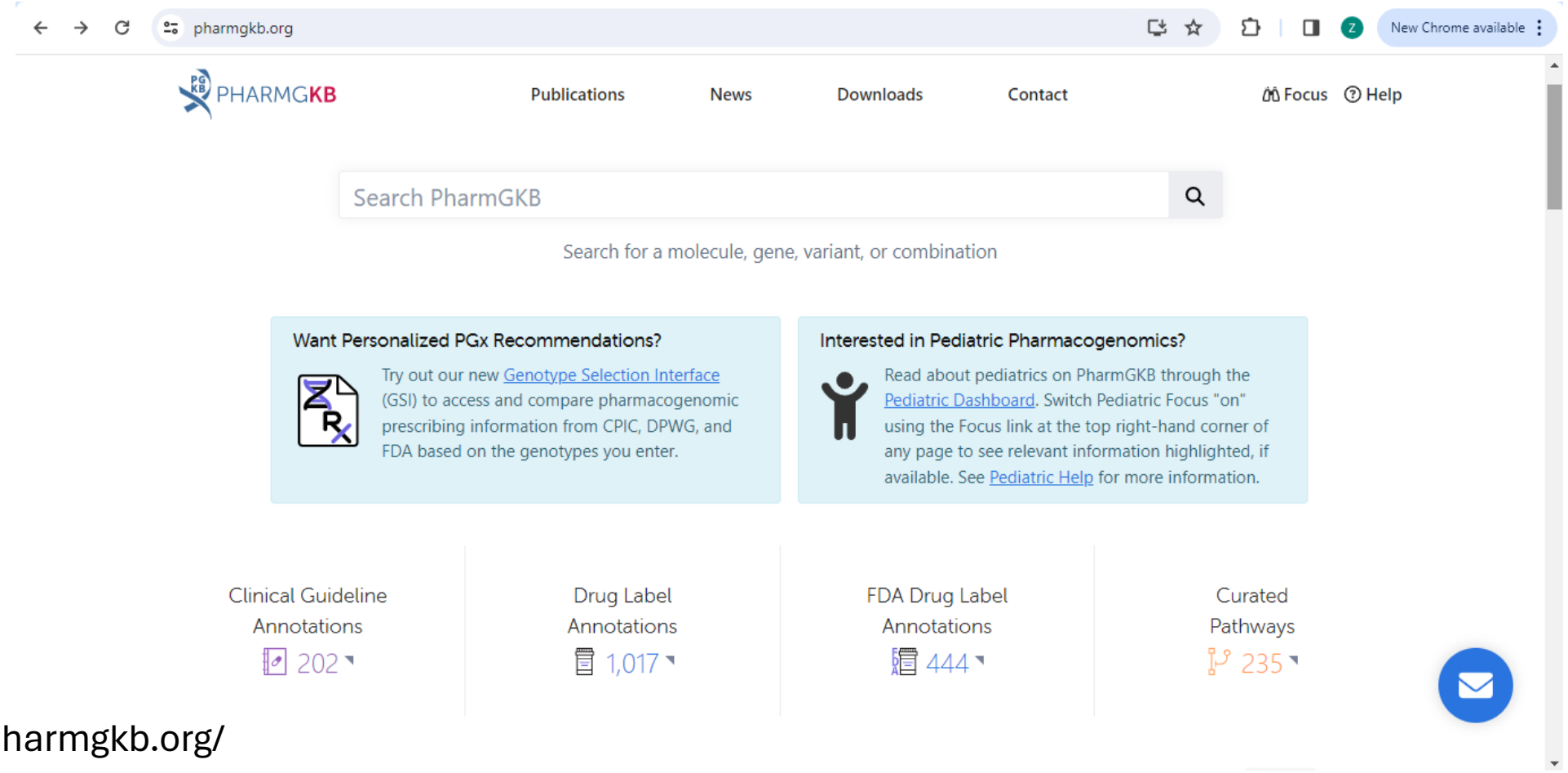
Pharmacokinetics			Pharmacodynamics/ADRs		
DPYD	*CES2*	*SLC47A1*	*RYR1*	*GRK4*	*DRD2*
ABCA1	*TPMT*	*CYP2C9*	*EGFR*	*VDR*	*NPR1*
COMT	*CYP3A4*	*NAT2*	*ESR1*	*ACE*	*DRD1*
ABCG1	*CYP2B6*	*ABCG2*	*RYR2*	*NR3C2*	*PTGIS*
UGT1A1	*POR*	*CYP1A2*	*DBH*	*HSD11B2*	*APOA1*
CYP2D6	*SLC22A1*	*G6PD*	*PEAR1*	*CRHR1*	*ADRB2*
ABCB1	*CYP3A5*	*MAOA*	*CACNA1S*	*ARID5B*	*VKORC1*

PGx: pharmacogenomics; ADRs: adverse drug reactions.


Good Response
Adverse Drug Reaction
Patients with Same Diagnosis & Medication
Partial Drug Response
No Response

Recommended PGx Data Bases

- ✓ **The Pharmacogenomics Knowledge Base (PharmGKB) is an organization created over 20 years ago and is one of the largest resources of PGx data. Its website (www.pharmgkb.org) publishes PGx information, including drug-gene pairs, phenotypes, pathways, dosing guidelines, drug label annotations, and variant and clinical guideline annotations.**

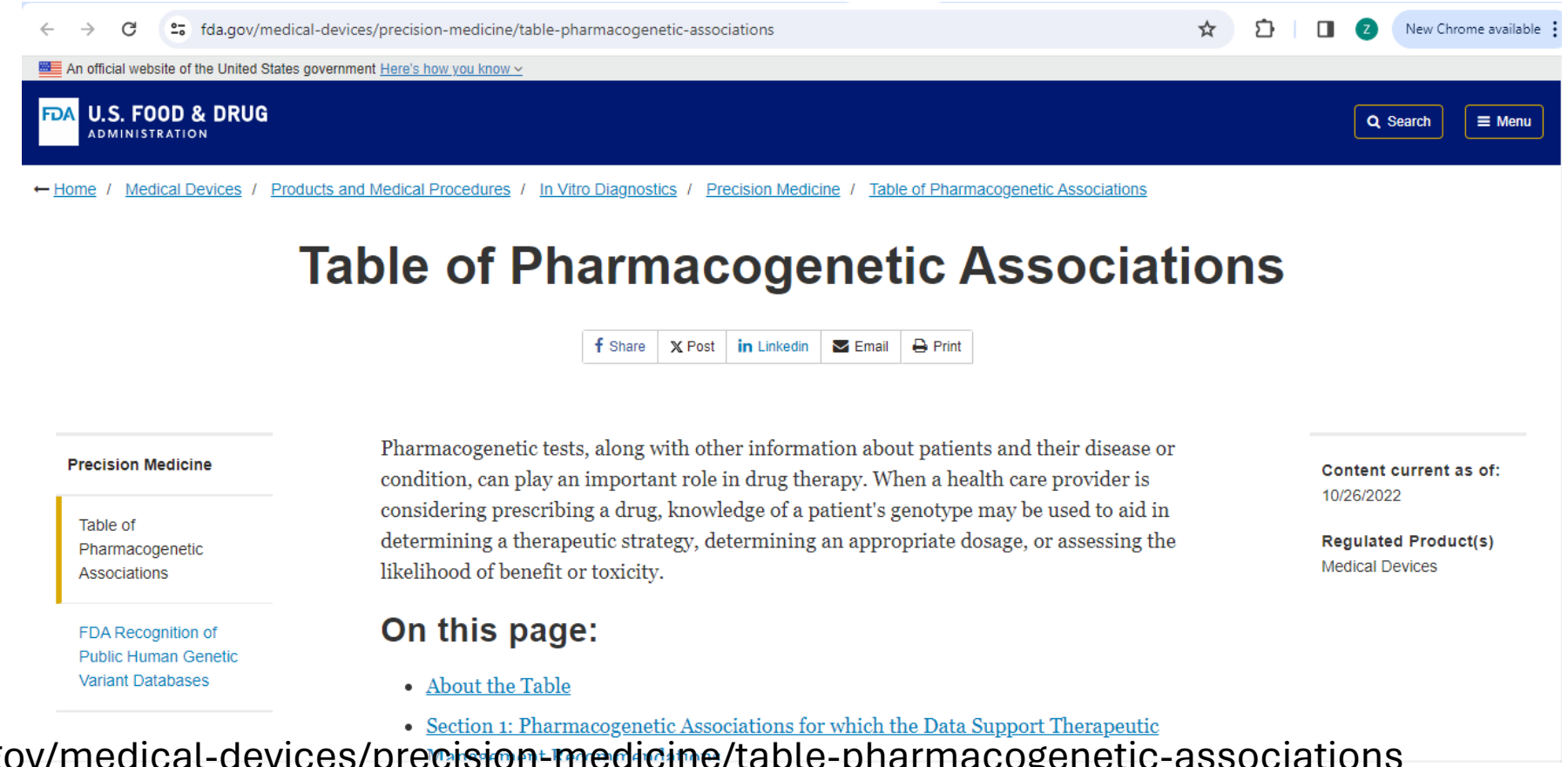


https://www.pharmgkb.org/

- ✓ **PharmVar, a centralized data repository, provides high-quality data on pharmacogene variation.**

https://www.pharmvar.org/

- ✓ **The FDA has a list of 517 gene–drug associations that have been included in drug labels, and its table of pharmacogenomic associations lists 121 drug–gene interactions.**
- ✓ **PGx markers listed by FDA come from two major platforms developed by the PGx Research Network which has under its wing, the PGKB, and the Clinical Pharmacogenetics Implementation Consortium (CPIC), the latter formed in 2009.**



https://www.fda.gov/medical-devices/precision-medicine/table-pharmacogenetic-associations

FDA Recognition of Public Human Genetic Variant Databases

On this page:

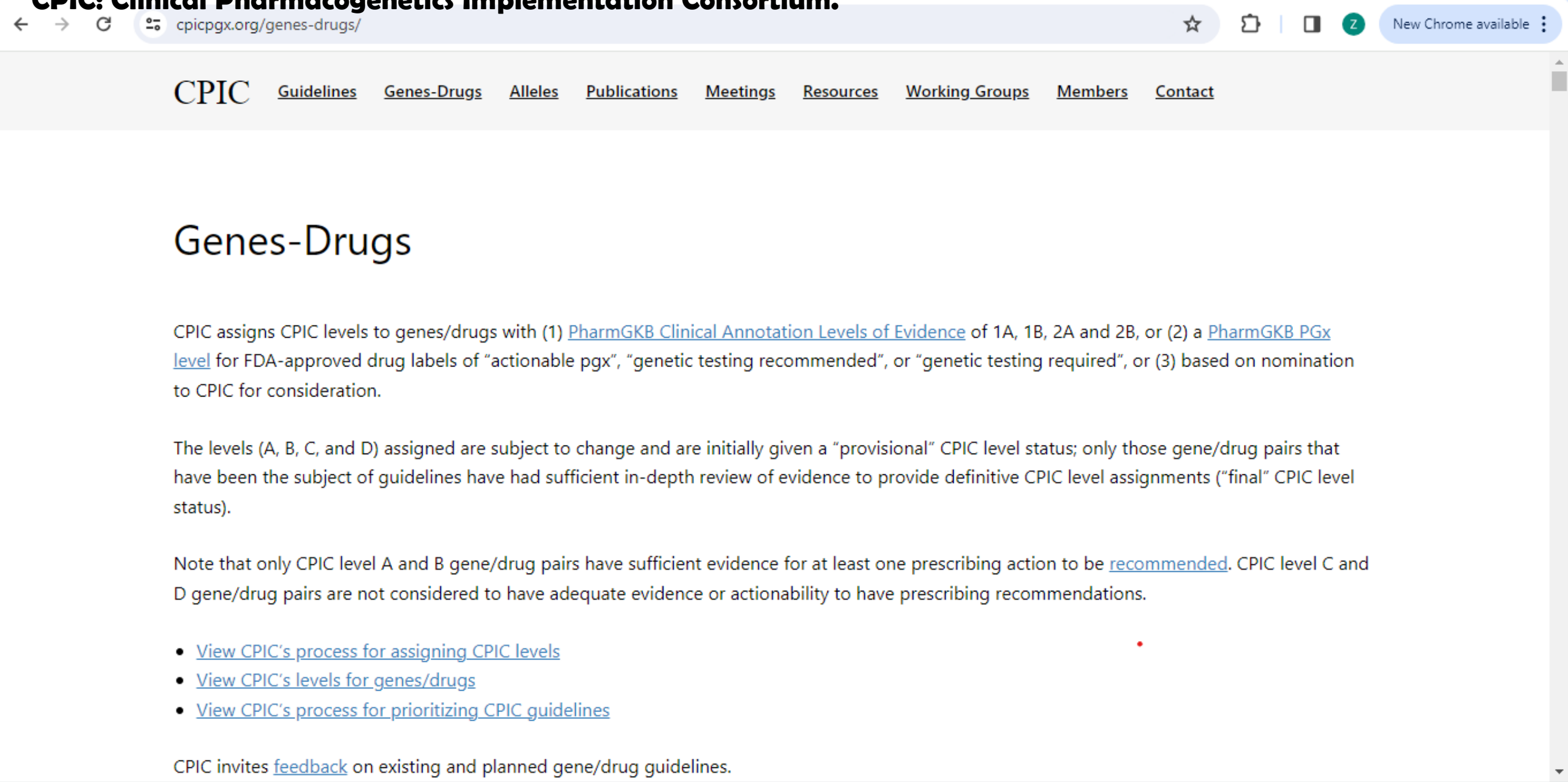
About the Table

For the pharmacogenetic associations listed in this table, the 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 (such as 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.

Top

✓ **Much of the content in drug labels is for information only, rather than to provide guidance on drug dosage or choice; thus, this information is probably largely ignored by prescribers.**

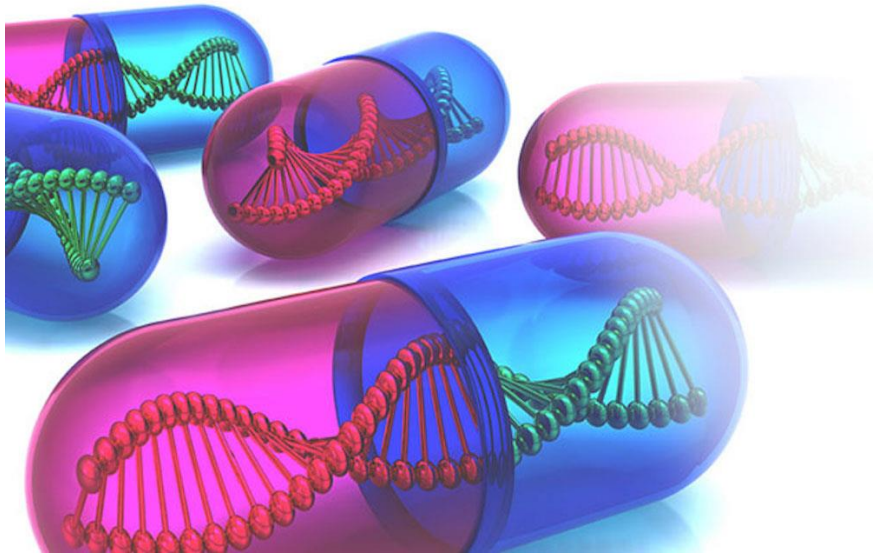
CPIC: Clinical Pharmacogenetics Implementation Consortium.



https://cpicpgx.org/genes-drugs/

- ✓ **The role of the PGKB is to collect, mine, annotate, curate, and assign a validation level to genetic markers of drug response according to the amount of clinical evidence associated to a drug trait. Their website currently lists genetic information for 309 drugs.**

- ✓ **One step further to the advancement of PGx implementation is approached by the CPIC from which the FDA takes genetic information for drug labeling, warnings, and testing recommendations.**



Since CPICs goal is to develop and implement dosing guidelines, they also classify drug pairs according to their potential inclusion in a clinical setting as:

***A variants*, with high evidence that should be used to change drug prescription.**
***B variants*, that could be used to improve prescription due to the availability of therapeutic alternatives.**
***C variants*, with some evidence but not yet convincing, impractical without clear drug alternatives.**
***D variants,* with few studies reported thus, unclear clinical actions.**

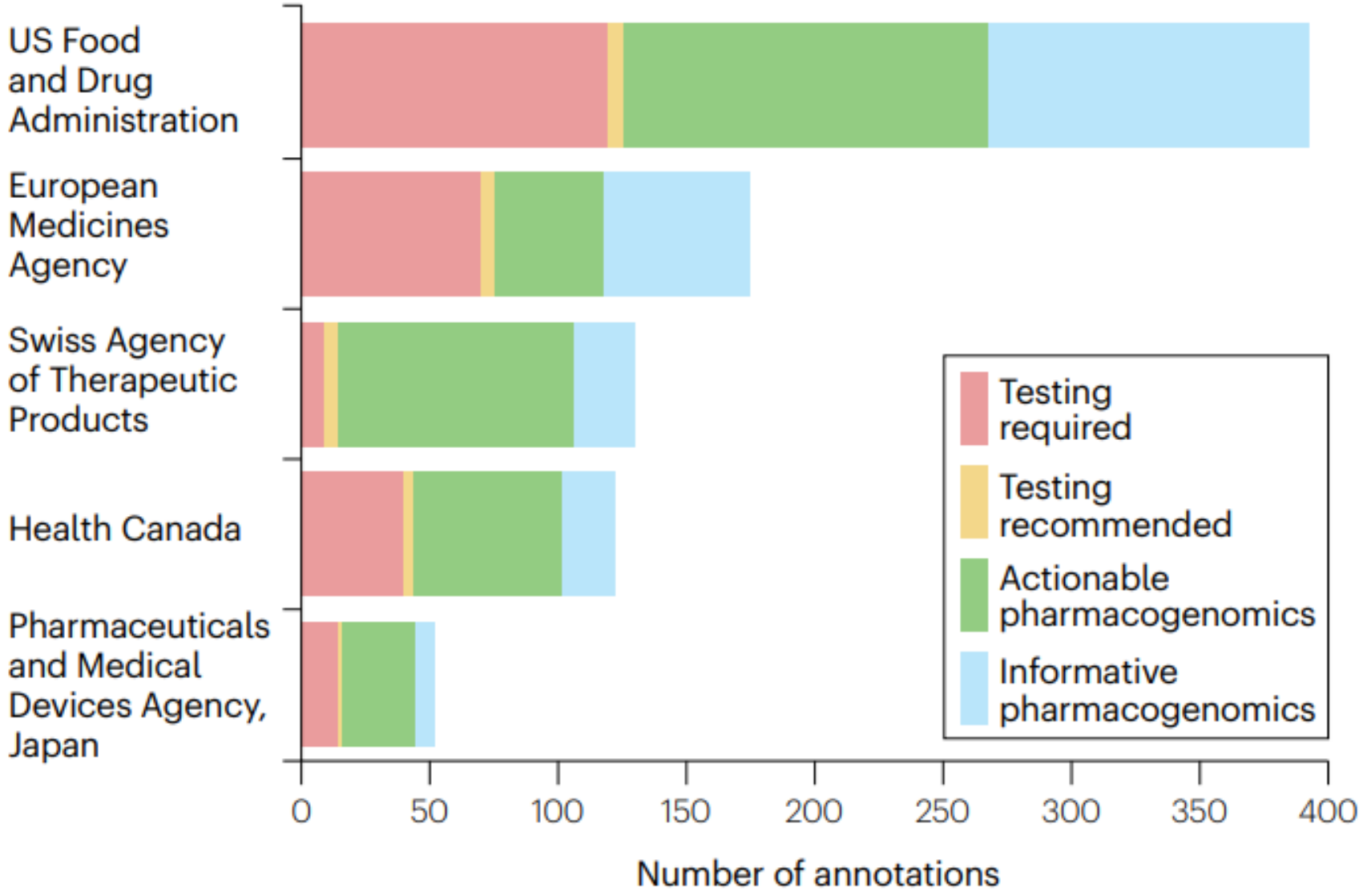
In brief, CPIC guidelines aim to guide patient care decisions for specific drugs utilizing genetic information.

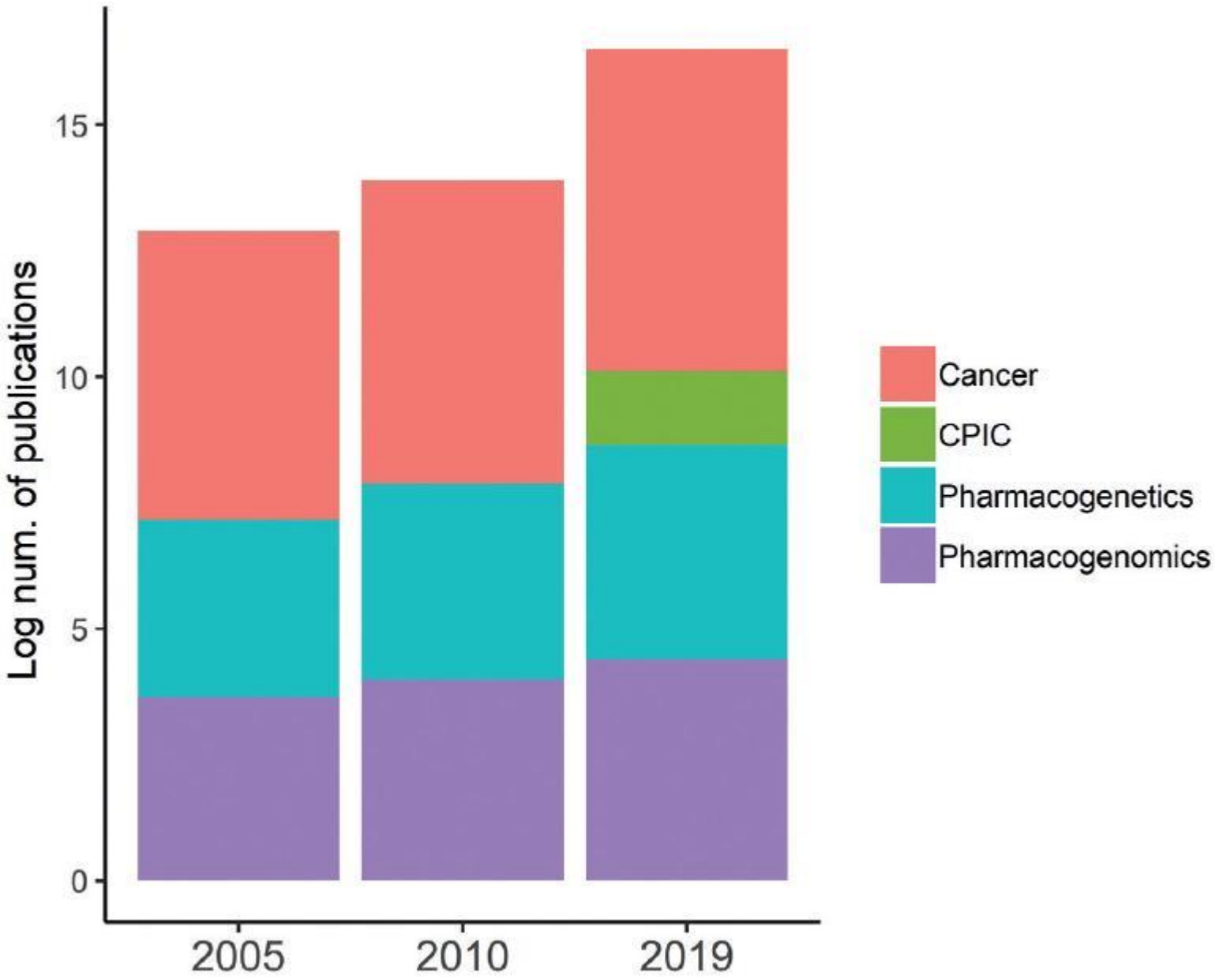
Drug-gene pairs with CPIC guidelines for clinical implementation:

Year/Update	Gene	Drug(s)
2011/2017	*CYP2C9, VKORC1, CYP4F2*	Warfarin
2013/2017	*DPYD*	Fluoropyrimidines
2013/2017	*HLA-A, HLA-B*	Carbamazepine, oxo-carbamazepine
2013	*CYP2C19*	Clopidogrel
2013	*IFNL3*	Peginterferon-alpha-based regimens
2013/2018	*TPMT, NUDT15*	Thiopurines
2014	*CYP2C9, HLA-B*	Phenytoin
2014	*CYP2D6*	Codeine
2014	*G6PD*	Rasburicase
2014/2017	*CFTR*	Ivacaftor
2014	*HLA-B*	Abacavir
2014	*SLCO1B1*	Simvastatin
2015	*CYP2D6, CYP2C19*	Selective serotonin reuptake inhibitors
2015	*CYP3A5*	Tacrolimus
2015	*HLA-B*	Allopurinol
2015	*UGT1A1*	Atazanavir
2016	*CYP2C19*	Voriconazole
2016	*CYP2D6*	Ondansetron
2016	*CYP2D6, CYP2C19*	Tricyclic antidepressants
2018	*CYP2D6*	Tamoxifen
2018	*RYR1, CACNAC1S*	Volatile anesthetic agents
2019	*CYP2B6*	Efavirenz
2019	*CYP2D6*	Atomoxetine

Adapted from https://cpicpgx.org/guidelines/.

Pharmacogenomic information contained in drug labels from different regulatory agencies:



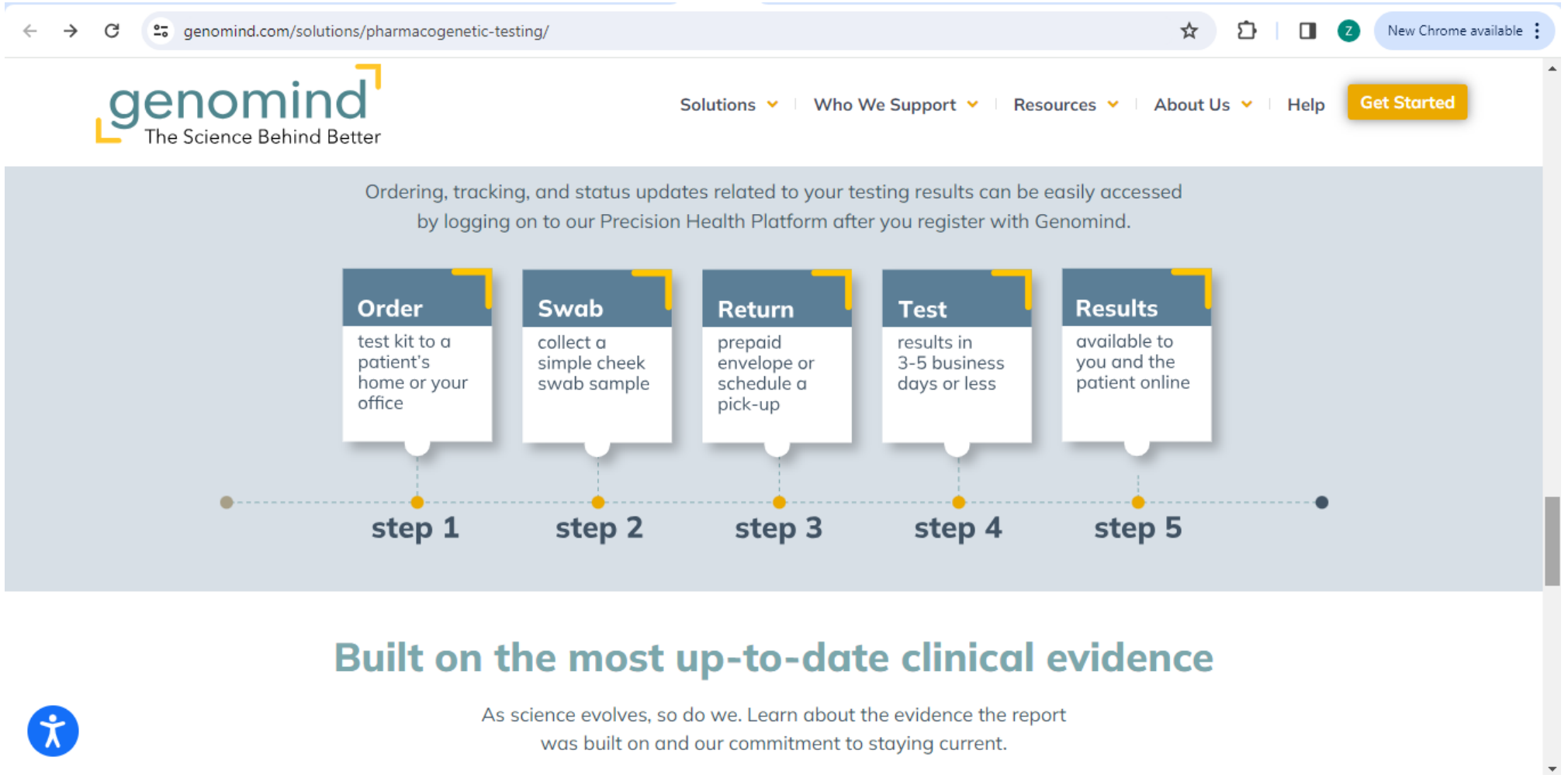


Growth of pharmacogenomics (PGx) published research overtime. Proportion of publications in PGx compared to those in cancer. The rate of growth of PGx reports overtime parallels that of cancer research. CPIC

- ✓ **Moreover, although many drug labels advise prescribers to avoid drug–drug interactions, drug–gene interactions that can lead to the same effect as drug interactions are often not considered.**

For example, **the drug label or summary of product characteristics for tamoxifen, an oestrogen receptor modulator used for breast cancer, asks prescribers to avoid drugs that might interact with tamoxifen and reduce its effect, but a genetic polymorphism in CYP2D6 that has the same effect as the drug interaction is given for information only, without any instruction to genotype the patient before drug use. As a result, approximately 1 in 10 women who are homozygous for non-functional CYP2D6 alleles, and are thus poor metabolizers, might potentially receive reduced benefit from tamoxifen.**

genomind **is an organization that goals to optimize the treatment experience for individuals and healthcare providers by unlocking the value of precision medicine through actionable genetic insights and innovative health technology.**



https://genomind.com/solutions/pharmacogenetic-testing/

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"...less trial and error, faster delivery of positive results"

"We aim to address the need for a better understanding of the role that genetics play in mental health and what this knowledge could do to improve outcomes from those dealing with mental health issues. This has led to tremendous advancements in streamlining treatment, resulting in less trial and error, faster delivery of positive results and in fact, saving numerous lives.

So many things in life feel like they are left to chance or beyond our control. The impact of your genetic makeup might seem to be one of these things, but that is not so! You now have the ability to learn what genetic hand you have been dealt so you and your healthcare provider can make more informed decisions about your mental health and wellness plan."

Dr. Ronald Dozoretz (Dr

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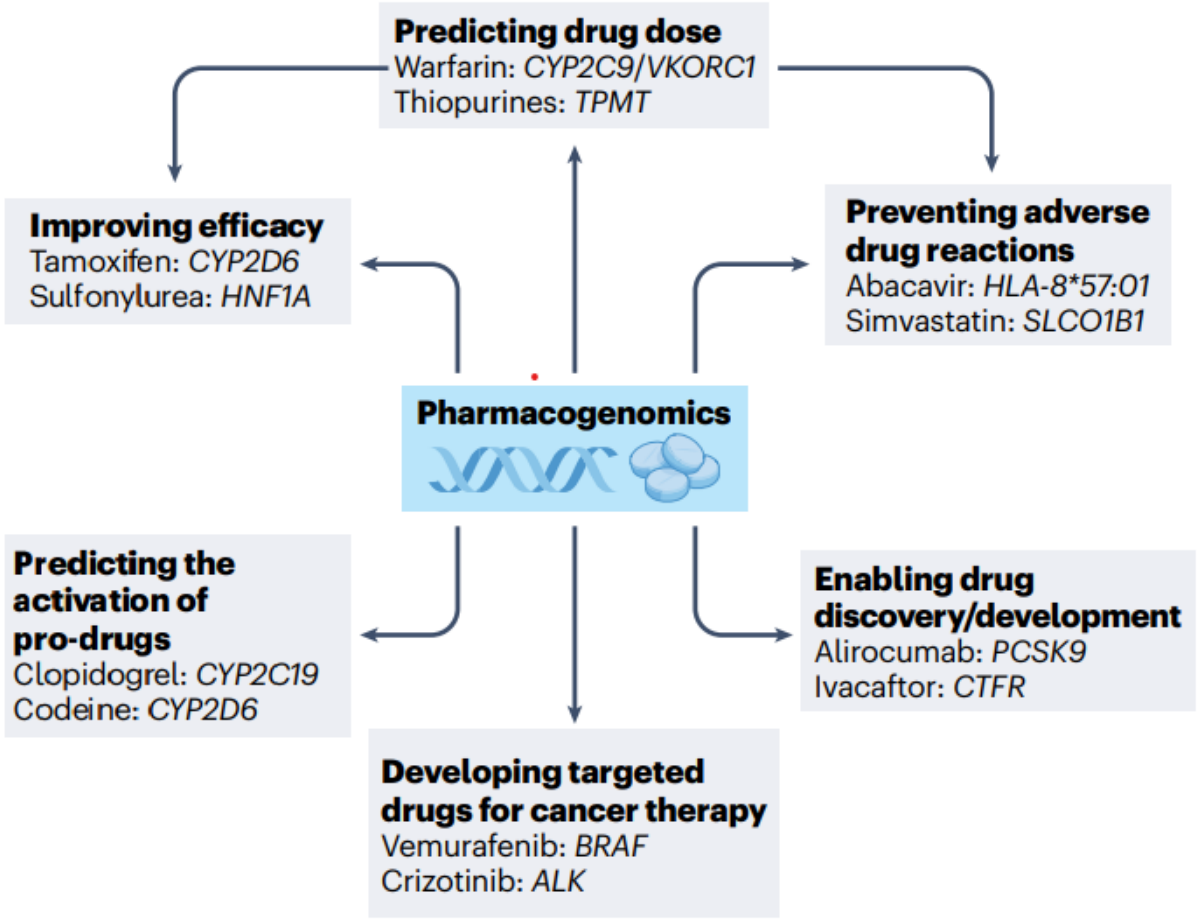
The Importance of Pharmacogenomics

- **Predicting drug dose**
- **Improving drug efficacy**
- **Predicting the activation of pro-drugs**
- **Preventing adverse drug reactions by prospectively genotyping individuals for at-risk alleles**
- **For drug discovery and development through evaluation of both germline and somatic genomes**
- **Developing targeted drugs for cancer therapy**



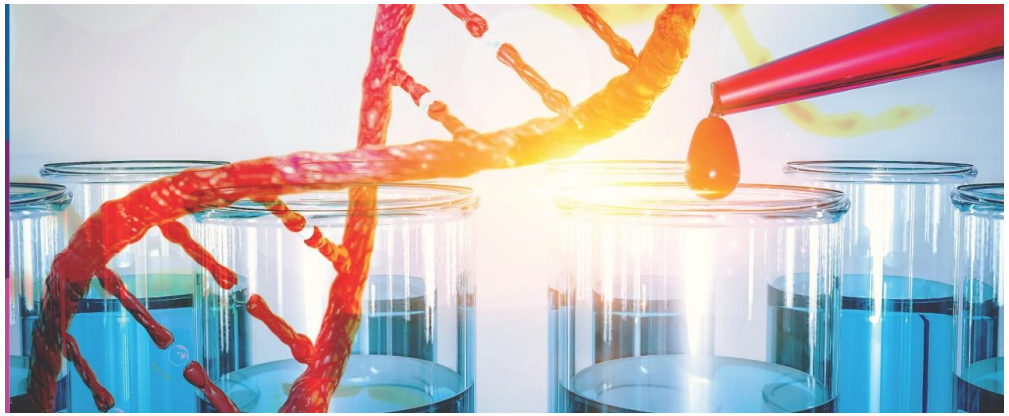
PHARMACOGENETIC TESTING

- **Pharmacogenetic testing** provides information about the genes to help health care provider to choose the medicine and dosage that are the "**best fit**" for the individual.

- The tests use a sample of saliva (spit), blood, or cells swabbed from cheek.

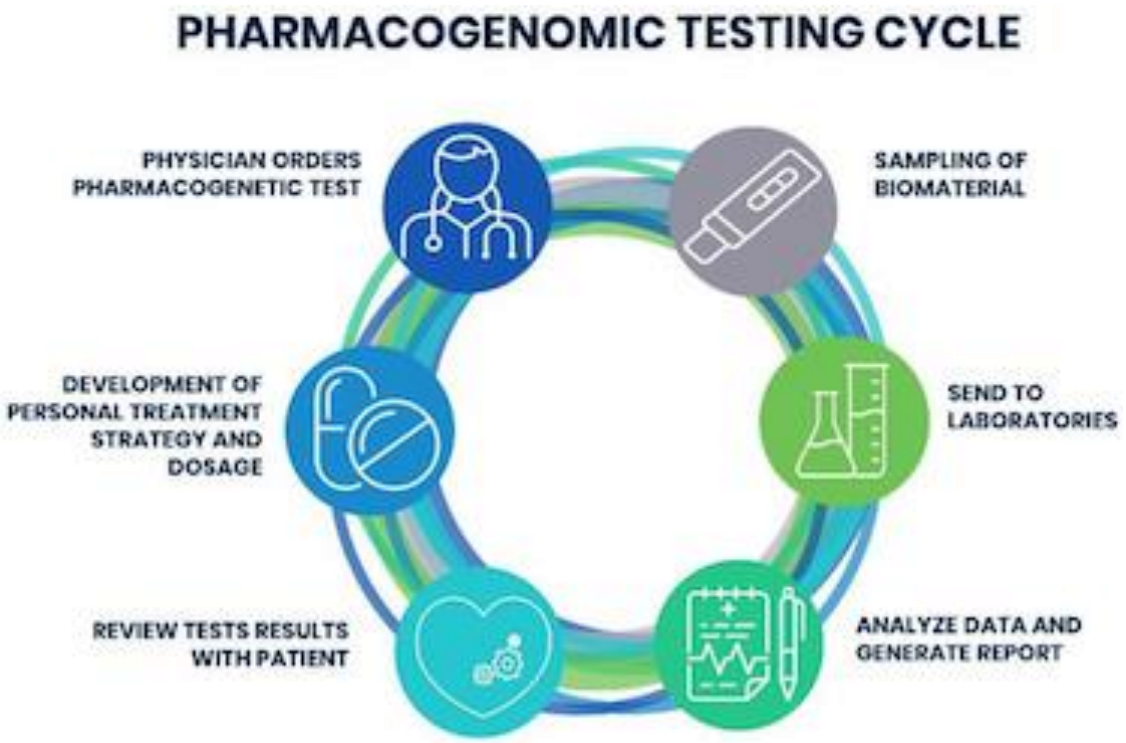
- Pharmacogenetic testing may be used to:

•Find out whether a certain medicine could be effective for individuals.
•Find out how much of the medicine is needed.
•Predict whether their will be a serious side effect from a medicine.



- **Clinical Labs offers a comprehensive range of pharmacogenetic testing in order to provide Clinicians and healthcare providers with important information to help decide on the most appropriate treatment for each individual, particularly in areas such as mental health, pain management, cardiology and oncology.**

- **Pharmacogenetic tests can detect polymorphisms in genes coding for drug metabolizing enzymes that predispose individuals to metabolizing drugs inadequately.**



Gene panels offered:

- *Cytochrome P450 Comprehensive Gene Panel including*:*
  CYP2D6, CYP2C9, CYP2C19, CYP3A4, CYP3A5, CYP1A2, SLCO1B1 and *VKORC1*

Single gene tests:

- *TPMT* (Medicare rebate)
- *DPYD*
- *UGT1A1*
- *CYP2D6*
- *CYPC9*
- *CY2C19*

*Please note that the panel *Cytochrome P450* Genes can be ordered separately or together (*CYP2D6, CYP2C9, CYP2C19, CYP3A4, CYP3A5, CYP1A2, SLCO1B1* and *VKORC1*).

To access Assoc. Prof. Mirette Saad's clinical article on Pharmacogenetic Testing from the December 2021 edition of Pathology Focus, CLICK HERE.

For a full listing of genes tested and drugs metabolised, see the table below.
Please note that this is a guide for gene selection. Some specific medications may not be reported if they are listed under a drug class that is metabolised by the relevant gene.

Selection guide of genes tested based on medications of use

GENE	Type of Metabolised Medication	Metabolised DRUG	Medicare Rebate

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Diagnostic Services

View our full selection of biochemical, biomarker, and genetic tests in our exclusive WEBPORTAL.

Sign up Now
For new users

View All Services
For registered users

Five Simple Steps

1 Order Sampling Kit

2 Collect the Sample

3 Register the DBS Card

4 Return the Sample

5 Receive your Report

- **Pharmacogenetic testing is not the same thing as genetic testing. Genetic tests that are done for health reasons can help diagnose diseases. They may also provide information about the risk for certain diseases. Pharmacogenetic testing cannot diagnose any conditions or tell about your risk for developing them.**

- **Pharmacogenetic tests are not available for all medicines. Examples of common medicines that have pharmacogenetic tests include:**

•Abacavir (an HIV treatment)
•Carbamazepine (an epilepsy treatment)
•Tamoxifen (a breast cancer treatment)
•Warfarin and clopidogrel (blood thinners)

Examples of drugs with alterations in efficacy due to variation in specific genes[a]

Drug	Indication	Gene	Efficacy trait	Clinical action[b]
Clopidogrel	Primary percutaneous coronary intervention, transient ischaemic attacks and strokes	*CYP2C19*	Risk of major adverse cardiovascular events[149] and cerebral ischaemic events[61]	Use other anti-platelet agents in *CYP2C19* PMs
Codeine	Pain relief	*CYP2D6*	Analgesic effect[64]	Use other analgesic agents in *CYP2D6* PMs
Eculizumab	Paroxysmal nocturnal haemoglobinuria	*C5*	Red blood cell haemolysis[150]	Consider alternative therapies in patients with C5 mutations
Lansoprazole, omeprazole, pantoprazole	Gastric acid suppression	*CYP2C19*	Ulcer healing, eradication of *Helicobacter pylori*[151]	Change dose according to metabolizer status
Metformin	Type 2 diabetes mellitus	*ATM, SLC2A2*	Improvement in HbA1c[152,153]	Potential to change dose but unclear
Olaparib, niraparib, rucaparib	Cancers of the ovary, breast, pancreas or prostate	*BRCA1, BRCA2*	Progression-free survival of the different cancers[154]	Use in patients with *BRCA1/2*- mutated cancers
Sulfonylurea	Maturity-onset diabetes of the young	*HNF1A*	Fasting plasma glucose reduction[155]	Change treatment from insulin to low-dose sulfonylurea
Sulfonylurea	Neonatal diabetes	*KCNJ11, ABCC8*	Diabetes control[156]	Change from insulin to high-dose sulfonylurea
Tamoxifen	Breast cancer	*CYP2D6*	Breast cancer recurrence and survival[157,158]	Use alternative therapeutic approaches in *CYP2D6* PMs
Voriconazole	Fungal infections	*CYP2C19*	Resolution of fungal infection[159]	Use alternative agent in URMs (lack of efficacy) and in PM (because of increased risk of toxicity)
Warfarin	Anticoagulation	*CYP2C9, VKORC1, CYP4F2*	Maintenance dose and time in therapeutic range for INR[114]	Alter dose based on genotype and clinical factors

Examples of immune-mediated adverse reactions associated with HLA alleles

Reactions	Drug	HLA class I	HLA class II
SCAR	Allopurinol	*HLA-B*58:01*[129]	NA
	Carbamazepine	*HLA-A*31:01*[47,130] *HLA-B*15:02*[131] *B*15:21*[132] *B*57:01*[133]	NA
	Dapsone	*HLA-B*13:01*[134,135]	NA
	Nevirapine	*HLA-C*04:01*[136,137]	NA
	Phenytoin	*HLA-B*15:02*[138]	NA
DRESS	Abacavir	*HLA-B*57:01*[139]	NA
	Vancomycin	*HLA-A*32:01*[140]	NA
DILI	Amoxicillin-Clavulanate	*HLA-A*02:01*[141]	*HLA-DRB1*15:01-DRB5*01:01-DQB1*06:02* haplotype[142,143]
	Flucloxacillin	*HLA-B*57:01*[46]	NA
	Ticlopidine	*HLA-A*33:03*[144,145]	NA
Agranulocytosis	Clozapine	*HLA-B*38* *HLA-B*39* *HLA-B*67*[146] *HLA-Cw7-B18* *HLA-Cw7-B39* haplotype[147]	*HLA-DRB5*02:01*[147]
Type I hypersensitivity reaction	β-Lactam antibiotics	NA	*HLA-DRB1*10:01*[102] *HLA-DRA* rs7192[148]

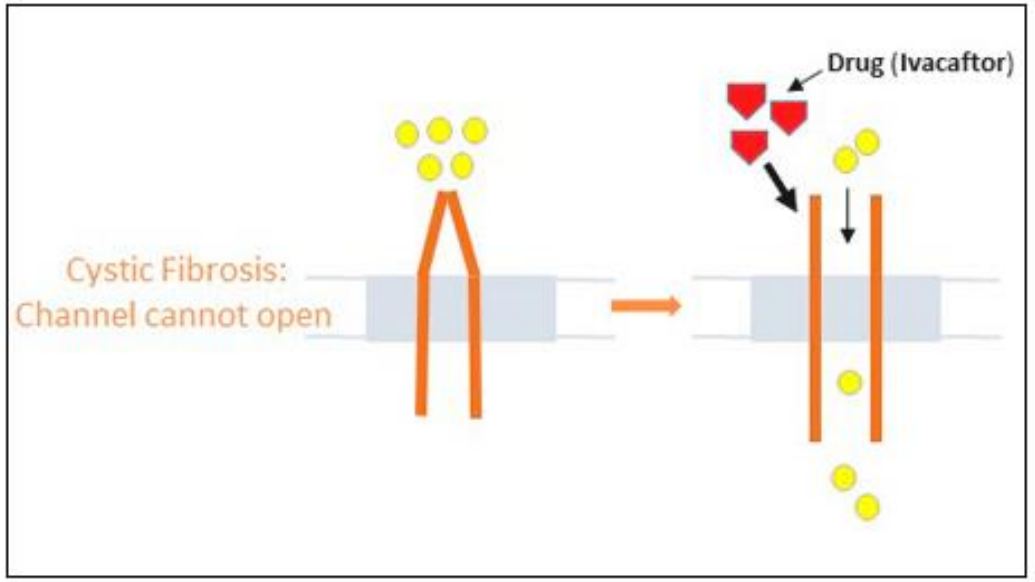
DILI, drug-induced liver injury; DRESS, drug reactions with eosinophilia and systemic symptoms; NA, not applicable; SCAR, serious cutaneous adverse reactions (includes Stevens–Johnson syndrome, toxic epidermal necrolysis and DRESS)

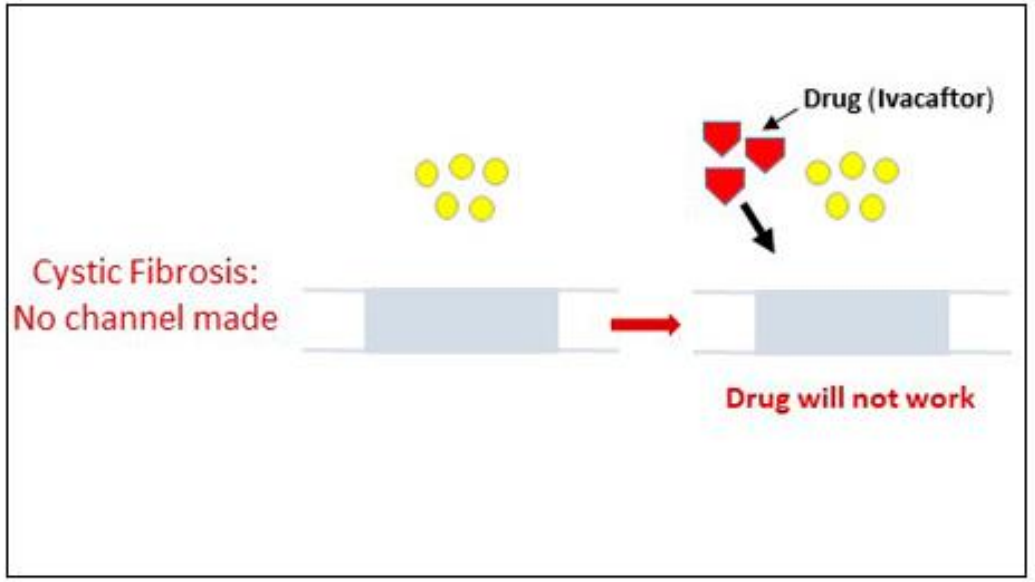
The Advances and Use of Pharmacogenetics in Some Clinical Situations

Cystic Fibrosis and Ivacaftor

Cystic fibrosis is caused by mutations in the *CFTR* gene which affect the CFTR protein.

The CFTR protein forms a channel, which acts as a passageway to move particles across the cells in the body.
For most people the protein is made correctly, and the channel can open and close.
Some mutations that cause cystic fibrosis result in a channel that is closed. The drug ivacaftor acts on this type of mutation by forcing the channel open. Ivacaftor would not be expected to work for people with cystic fibrosis whose mutations cause the channel not to be made at all.





CANCER PHARMACOGENOMICS

- **Cancer Pharmacogenomic is broader field of pharmacogenomics to study how variances in the genome influences an individual's response to different cancer drug treatments.**

- **In clinical settings, it has commonly been observed that the same types and doses of treatment can result in substantial differences in efficacy and toxicity across patients. Thus, the application of pharmacogenomics within the field of cancer can offer key advantages for *personalizing cancer therapy, **minimizing treatment toxicity, and ***maximizing treatment efficacy.**

There are certain examples related to pharmacogenomic testing in the cancer care:

Colorectal Cancer

Irinotecan is a kind of chemotherapy. Where the doctors normally use it to treat the colon cancer. In certain people, genetic variations cause a deficiency of the enzyme name UGT1A1. This enzyme is mainly responsible for metabolizing the Irinotecan.

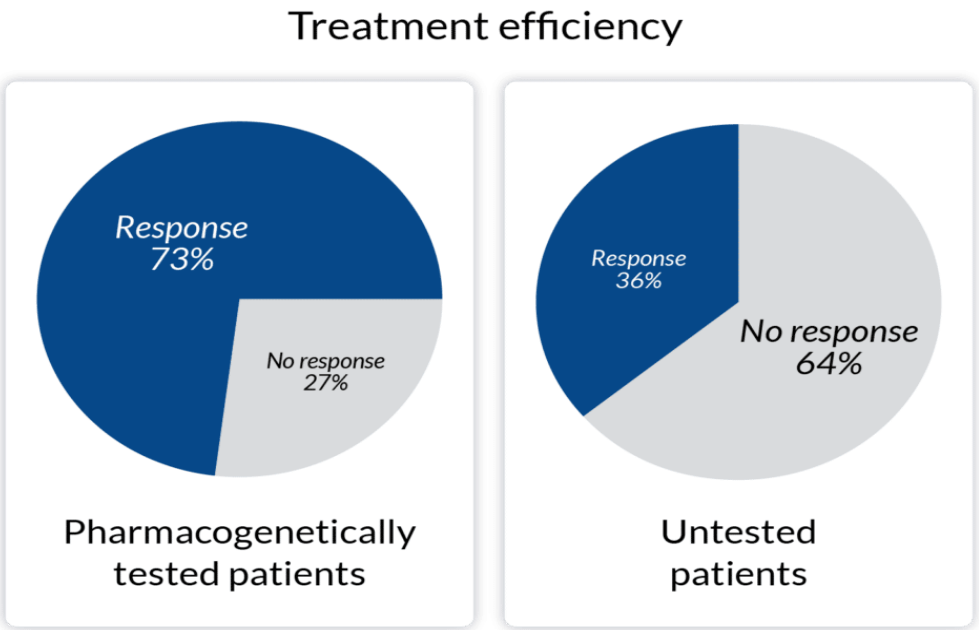
Acute lymphoblastic leukemia (ALL)

About 10% of the people have genetic variations in the enzyme thiopurine methyltransferase (TPMT). TPMT is mainly responsible for metabolizing the chemotherapy for ALL.

IN MENTAL HEALTH..

Pharmacogenetic testing makes determining a suitable antidepressant medication faster.

In a recent study of patients with severe depression, 73 % achieved a drug response in 12 weeks when pharmacogenetic test results were available for the doctor. Without the test results only 36 % of the patients achieved drug response in the same time period. Pharmacogenetic testing has also been shown to decrease the number of emergency room visits and hospitalizations of mental health patients.



In HYPERTENSION..

- Apart from poor medication adherence, the low efficacy of some therapies could also be related to inter-individual genetic variability.

- Genetic factors not only affect blood pressure (BP) elevation but also contribute to inter-individual variability in response to antihypertensive treatment.

- Genetic polymorphisms can influence drug responses through genes engaged in the pathogenesis of hypertension that are able to modify the effects of drugs.

- The results of numerous studies confirm that genotype-based antihypertension therapies are the most effective and may help to avoid the occurrence of major adverse events, as well as decrease the costs of treatment.

Pharmacogenetics Approach For The Improvement Of Covid-19 Treatment

- **The treatment of coronavirus unwellness 2019 has been a challenge. The effectivness of many medication has been evaluated and variability in drug response has been ascertained.**

- **Genetics may justify this variation and improve patients' outcomes with this advanced disease.**

- **A number of studies elucidate the pharmacogenetic variants rumored for medication used for COVID-19 treatment (remdesivir, oseltamivir, lopinavir, ritonavir, azithromycin, antimalarial drug, anti-inflammatory, ivermectin, and dexamethasone).**

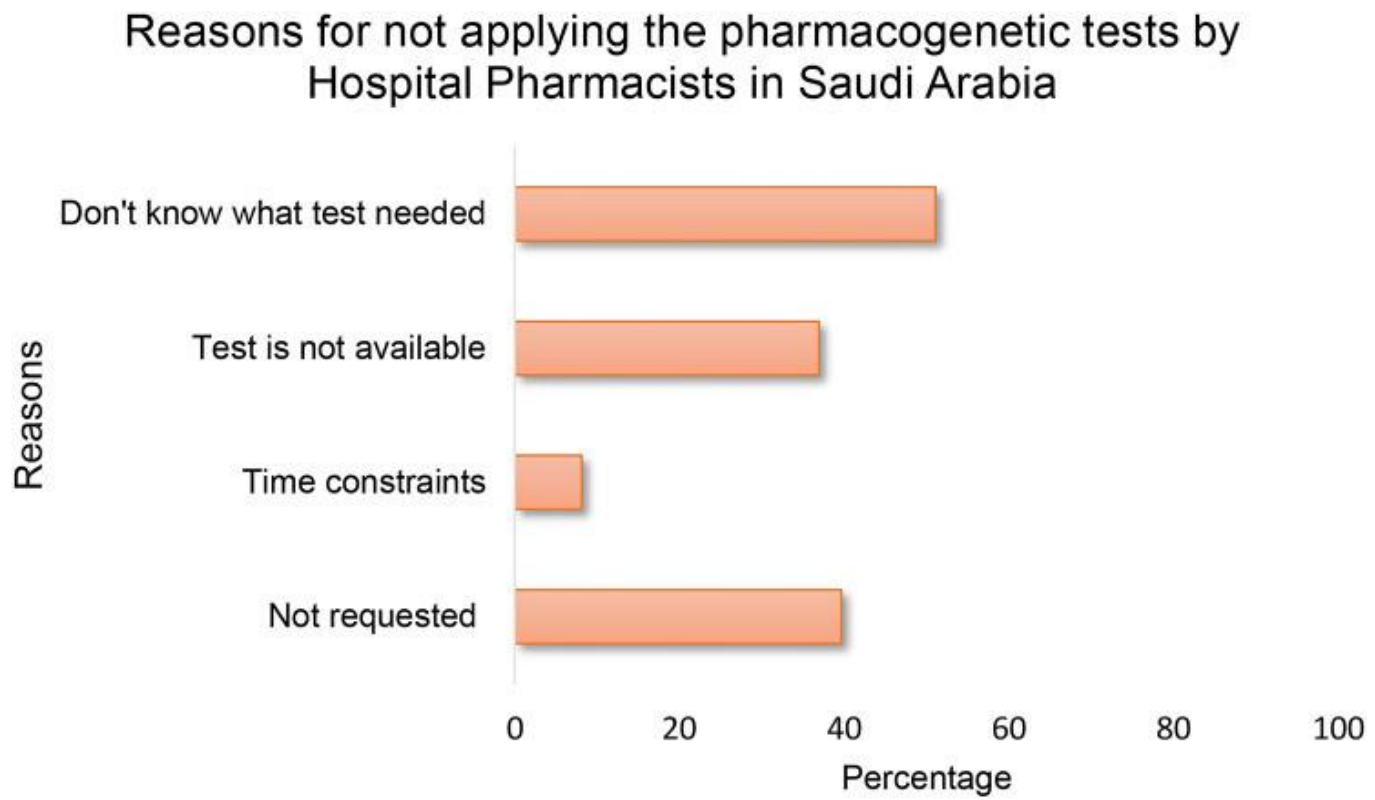
In a Study Titled:

Knowledge, Perception, and Application of Pharmacogenomics Among Hospital Pharmacists in Saudi Arabia

- **Only 30% of the pharmacists had received any type of formal training on PGx. Of these, only 9 participants had actually put the knowledge into practice.**
- **Participants showed a moderate to low level of pharmacogenomic knowledge. The low knowledge and the availability of the pharmacogenetic test are the main barriers for the low adoption of the pharmacogenomics in the clinical practice.**
- **Approximately 83% felt the need to know more about pharmacogenomics.**
- **However, 50% of the total participants reported that their hospital management is unaware of the pharmacogenomics importance in clinical practice.**

This study emphasizes on two needs which can help promote the use and implementation of pharmacogenomics.

- ✓ **One is the need to update the pharmacy education and training programs with pharmacogenomic-related areas to raise the pharmacist's knowledge and practical skill to apply pharmacogenomics in the clinical practice effectively.**
- ✓ **Another need is to increase the awareness of the decision and policy-makers with the importance of pharmacogenomics for the patient benefit and safety.**



Ethics

In the area of bioethics Pharmacogenetics has become a controversial issue. Privacy is the major concerns. The ethical issues raised by clinical pharmacogenomics fall broadly into three interrelated categories: ownership, access, and use.

Conclusion & Future Perspectives

- Research in pharmacogenomics has increased since the completion of the Human Genome Project.
- Computational advances have facilitated cheaper and faster sequencing.
- Data on the utility of some pharmacogenomic associations are increasing, but implementing these into clinical practice has been very slow.
- It is a large step to carry pharmacogenetic technology into everyday medical decisions.
- Pharmacogenomics is just one component of the drive towards personalized or precision medicine.
- Multimodal algorithms that incorporate both clinical (for example, age, sex and body weight) and genetic factors are needed. The development of such multimodal algorithms will undoubtedly be enhanced by the use of advanced digital tools.

Implications & Recommendations:

- ✓ More research to identify new drug–gene associations is still needed.
- ✓ A pharmacogenomic service should be accompanied by funding for research, not only biomedical research, but also research into ethical, legal, and social issues.
- ✓ Development of personalized drug therapies using pharmacogenetics can be increased, as the cost per genetic test decreases.
- ✓ Appropriate funding is needed to the researchers and health care providers to go on with the pharmacogenetic testing.
- ✓ A comprehensive education and training package is needed in Pharmacogenomic-related areas to raise the health care workers' knowledge and practical skill to apply pharmacogenomics in clinical practice effectively.
- ✓ Support is needed for clinicians, including clinical decision support systems, to minimize errors and maximize cost efficiency.

> Pharmacogenomics can play an important role in identifying responders and non-responders to medications, avoiding adverse events, and optimizing drug dose.
>
> - Food and Drug Administration

> PGx is the future of medicine. I use it to bring precision to my prescribing. Now, I can more accurately predict adverse drug events, even outside of my specialty. This process builds better relationships with patients and other providers.

Jennifer Reed, FNP-BC, PMHNP-BC

Private Practice Owner, Board Certified in Family Practice and Psychiatric Mental Health

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Thank you