Adnà

Mike, here is your Pharmagenomics Report


Your DNA
has answers

2. Summary

Pharmacogenomics: Cardiology

- Phenprocoumon
- Pravastatin
- Warfarin
- Hydrochlorothiazide
- Simvastatin

Pharmacogenomics: Neurology

- Amisulpride
- Carbamazepine
- Escitalopram
- Paliperidone
- Risperidone
- Aripiprazole
- Citalopram
- Haloperidol
- Quetiapine
- Ziprasidone

Pharmacogenomics: Pain

- Aspirin
- Buprenorphine
- Meperidine
- Naltrexone
- Tramadol
- Alfentanil
- Fentanyl
- Morphine
- Pentazocine

Pharmacogenomics: Onco

- Cisplatin
- Irinotecan
- Methotrexate
- Fluorouracil, capecitabine, pyrimidine analogues, tegafur and Neoplasms
- Mercaptopurine
- Vincristine

Pharmacogenomics: Other

- Peginterferon Alpha-2b
- Tacrolimus
- Ribavirin
- Viagra (Sildenfail)

Caption:

- We have not found anything in your genetics that indicates a predisposition to an abnormal effect of this drug. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype you have a greater predisposition for the drug to have an abnormal effect on you. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype you have a greater predisposition for the drug to have a harmful effect on you. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype, you have a greater predisposition to respond positively to this drug. Other non-analyzed and non-genetic genetic factors may play a role.

Pharmacogenomics: Cardiology

Simvastatin

Simvastatin is a lipid-lowering agent that is derived synthetically from the fermentation of Aspergillus terreus. It is a potent, competitive inhibitor of 3-hydroxy-3-methylglutaryl coenzyme A reductase (hydroxymethylglutaryl COA reductases), which is the rate-limiting enzyme in cholesterol biosynthesis. It may also interfere with steroid hormone production. Due to the induction of hepatic LDL receptors, it increases the breakdown of LDL cholesterol.

Your genetic map

Gene	SNP	Genotype
SLCO1B1	rs4149056	TT

What do your genetics tell us?

Patients with the TT genotype may be at a lower risk of simvastatin-related myopathy as compared to patients with the CT or CC genotype. Other genetic and clinical factors may also affect a patient's risk for toxicity.

More information:

https://www.ncbi.nlm.nih.gov/pubmed/28482130
https://www.ncbi.nlm.nih.gov/gtr/conditions/C0220991

Pharmacogenomics: Neurology

Escitalopram

Escitalopram, also known by the brand names Lexapro, and Cipralex, among others, is an antidepressant of the Selective Serotonin Reuptake Inhibitor (SSRI) class. It is approved by the U.S. Food and Drug Administration (FDA) for the treatment of adults and children over 12 years of age with Major Depressive Disorder (MDD) or Generalised Anxiety Disorder (GAD).

Your genetic map

Gene	SNP	Genotype
CYP2C19	rs12248560	CC

What do your genetics tell us?

Patients with the CC genotype may exhibit decreased metabolism of citalopram or escitalopram as compared to patients with the TT or TC genotype. Other genetic factors, including the other CYP2C19 alleles *2 rs4244285,*3 rs4986893, and clinical factors, may also affect a patient's citalopram or escitalopram metabolism.

More information:

https://www.ncbi.nlm.nih.gov/pubmed/17625515

Pharmacogenomics: Onco

Fluorouracil, capecitabine, pyrimidine analogues, tegafur and Neoplasms

Fluorouracil (5-FU), sold under the brand name Adrucil, among others, is a medication used to treat cancer. By injection into a vein, it is used for colon cancer, esophageal cancer, stomach cancer, pancreatic cancer, breast cancer, and cervical cancer. As a cream it is used for actinic keratosis and basal cell carcinoma. It is a potent antimetabolite used in the treatment of cancer. It is a drug that blocks the methylation reaction of deoxyuridic acid, converting it into thymidylic acid by inhibiting an enzyme that is important for the synthesis of thymidine, which, being part of the DNA molecule, stops its formation. The drug is specific to the S phase of the cell phase cycle. 5-Fluorouracil is involved in the synthesis of DNA and inhibits, to a small degree, the formation of RNA. The two actions combine to promote a metabolic imbalance that results in cell death. The inhibitory activity of the drug, by its analogy with uracil, has an effect on the rapid growth of the neoplastic cells, which, preferentially, take advantage of the uracil molecule for nucleic acid biosynthesis.

Your genetic map

Gene	SNP	Genotype
DPYD	rs67376798	TT

What do your genetics tell us?

TT-genotype patients treated with fluoropyrimidine-based chemotherapy may exhibit 1) increased clearance of the drug and 2) decreased, but not absent, risk and reduced severity of drug toxicity as compared to patients with the AT genotype. The combination (FOLFOX, FOLFIRI or FEC) and delivery of the drug may influence risk for toxicity. Other genetic and clinical factors may also have an influence.

More information:

https://www.ncbi.nlm.nih.gov/pubmed/17700593
https://www.ncbi.nlm.nih.gov/pubmed/23603345

Pharmacogenomics: Onco

Vincristine

Vincristine is an anti-tumour vinca alkaloid isolated from Vinca Rosea. It is marketed under several brand names, many of which have different formulations, such as Marqibo (liposomal injection) and Vincasar. Vincristine is indicated for the treatment of acute leucemia, malignant lymphoma, Hodgkin's disease, acute erythraemia, and acute panmyelosis. Vincristine sulfate is often chosen as part of polychemotherapy because of its lack of significant bone–marrow suppression (at recommended doses) and unique clinical toxicity (neuropathy).

Your genetic map

Gene	SNP	Genotype
CEP72 DT	rs924607	TT

What do your genetics tell us?

Patients with the TT genotype may be at an increased risk of peripheral nervous system diseases when treated with vincristine as compared to patients with the CC or TC genotype. Other genetic and clinical factors may also influence a patient's response to vincristine.

More information:

https://www.ncbi.nlm.nih.gov/pubmed/25710658

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