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Personalizing drug treatment using pharmacometabonomic approach

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Current treatment approach

• Based on large cohort studies – blockbuster medicine

• One-size-fits-all model - Do not account for individual differences

• Increased cost and safety concern (without proven efficacy)

• The response rates on drugs is still unsatisfactory, varying widely from 20% to 75% depending on the drug and the disease


Drug development failure

• Right medication,
• Right dose,
• **Right patient,**
• Right time and
• Right route
Personalized medicine

What is personalized medicine?

• Use of patient’s characteristics including demographics, histories and molecular information (genetic, protein and metabolic profile) to better define therapies

• Integration of diagnostics and therapeutics – known as theranostics (a diagnostic therapy that identifies patients most likely to be helped by a new medication, and targets drug therapy based on the test results).

• Philosophy - every patient has a unique biology and pathophysiology that should be reflected in the choice of pharmacotherapy, thus resulting in an improved treatment outcome

Why is PM better than the current approach?

• Treat the patient, **NOT** just the disease

• Provide for individual differences – a tailored approach

• Predict susceptibility and risk factors, pre-empt disease progression, target intervention, avoid ineffective treatments and prevent adverse reactions.

• Reduce costs in the long term

• Stratify, genotype and phenotype diseases
Omics genotypes and phenotype

Genomics – study of genes

Transcriptomics – study of mRNA

Proteomics – study of proteins

Metabolomics – study of metabolites
Pharmacogenomics

- Pharmacogenomics - study that examines the impact of genetic variation on the response to medications

- Drug metabolizing enzymes, transporters and receptors are encoded by several hundred genes, playing a pervasive role in ADME and drug targeting

Limitations

• A major factor underlying inter-individual variation in drug effects is variation in metabolic phenotype, which is influenced not only by genotype but also by environmental factors such as nutritional status, the gut microbiota, age, disease and the co- or pre-administration of other drugs which modulate drug pharmacokinetic (ADME), efficacy and toxicity

• Thus, although genetic variation is clearly important, it seems unlikely that personalised drug therapy will be enabled for a wide range of major diseases using genomic knowledge alone
Pharmacoproteomics

- **HercepTest**
  - HER2 protein
  - Trastuzumab

- **EGFR pharmDX Kit**
  - EGFR protein
  - Cetuximab

Trastuzumab

Cetuximab
MY GENOME MADE ME DO IT!
• Pharmacometabonomics - the prediction of the outcome (for example, efficacy or toxicity) of a drug or xenobiotic intervention in an individual based on a mathematical model of pre-intervention metabolite signatures

• One of the major factors influencing a patient’s response to any medication is drug pharmacokinetic (ADME). Differences in the balance of pharmacokinetic leading to detoxification vs. toxicity are the difference between a treatment being safe and effective or causing an adverse drug reaction

References:


• Ian D. Wilson. Drugs, bugs, and personalized medicine: Pharmacometabonomics enters the ring. PNAS August 2009; 106(34):14187–14188
Advantages of metabolomics approach:

- affected by both genes and environment
- closer to phenotype
- simple and non-invasive
- relatively ease of analysis
- potential to identify novel biochemical pathways
Pharmacometabonomics

Urine samples of 10 rats

Inject galactosamine hydrochloride

Liver bioassay

NMR

NMR spectra

PCA

Inhaled steroid use

Scatterplots of the discriminating principal components, blue triangles show subjects on ICS

• M Basanta, B Ibrahim, R Dockry, D Douce, M Morris, D Singh, A Woodcock and SJ Fowler. Exhaled volatile organic compounds for phenotyping chronic obstructive pulmonary disease; a cross-sectional study. Respiratory Research 2012;13(1);72
Eosinophils and neutrophils

Scatterplots of the discriminating PCs derived from the models, blue triangles show:
a) sputum eosinophils ≥ 2%; b) sputum neutrophils ≥ median

M Basanta, B Ibrahim, R Dockry, D Douce, M Morris, D Singh, A Woodcock and SJ Fowler. Exhaled volatile organic compounds for phenotyping chronic obstructive pulmonary disease; a cross-sectional study. Respiratory Research 2012;13(1);72
Warfarin use

Stable

Unstable
Conclusion

• If we can say genetic contribute to a disease, of course it can also contribute to drug response.
• Limitations of pharmacogenomics lead to the new approach known as pharmacometabonomics.
• This method as high potential to personalize drug treatment as it looks at the contribution of both genetics and environmental factors to the drug effects.
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