Personalized Medicine – Quo Vadis?

Conference on Personalized Medicine:
Breaking Down the Barriers and Achieving Results

Harvard Medical School
October 11, 2007

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CDER/FDA

Outline

- Personalized Medicine – what is it?
- Then and now – what we can do today that we couldn't do before
- Biomarkers and (genetic) testing
- Dose and drug selection – some key points to consider
- Drug-test co-development – a paradigm change?
- Other considerations on the quest to get medicine less impersonal

- *Theme:* Evidence and Benefit – Risk considerations

IV. Definition of Personalized Medicine

Personalized medicine means different things to different people. Some have suggested that personalized medicine is the application of genomic data to better target the delivery of medical interventions. Others have suggested that it is a crucial tool in the discovery and clinical testing of new products. And others have suggested that it involves the application of sophisticated, clinically useful diagnostic tools that may help determine a patient's predisposition to a particular disease or condition. In fact, personalized medicine can encompass all of those concepts.

In theory, personalized medicine is the management of a patient's disease or disease predisposition, by using molecular analysis[2] to achieve the optimal medical outcomes for that individual — thereby improving the quality of life and health, and potentially reducing overall healthcare costs.

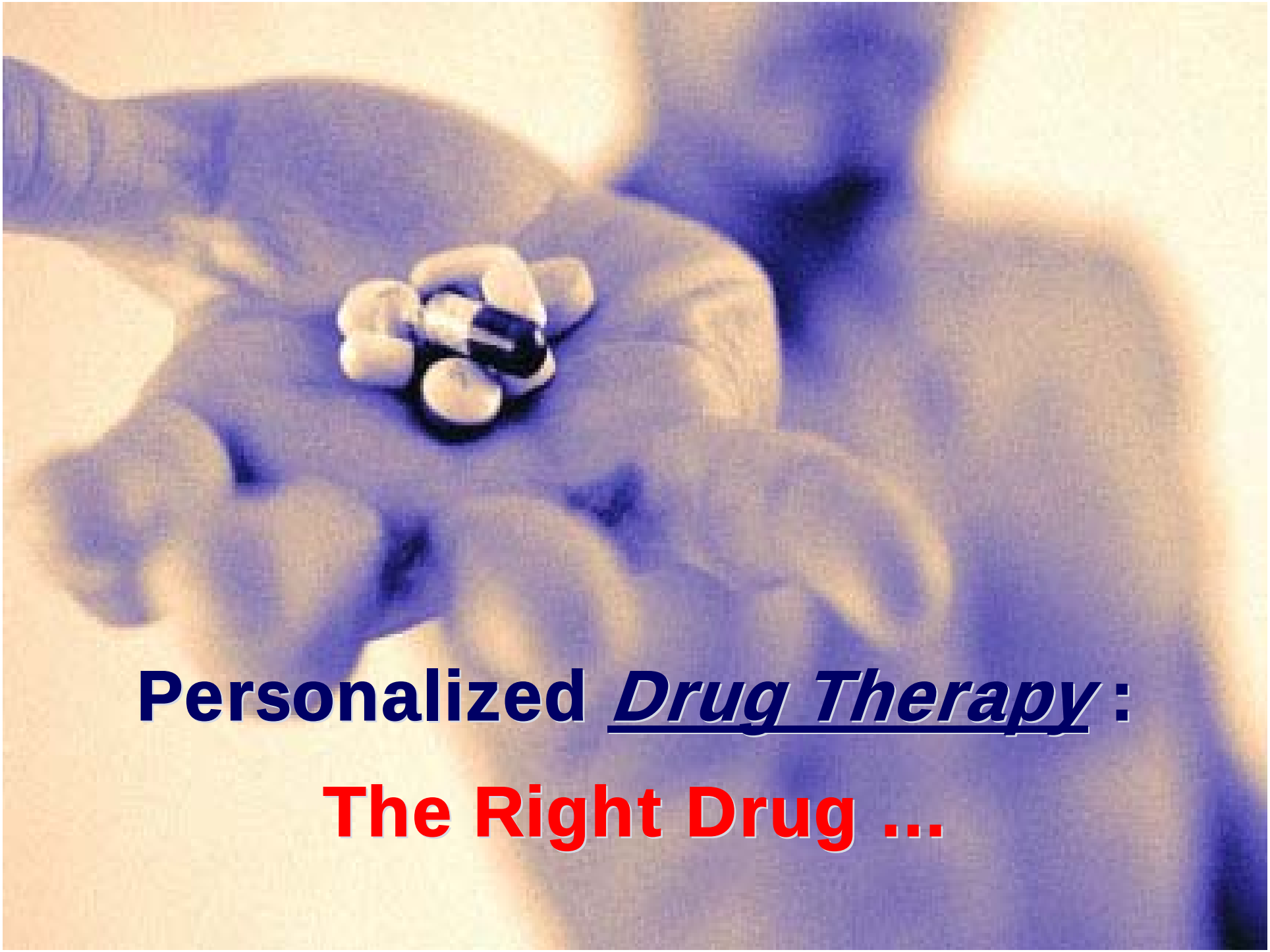
In practice, personalized medicine is a comprehensive approach utilizing:

- Molecular analysis of both patients and healthy individuals to guide decisions throughout all stages of the discovery and development of pharmaceuticals and diagnostics; and
- Applying this knowledge in clinical practice for a more efficient delivery of accurate and quality healthcare through improved prevention, diagnosis, treatment, and monitoring methods.

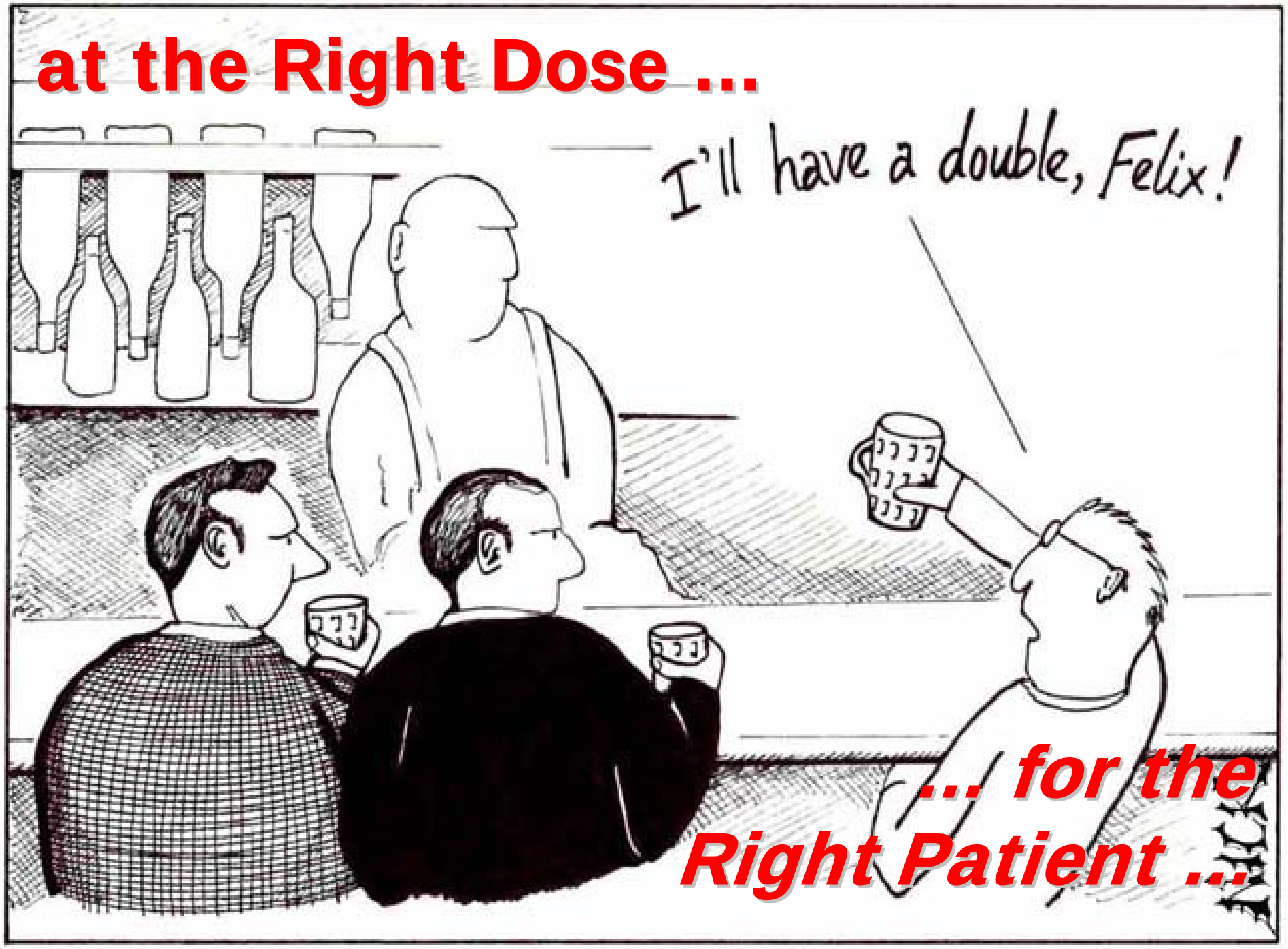
V. Public Policy Issues Impacting Personalized Medicine

Several clusters of significant public policy issues mark the pathway to the growth and acceptance of personalized medicine. While none of these issues is unique to personalized medicine, government regulation of clinical trials, intellectual property rights, licensing practices, healthcare reimbursement, and privacy are among the areas that may need to be re-examined.

Currently, public policy decisions in the healthcare arena are made in a fragmented and uncoordinated way, without considera-

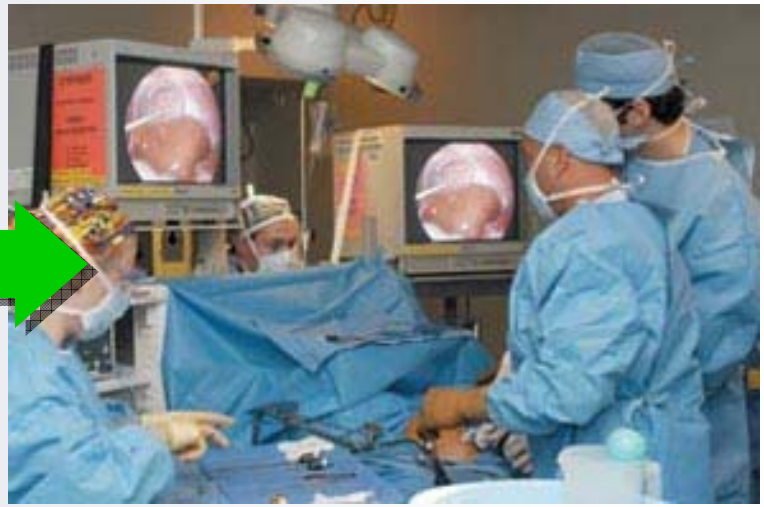

Personalized *Drug Therapy* :
The Right Drug …

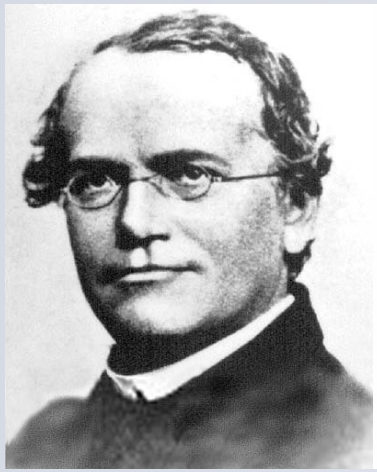
at the Right Dose …

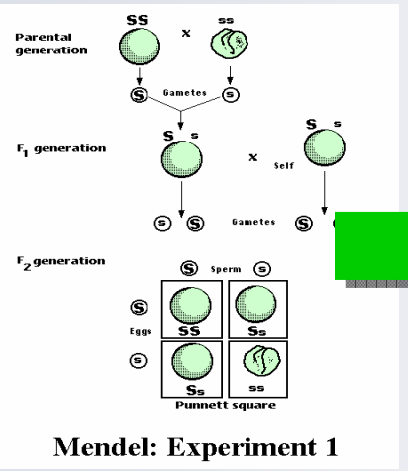


… at the Right Time.

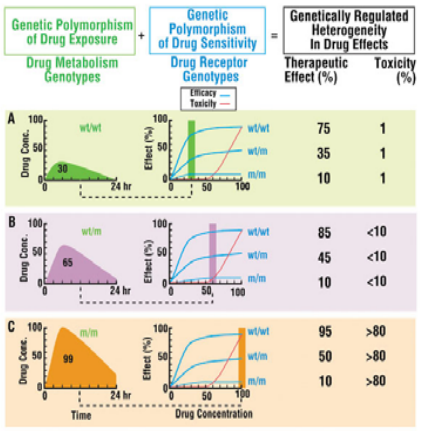
(Personalized) Medicine: Then and Now



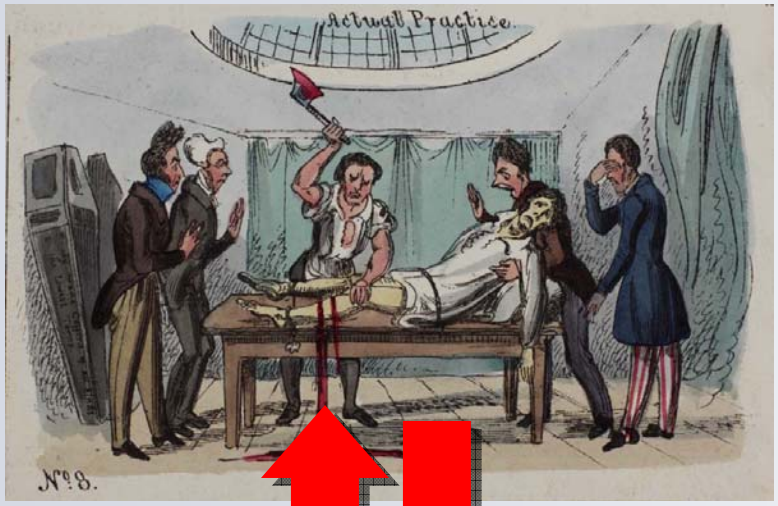


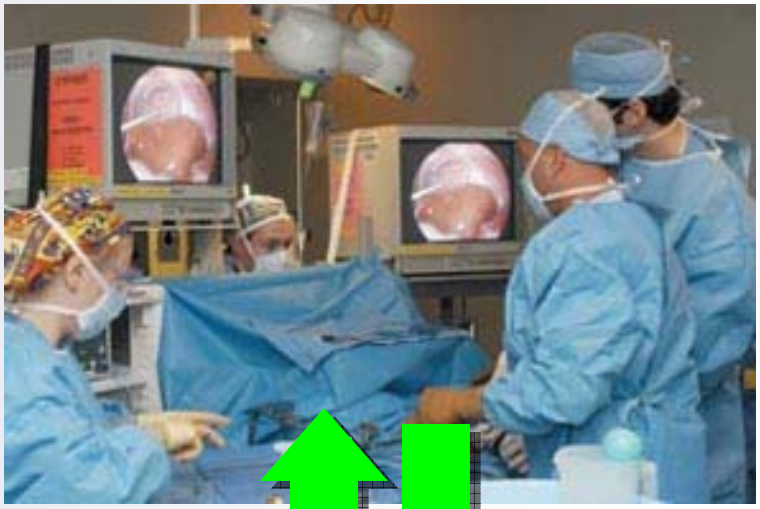


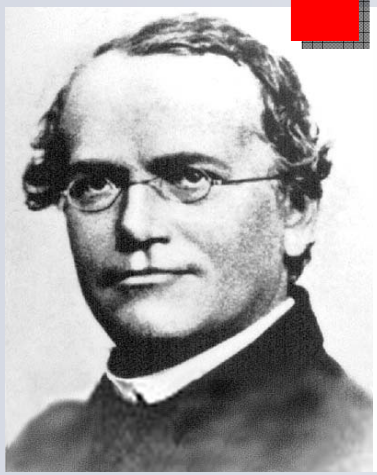
Mendel: Experiment 1

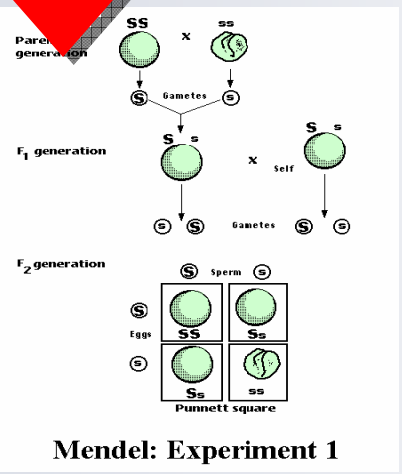


(Personalized) Medicine: Then and Now

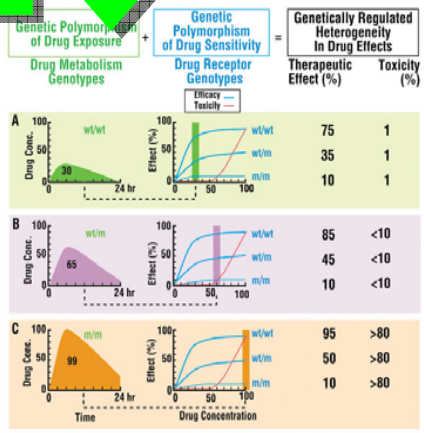








Mendel: Experiment 1



Example: Leukemia and Lymphoma

1950	"Disease of the Blood"	
1960	Leukemia	Lymphoma
1970	Chronic Leukemia Acute Leukemia Preleukemia	Indolent Lymphoma Aggressive Lymphoma
2007	~38 Leukemia types identified: Acute myeloid leukemia (~12 types) Acute lymphoblastic leukemia (2 types) Acute promyelocytic leukemia (2 types) Acute monocytic leukemia (2 types) Acute erythroid leukemia (2 types) Acute megakaryoblastic leukemia Acute myelomonocytic leukemia (2 types) Chronic myeloid leukemia Chronic myeloproliferative disorders (5 types) Myelodysplastic syndromes (6 types) Mixed myeloproliferative/myelodysplastic syndromes (3 types)	~51 Lymphomas identified: Mature B-cell lymphomas (~14 types) Mature T-cell lymphomas (15 types) Plasma cell neoplasm (3 types) Immature (precursor) lymphomas (2 types) Hodgkin's lymphoma (5 types) Immunodeficiency associated lymphomas (~5 types) Other hematolymphoid neoplasms (~7 types)

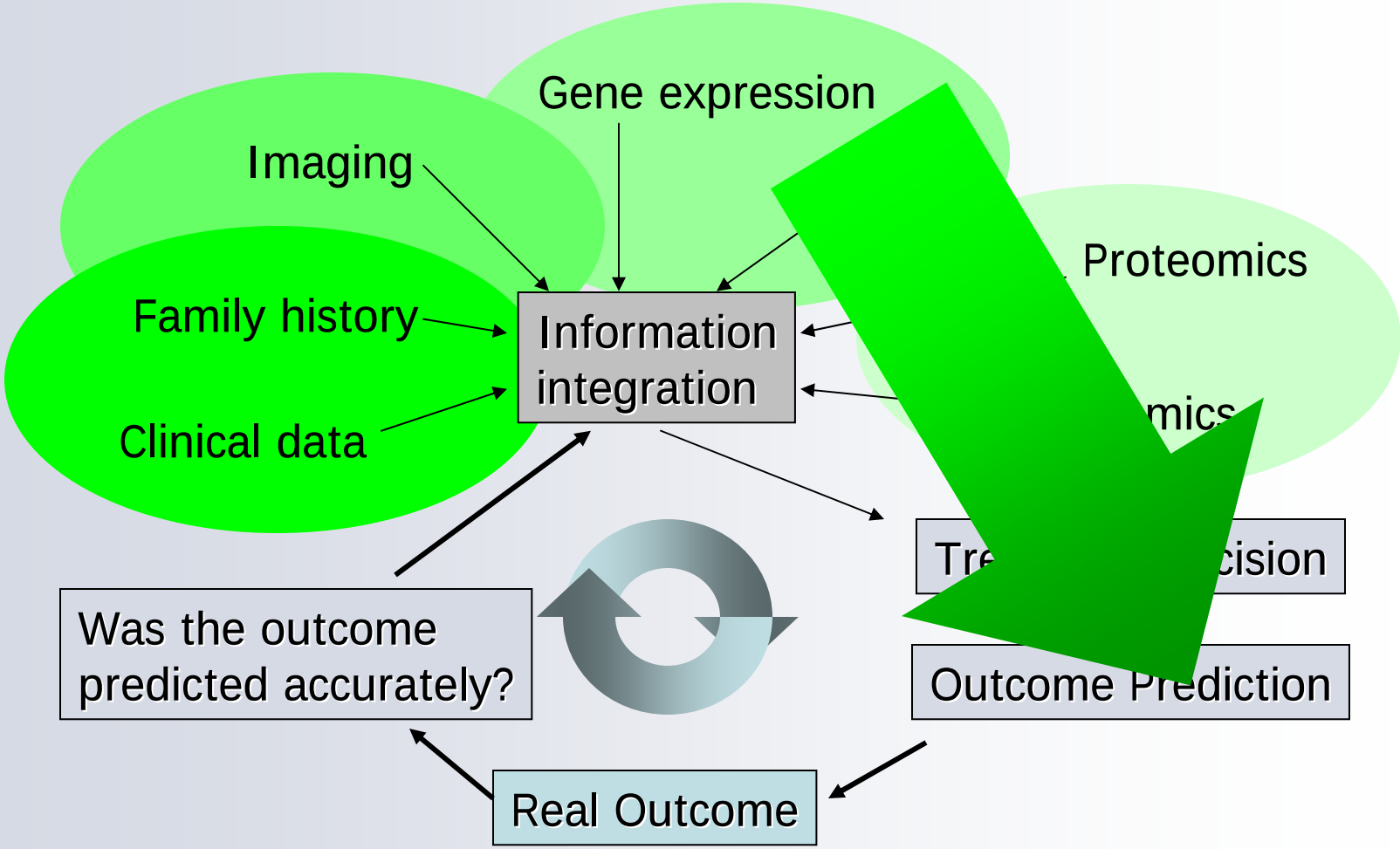
5 Year Survival

~ 0 %

~ 70 %

After Mara Aspinall, Genzyme Genetics (modified)

Idea: Use Molecular Markers to Make Better Treatment Decisions



WSJ.com THE WALL STREET JOURNAL. ONLINE

August 16, 2007

PAGE ONE

PERSONAL DOSE

In Milestone, FDA Pushes Genetic Tests Tied to Drug

Agency Seeks to Tame Risks of Blood Thinner; Some Doctors Protest

U.S.News & WORLD REPORT

Saturday, September 29, 2007

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Genetic Test Approved for Sensitivity to Blood Thinner

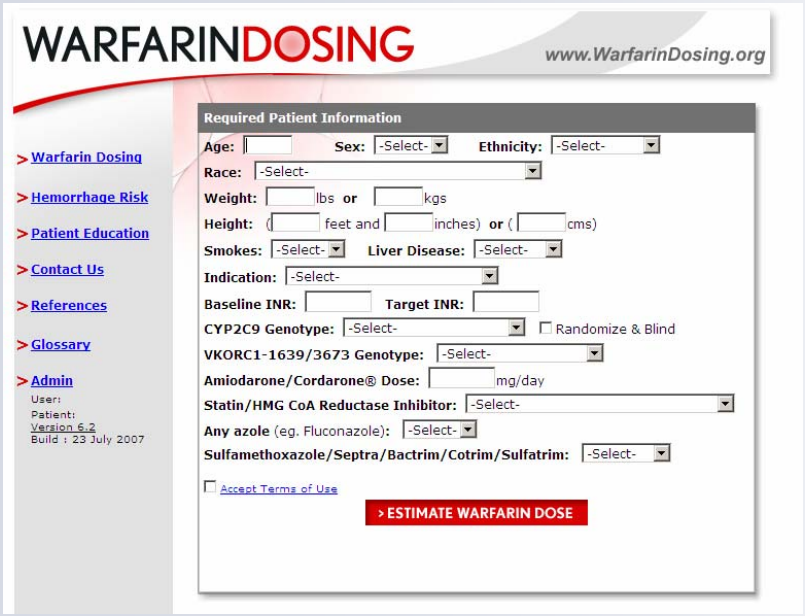
Some people who take Coumadin at higher risk of bleeding

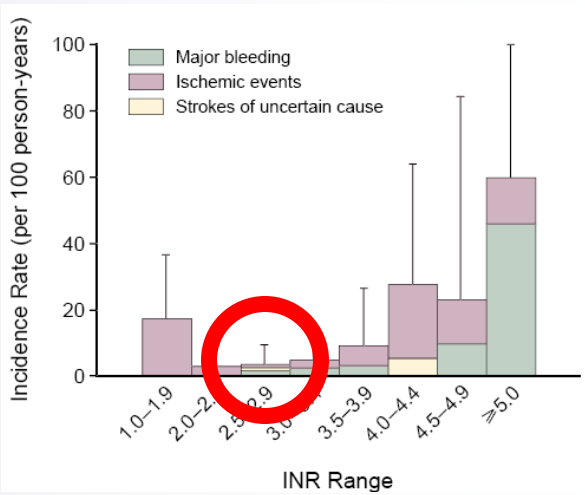
The lower initiation doses should be considered for patients with certain genetic variations in CYP2C9 and VKORC1 enzymes as well as for elderly and/or debilitated patients and patients with potential to exhibit greater than expected PT/INR responses to COUMADIN (see **CLINICAL PHARMACOLOGY** and **PRECAUTIONS**).

http://www.fda.gov/cder/foi/label/2007/009218s105lblv2.pdf

CYP2C9 and VKORC1 Testing – Better Estimation of Warfarin Starting Dose

Genetics and other clinical factors can help to assess approx. 60 percent of the variability in warfarin dose



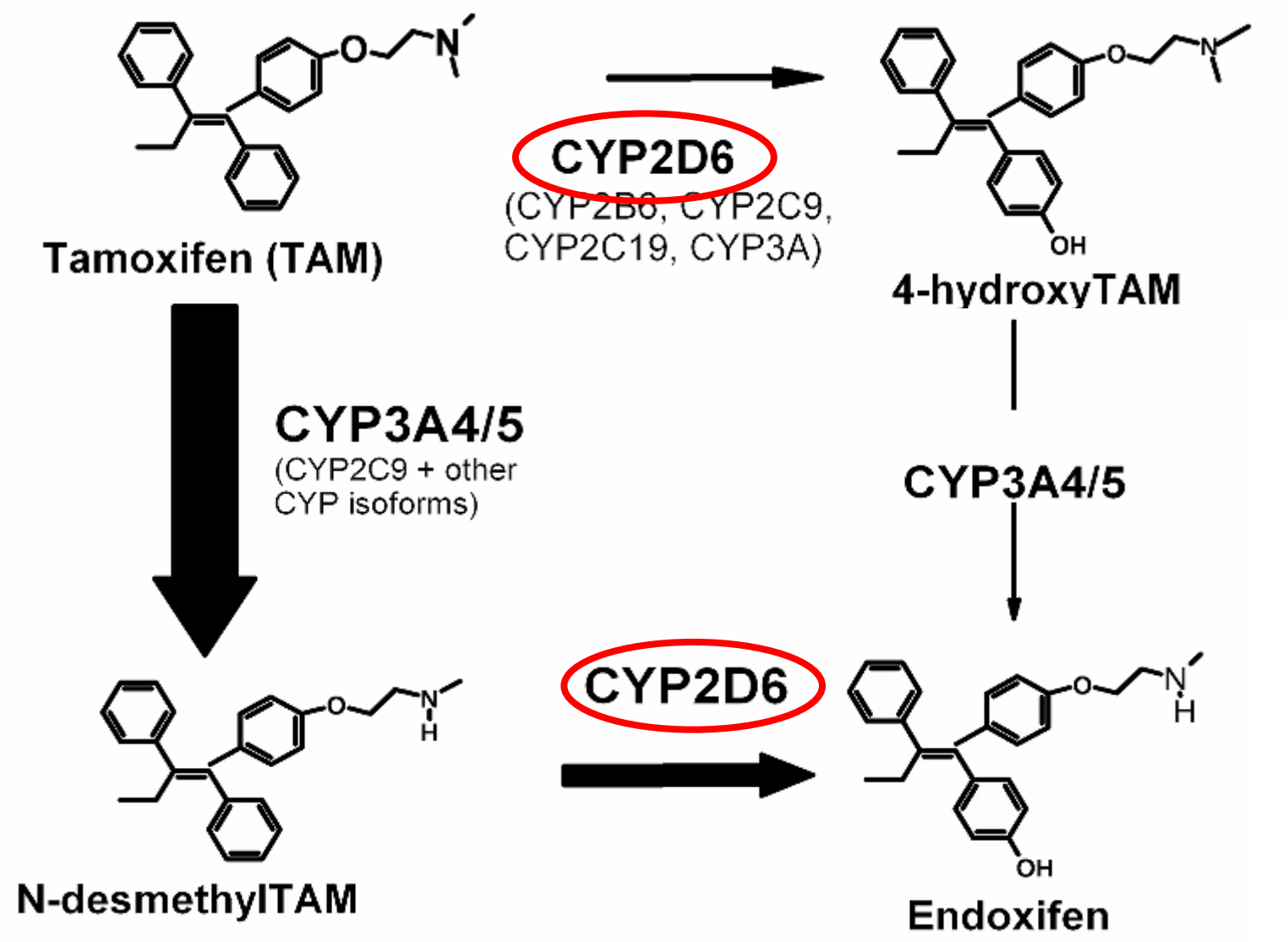


N Engl J Med 1995; 333: 5-10

KEY POINTS TO KEEP IN MIND…

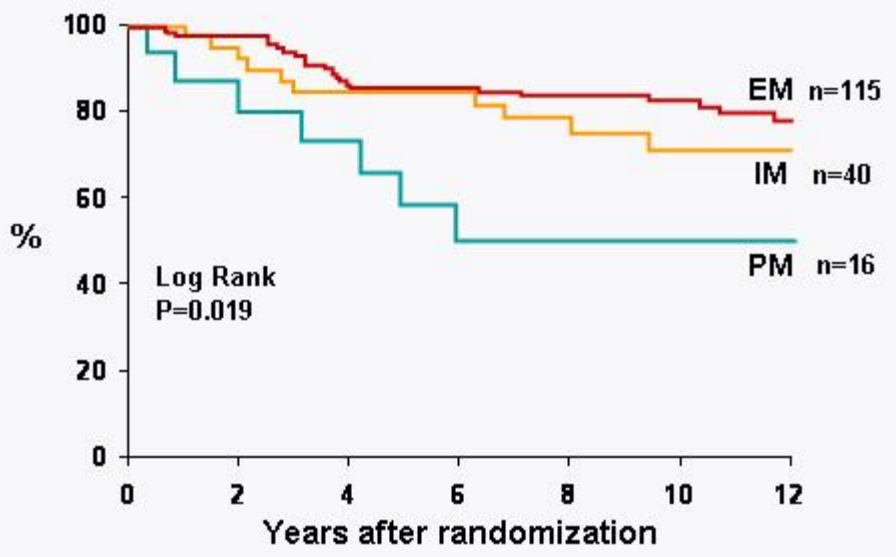
- ***Genetic tests not required***
- ***Encourage doctors to consider genetics in initial warfarin doses***
- ***Genetic tests are available***
- ***Prevalence of genetic variants in different ethnic/racial groups***
- ***Non-genetic factors also important***
- ***INR monitoring is still essential***

Tamoxifen Metabolic Pathway



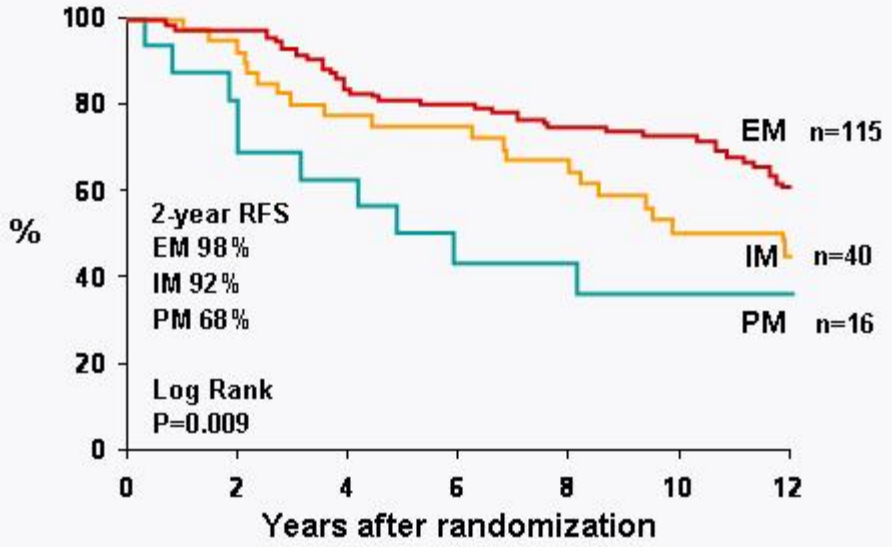
Jin, Y. et al. J Natl Cancer Inst 2005;97:30-39

Time to Breast Recurrence



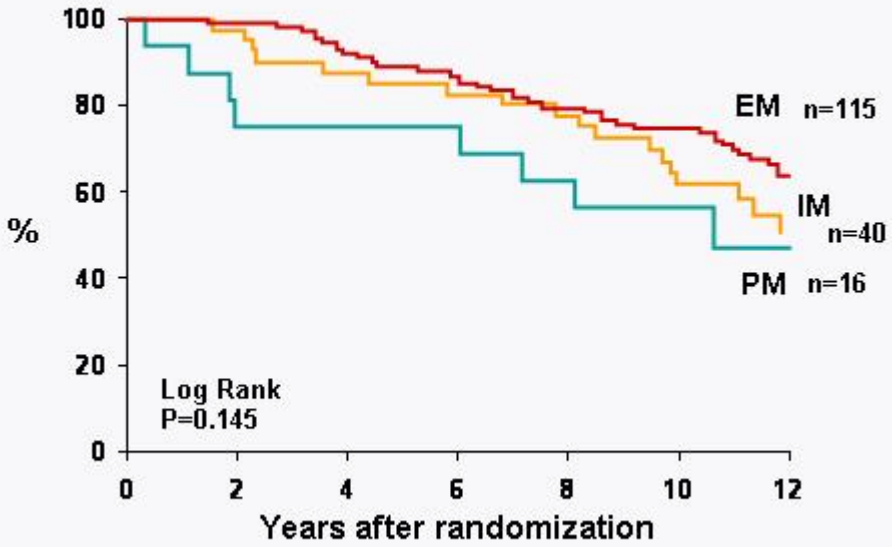
Goetz et al. Breast Cancer Res Treat. 2006 (In press)

Relapse-Free Survival



Goetz et al. Breast Cancer Res Treat. 2006 (In press)

Overall Survival



Goetz et al. Breast Cancer Res Treat. 2006 (In press)

Conclusion

- In this trial, CYP2D6 metabolism was an independent predictor of clinical outcome in postmenopausal women with ER positive early breast cancer
- The effect of impaired metabolism was most marked in poor metabolizers
- Consistent with clinical data that tamoxifen activation to endoxifen is dependent upon CYP2D6
- These data suggest that determination of CYP2D6 genotype may be of value in selecting adjuvant hormonal therapy and moderate/potent CYPY2D6 inhibitors should not be co-administered with tamoxifen

Useful, because alternatives exist: Hormonal Therapies of Breast Cancer

- Selective Estrogen Receptor Modulator
  - Tamoxifen
- Aromatase Inhibitors
  - Anastrazole (Arimidex)
  - Letrozole (Femara)
  - Exemestane (Aromasin)

Table of Valid Genomic Biomarkers in the Context of Approved Drug Labels

Pharmacogenomic information is contained in about ten percent of labels for drugs approved by the FDA. A significant increase of labels containing such information has been observed over the last decade. In order to provide a reference for genomic biomarkers in labels of FDA-approved drug products, we created the table shown below. Genomic biomarkers can play an important role in identifying responders and non-responders, avoiding toxicity and adjusting the dosage of drugs to optimize their efficacy and safety. In the context of drug labels, these genomic biomarkers can be classified on the basis of their specific use, for example:

- Clinical response and differentiation,
- Risk identification,
- Dose selection guidance,
- Susceptibility, resistance and differential disease diagnosis,
- Polymorphic drug targets.

The table portrays a view on valid genomic biomarkers in the context of FDA-approved drug labels. It provides a comprehensive list of these markers and links to pharmacogenomic data, taking into account multiple regulatory contexts in which these biomarkers were approved. Most drug labels in this table provide pharmacogenomic information with no immediate recommendation for a specific action (i.e. genetic testing); however a few labels recommend or require genetic testing thereby specifying the use of these markers for reaching a therapeutic decision.

The table includes:

- Context-specific biomarker (column 1)
- Reference drug label information about the biomarker context within which the drug was approved (column 2 subsection 1)
- Test criteria (column 2 subsection 2)
- Prototypic drug associated with the label information defining the biomarker context (column 2 subsection 3)
- Other drugs in a similar context (column 3)
- Pertinent references (column 4).

Drugs sharing the context of a specific biomarker in their labels have had their pharmacogenomic information extracted into this table. This information can be accessed by placing the mouse over the symbol under the right side of the drug name. All approved drugs in this table are linked to labels at Drugs@FDA which can be accessed by clicking over symbols under the left side of the drug name. The table will be updated on a quarterly basis.

The information provided in "label context" is taken from different sections of the actual drug labels.

The term "valid" biomarker has been defined in the "Guidance for Industry: Pharmacogenomic Data Submissions". Therein, a valid biomarker is described as a "biomarker that is measured in an analytical test system with well established performance characteristics and for which there is an established scientific framework or body of evidence that elucidates the physiologic, toxicologic, pharmacologic, or clinical significance of the test results." The classification of biomarkers is context specific.

A critical aspect of many of these drugs is the role they play in drug-drug interactions. This list does not address drug-drug interactions. More information on drug-drug interactions, please see Drug Development and Drug Interactions.

Reference is made to the requirement of testing for the biomarker:
1 = test required;
2 = test recommended;
3 = information only

Biomarker	Label Context	Examples of other	References

Reference is made to the requirement of testing for the biomarker:
1 = test required;
2 = test recommended;
3 = information only

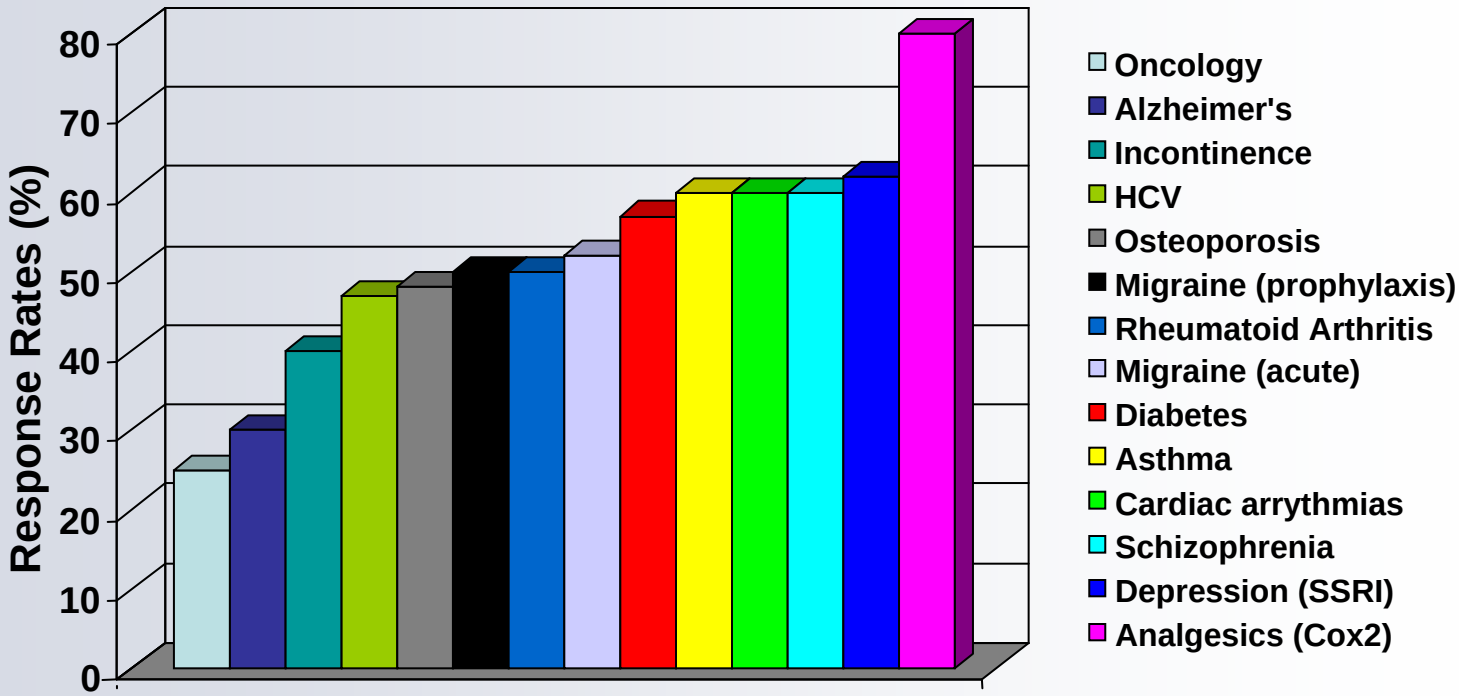
Biomarker	Label Context			Examples of other Drugs Associated with this Biomarker	References (PubMed ID)
	Representative Label	**Test**	**Drug**		
C-KIT expression	**Gastrointestinal stromal tumor *c-Kit* expression** "*In vitro*, imatinib inhibits proliferation and induces apoptosis in gastro-intestinal stromal tumor (GIST) cells, which express an activating c-kit mutation." "Gleevec is also indicated for the treatment of patients with Kit (CD117) positive unresectable and/or metastatic malignant gastrointestinal stromal tumors (GIST)."	**3**	Imatinib mesylate		12851888 16226710 16294026
CYP2C19 Variants	**CYP2C19 Variants (Poor Metabolizers-PM and Extensive Metabolizers-EM) with genetic defect leads to change in drug exposure**. "*In vivo* studies indicated that CYP2C19 is significantly involved in the metabolism of voriconazole. This enzyme exhibits genetic polymorphism. For example, 15-20% of Asian populations may be expected to be poor metabolizers. For Caucasians and Blacks, the prevalence of poor metabolizers is 3-5%. Studies conducted in Caucasian and Japanese healthy subjects have shown that poor metabolizers have, on average, 4-fold higher voriconazole exposure (AUCτ) than their homozygous extensive metabolizer counterparts. Subjects who are heterozygous extensive metabolizers have, on average, 2-fold higher voriconazole exposure than their homozygous extensive metabolizer counterparts."	**3**	Voriconazole	Omeprazole[m1] Pantoprazole [m2] Esomeprazole[m3] diazepam[m4] Nelfinavir[m5] Rabeprazole[m6]	12867215 11866669
CYP2C9 Variants	**CYP2C9 Variants PM and EM genotypes and drug exposure**; "Patients who are known or suspected to be P450 2C9 poor metabolizers based on a previous history should be administered celecoxib with caution as they may have abnormally high plasma levels due to reduced metabolic clearance."	**3**	Celecoxib	Warfarin[m7]	16118328 15637526 15714076 15037866 14558433
CYP2D6 Variants	**CYP2D6 Variants** "Atomoxetine is metabolized primarily through the CYP2D6 enzymatic pathway. People with reduced activity in this pathway (PMs) have higher plasma concentrations of atomoxetine compared with people with normal activity (EMs)."	**3**	Atomoxetine	Venlafaxine;[m8] Risperidone;[m9] Tiotropium bromide inhalation;[m10] Tamoxifen;[m11] Timolol Maleate;[m12]	
CYP2D6 with alternate Context	**CYP2D6 PM and EM Variants and drug exposure and risk**- "population, who are known to have a genetic defect leading to reduced levels of activity of P450 2D6. Fluoxetine, like other agents that are metabolized by P450IID6, inhibits the activity of this isoenzyme, and thus may make normal metabolizers resemble "poor metabolizers." Therapy with medications that are predominantly metabolized by the P450IID6 system and that have a relatively narrow therapeutic index should be initiated at the low end of the dose range if a patient is receiving fluoxetine concurrently or has taken it in the previous 5 weeks."	**3**	Fluoxetine HCL	Fluoxetine HCL and Olanzapine;[m13] Cevimeline hydrochloride[m14] Tolterodine;[m15] Terbinafine;[m16] Tramadol + Acetamophen [m17] Clozapine[m18] Aripiprazole;[m19] Metoprolol;[m20]	16472103 16384813; 15063083; 16271013 16236141 15828850 15492763 15037866 14639062 10431214 1302039

In the Works

- *New guidance* for industry on “Clinical Pharmacogenomics in early drug development”
- Related to PK/PD and Pharmacogenomics (e.g. what should we do with pharmacogenomics and drug metabolism genotypes)
- Determine:
  - Details on "what are the questions" (i.e., the goals of a PGx study)
  - How to go about getting results that matter (i.e., study designs and the use of M/S to design adequate studies)
  - What to do with the results of PGx studies (i.e., data analysis and labeling)
- *Planned to have a draft ready in early 2008*

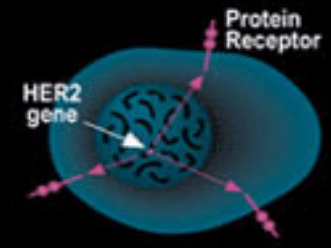
The Right Drug for the Wrong Patient ?

The response rate to current medicines is often unacceptably low:

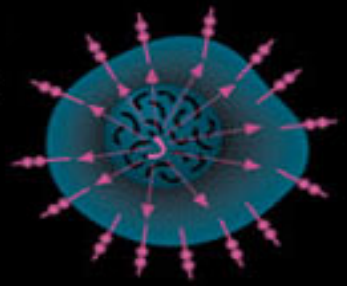


After *Spear et al. TRENDS in Molecular Medicine Vol.7 No.5 May 2001*

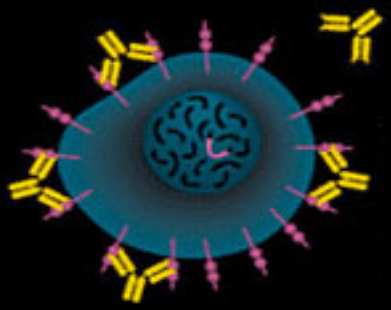
Trastuzumab (Herceptin®)



In a normal breast tissue cell, the Her-2 gene is expressing cell surface receptor required for normal cell growth.



In certain types of breast cancers, the Her-2 gene is **over-expressing** this cell surface receptor, contributing to cancerous cell growth.
This is the case in ~30% of breast cancers.



Herceptin (trastuzumab) is an antibody that blocks the cell surface receptor and thereby prevents further growth. As a result, disease progression is slowed down.

Gefitinib (Iressa)

- Selective inhibitor of EGFR tyrosine kinase domain
- Approved under sub-part H (accelerated approval) for treatment of NSCLC in 2004
- In Dec 2004, pivotal trial (ISEL) did not show survival benefit over placebo
- Nevertheless a subset of patient (~10%) showed significant improvements
- Market witdrawal and access program put in place in 2005
- Current indication: IRESSA is indicated as monotherapy for the continued treatment of patients with locally advanced or metastatic non-small cell lung cancer after failure of both platinum-based and docetaxel chemotherapies who are benefiting or have benefited from IRESSA

Gefitinib (Iressa), cont'd

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 MAY 20, 2004 VOL. 350 NO. 21

Activating Mutations in the Epidermal Growth Factor Receptor Underlying Responsiveness of Non–Small-Cell Lung Cancer to Gefitinib

Thomas J. Lynch, M.D., Daphne W. Bell, Ph.D., Raffaella Sordella, Ph.D., Sarada Gurubhagavatula, M.D., Ross A. Okimoto, B.S., Brian W. Brannigan, B.A., Patricia L. Harris, M.S., Sara M. Haserlat, B.A., Jeffrey G. Supko, Ph.D., Frank G. Haluska, M.D., Ph.D., David N. Louis, M.D., David C. Christiani, M.D., Jeff Settleman, Ph.D., and Daniel A. Haber, M.D., Ph.D.

Gefitinib (Iressa), cont'd

- New *exploratory* biomarkers for prediction of response to gefitinib have been identified:
- **Genetic variations in tumor**
  - Positive results of (small) prospective trials
    - Example: results published at ASCO 2007; abstract #7504, Sequist et al; 31 patients with genetic variations in EGFR treated; RR 58% - problem is, that there are no matched controls, i.e. we don't know how a patient with the same genetic variation would progress without treatment
- **EGFR gene copy number**
  - In same study, 71% of treated patients had also gene amplification or polysomy
  - Several other reports illustrate that gene copy number (FISH) could be an important predictive factor for gefitinib therapy

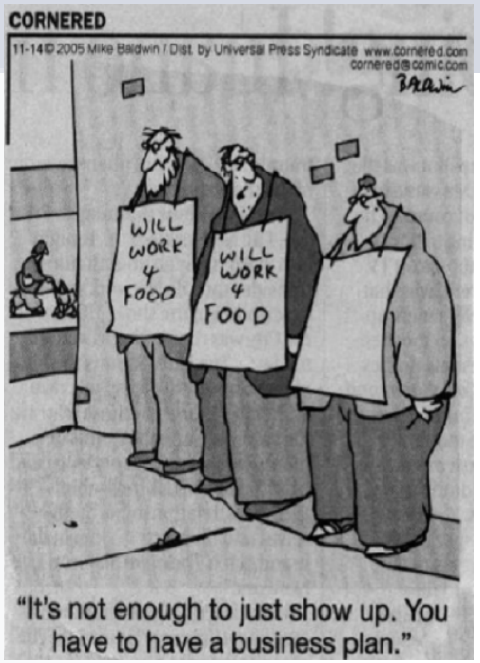
Some Key Questions About Getting This Evidence and Consequences

- At what point is retrospective data good enough?
  - E.g., recent warfarin study results confirm conclusions reached two years ago based on retrospective data
  - How can we better use existing data sources?
- When are randomized controlled trials to create the evidence for genetic testing really needed?
  - E.g., warfarin trial: when should genetic test be performed?
- But: multivariate problem with highly complex tests: how to avoid random and meaningless associations?

Breaking Down the Barriers

- **… two fundamental aspects of personalized medicine that don't fit our current paradigm of drug development and approval:**
  - "Superiority" on a population basis does not necessarily reflect the best choice for an individual

    (A treatment with a 10% advantage over a comparator may still be the wrong treatment for a lot of people)
  - "Low efficacy" can still mean that a subset of patients has a dramatic response – how can we ensure that these patients are identified and the drug is being developed ?
- ***New and innovative approaches are needed…***



Genome-wide SNP Analyses

The Pharmacogenomics Journal (2007), 1–10

www.nature.com/tpj

ORIGINAL ARTICLE

Genome-wide pharmacogenetic investigation of a hepatic adverse event without clinical signs of immunopathology suggests an underlying immune pathogenesis

A Kindmark[1], A Jawaid[2], CG Harbron[2], BJ Barratt[2], OF Bengtsson[1], TB Andersson[1], S Carlsson[1], KE Cederbrant[3], NJ Gibson[2], M Armstrong[2], ME Lagerström-Fermér[1], A Dellsén[1], EM Brown[2], M Thornton[2], C Dukes[2], SC Jenkins[2], MA Firth[2], GO Harrod[2], TH Pinel[2], SME Billing-Clason[1], LR Cardon[4] and RE March[2]

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E-mail: Ruth.March@astrazeneca.com

One of the major goals of pharmacogenetics is to elucidate mechanisms and identify patients at increased risk of adverse events (AEs). To date, however, there have been only a few successful examples of this type of approach. In this paper, we describe a retrospective case–control pharmacogenetic study of an AE of unknown mechanism, characterized by elevated levels of serum alanine aminotransferase (ALAT) during long-term treatment with the oral direct thrombin inhibitor ximelagatran. The study was based on 74 cases and 130 treated controls and included both a genome-wide tag single nucleotide polymorphism and large-scale candidate gene analysis. A strong genetic association between elevated ALAT and the MHC alleles DRB1*07 and DQA1*02 was discovered and replicated, suggesting a possible immune pathogenesis. Consistent with this hypothesis, immunological studies suggest that ximelagatran may have the ability to act as a contact sensitizer, and hence be able to stimulate an adaptive immune response.
The Pharmacogenomics Journal advance online publication, 15 May 2007; doi:10.1038/sj.tpj.6500458

Keywords: pharmacogenetics; pharmacogenomics; adverse event; immune system; liver injury

Introduction

Ximelagatran, marketed as Exanta, was developed for the prevention and treatment of thromboembolism. In patients treated with ximelagatran for more than 35 days, transient elevated levels of serum alanine aminotransferase (ALAT)

"Our data further suggest that a biomarker test based on DRB1*07 would have been able to detect patients at risk of the AE with sensitivity of 47% and specificity of 83%."

What does FDA think?

If at-risk patients can be excluded, a suspected hepatotoxic drug would be potentially approvable, in the context of the overall risk/benefit analysis for the drug.

Whole Genome Scans

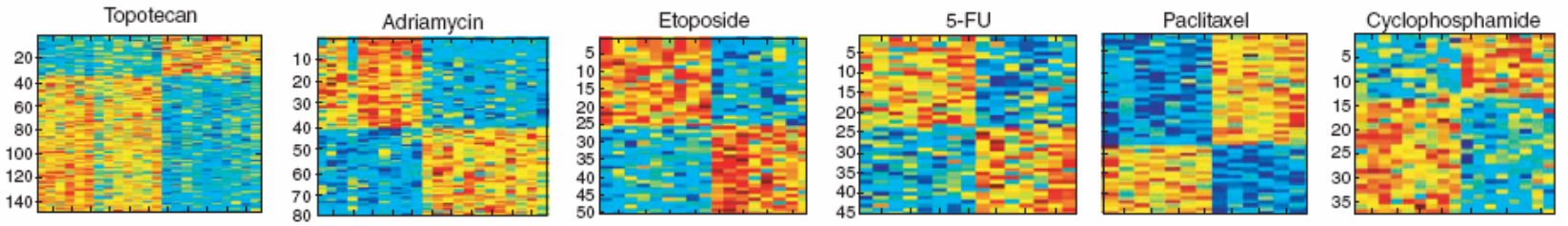
"Man, that record came out and was real big in Memphis. They started playing it, and it got real big. Don't know why – the lyrics had no meaning."

Elvis Presley

New gene expression approaches to guide the use of existing chemotherapy

Genomic signatures to guide the use of chemotherapeutics

Anil Potti[1,2], Holly K Dressman[1,3], Andrea Bild[1,3], Richard F Riedel[1,2], Gina Chan[4], Robyn Sayer[4], Janiel Cragun[4], Hope Cottrill[4], Michael J Kelley[2], Rebecca Petersen[5], David Harpole[5], Jeffrey Marks[5], Andrew Berchuck[1,6], Geoffrey S Ginsburg[1,2], Phillip Febbo[1–3], Johnathan Lancaster[4] & Joseph R Nevins[1–3]



Potti et al Nature Medicine (2006) 12(11):1294-1300

Genomic signatures to guide the use of chemotherapeutics

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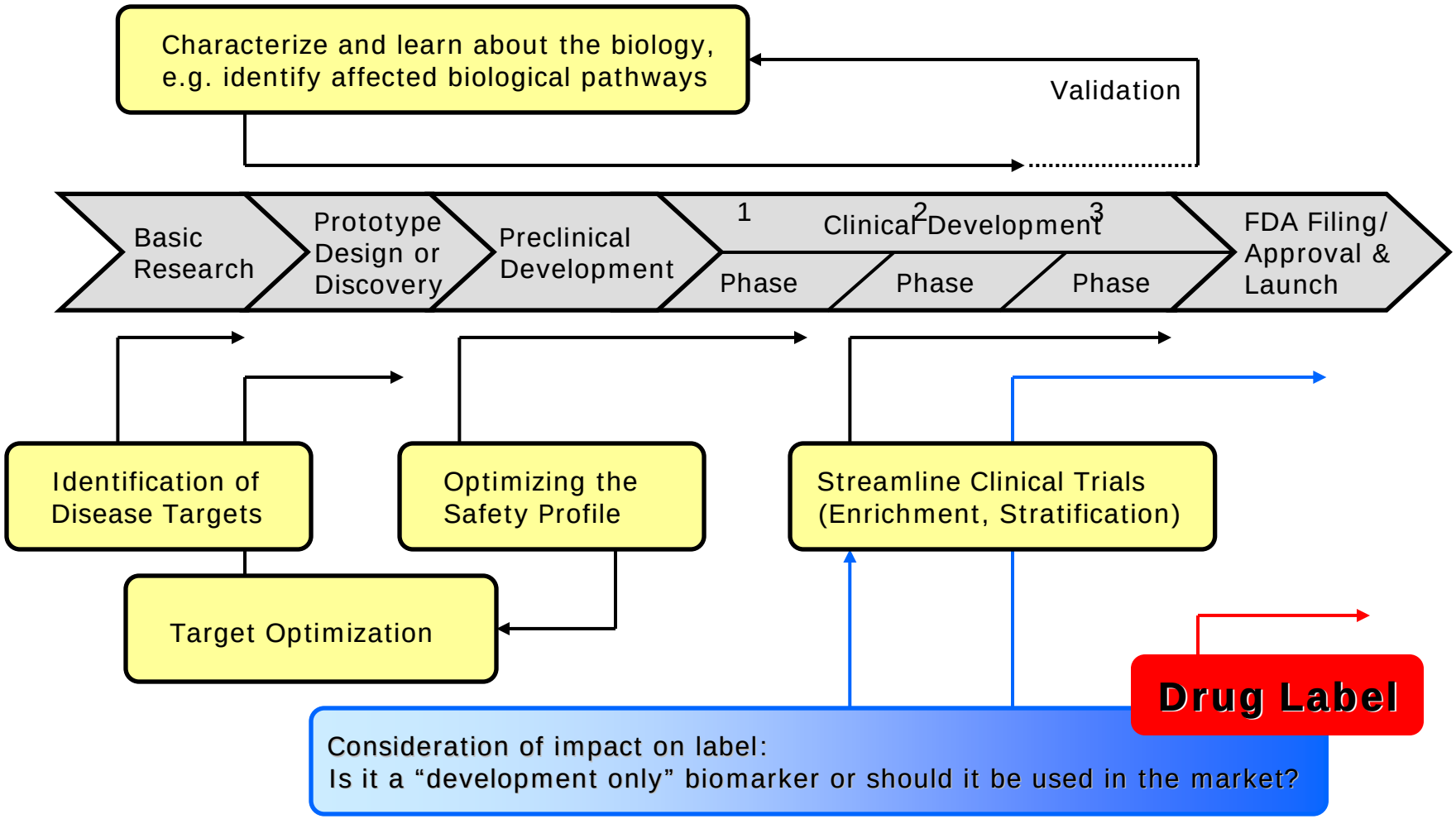
- Gene expression signatures predict sensitivity to individual chemotherapeutic drugs
- Signatures can accurately predict clinical response
- When combined, could also predict response to multidrug regimens
- "The development of gene expression profiles that can predict response to commonly used cytotoxic agents provides opportunities to **better use** these drugs, including using them in combination with existing targeted therapies"

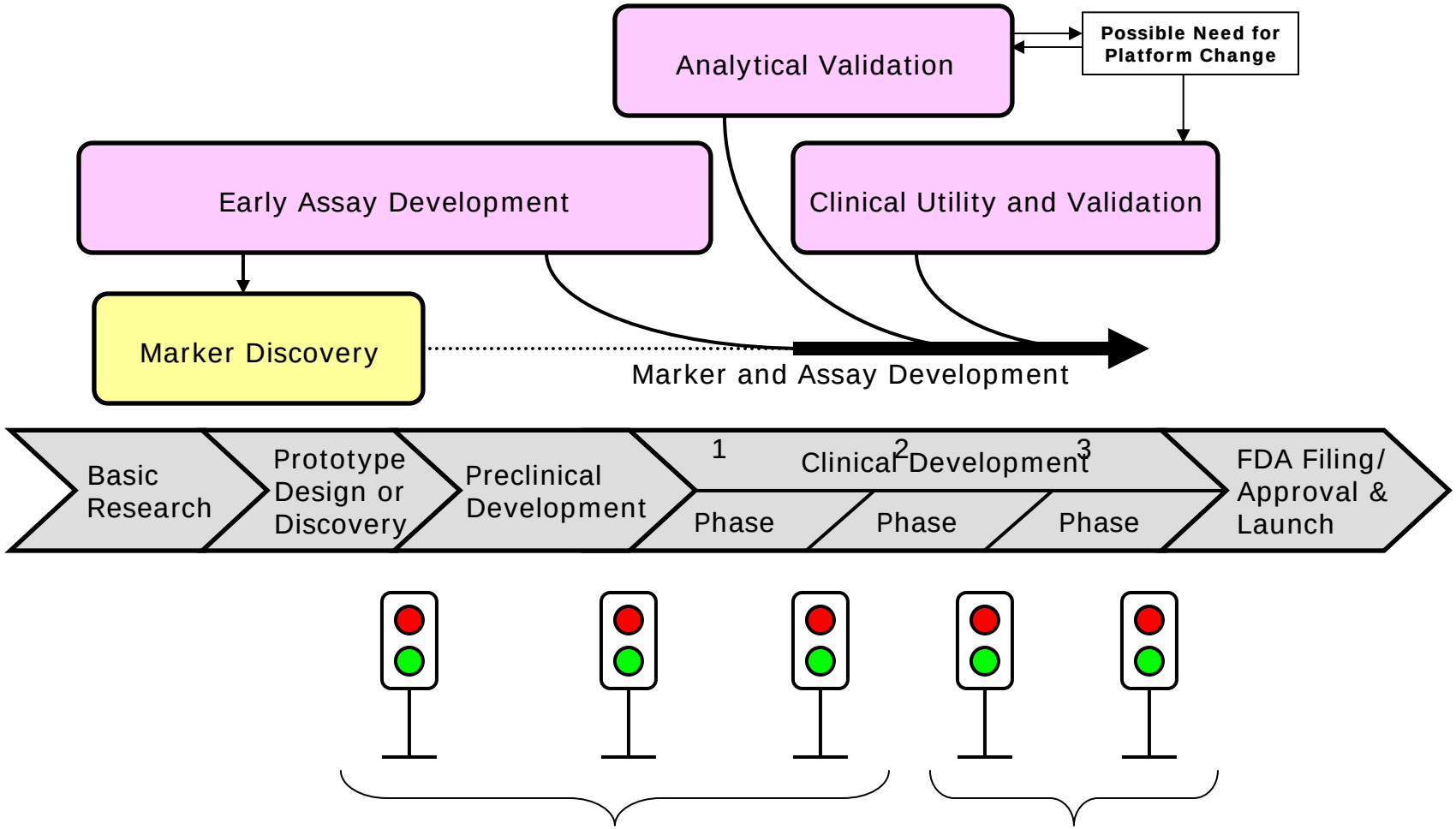
  → ***Useful for drug selection !***

- But how can we **better develop** these drugs in the first place?
  - *Drug-Test Co-Development:* making the biomarker an integral part of the drug development process

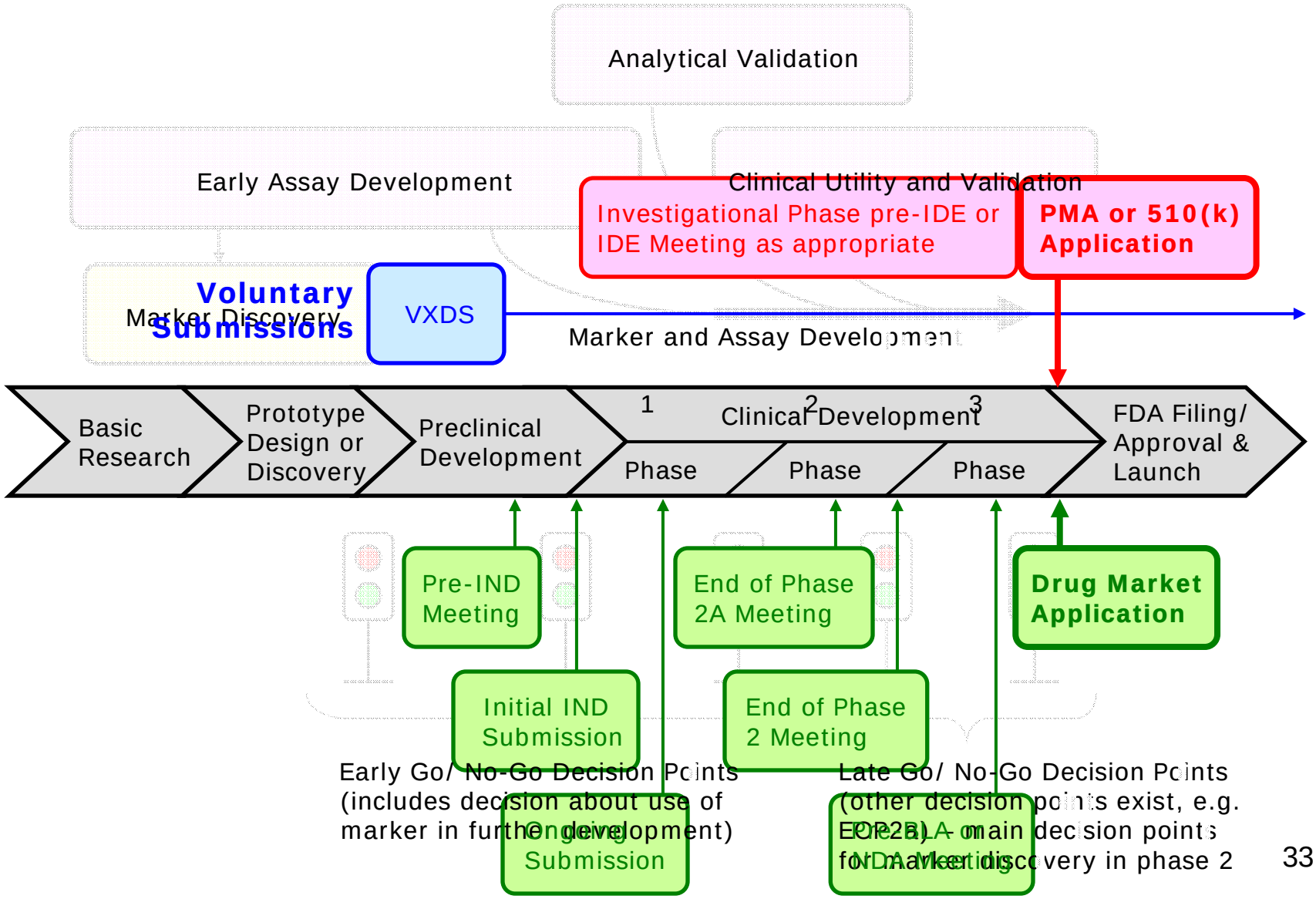
Drug-Test Co-Development



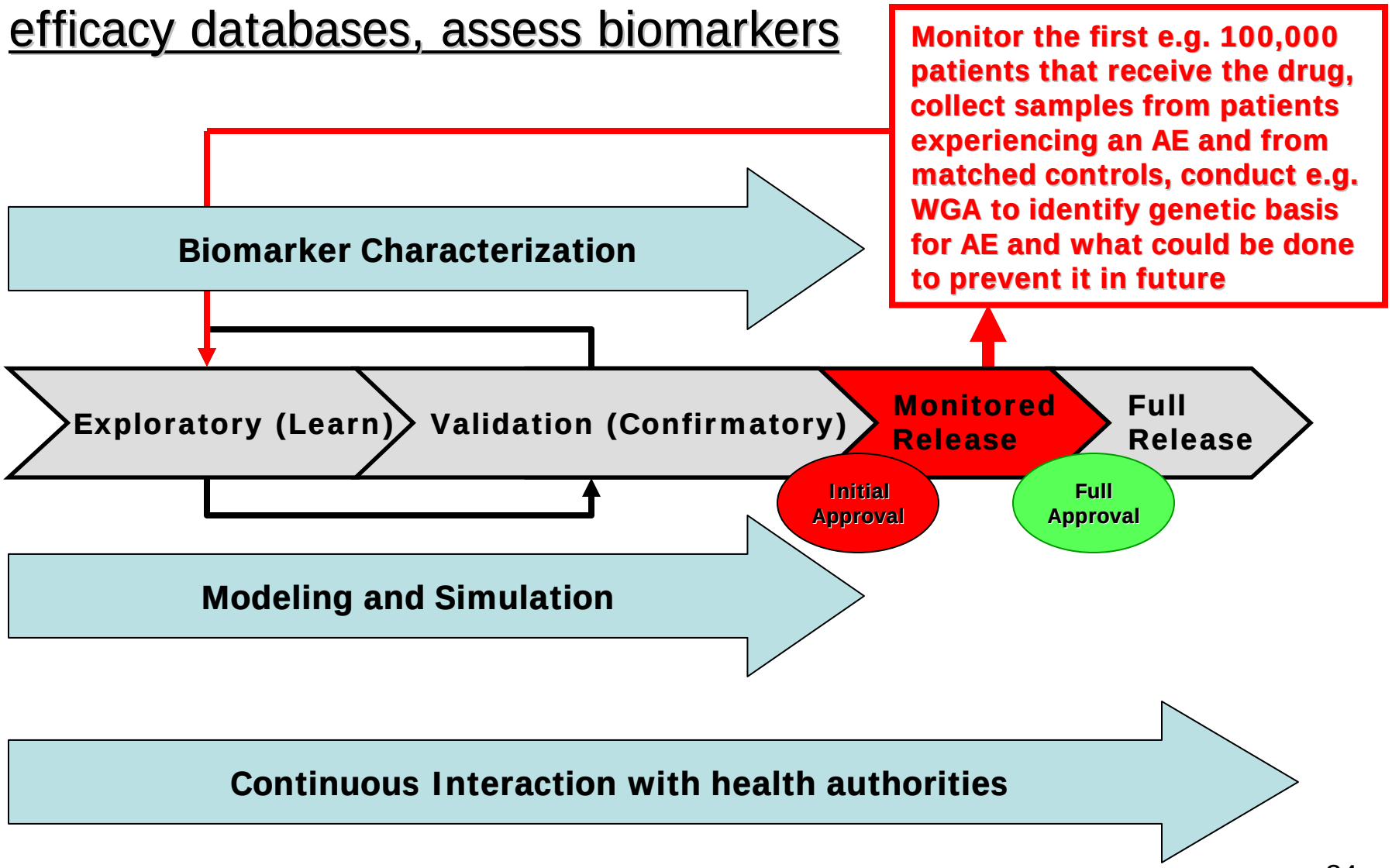
Biomarker and assay development process



Sponsor – Regulator Interactions



But why stop learning when the drug is on the market ? A proposal to create larger safety and efficacy databases, assess biomarkers



What We Could Learn Using this Approach

- Who really benefits from a particular treatment
- Who might be at risk for an adverse event
  - (this is the only strategy that would help us to learn more about the molecular mechanisms of rare adverse events: "retrospective" sample collection approaches do not work)
- If indeed we have the right dose
- Comparative effectiveness
- Clinical utility of testing (reimbursement?)
- Actual response rate and what factors may influence it
- Aspects of compliance
- How to educate physicians about molecular medicine
- ...

Translation into Clinical Practice –

The two Elephants in the Room:

Reimbursement – How Much Evidence Is Needed ?

Alternative Reimbursement

"Drug companies like to say that their most expensive products are fully worth their breathtaking prices. Now one company is putting its money where its mouth is — by offering a ***money-back guarantee****.*

Johnson & Johnson has proposed that ***Britain's national health service pay for the cancer drug Velcade, but only for people who benefit*** *from the medicine, which can cost $48,000 a patient. The company would refund any money spent on patients whose tumors do not shrink sufficiently after a trial treatment."*

Pricing Pills by the Results - Andrew Pollack, The New York Times, July 14, 2007

Flipside

"I and others suggested a money-back guarantee on a cancer drug looked silly," said Dr. Tunis, who is now director of the nonprofit Center for Medical Technology Policy.

" 'Oh, I'm sorry your grandma died. Here's your money back.' "

Pricing Pills by the Results - Andrew Pollack, The New York Times, July 14, 2007

Transcription of Genes

During transcription, which occurs in the cell's nucleus, a messenger RNA (mRNA) strand is synthesized using the gene's DNA as a template. The double-stranded DNA opens up to expose each single strand.

The strand encoding the gene becomes the template for the synthesis of an mRNA strand.

The mRNA strand is synthesized by sequential addition of nucleotides that are complementary to those of the DNA template strand.

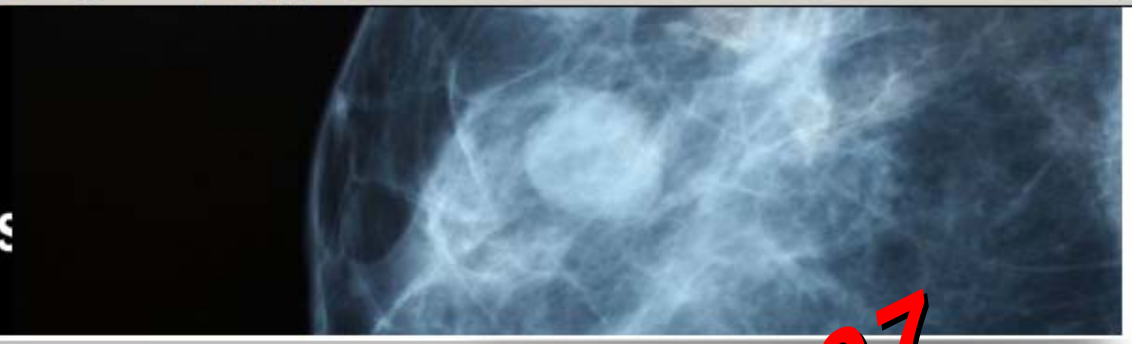
Play | Replay

After viewing the animation, click Next to continue.

THE FUTURE OF MEDICINE

PHARMACOGENOMICS

AN ONLINE COURSE



NAVIGATE

Section 1: Intro to Pharmacogenomics
Section 2: Analyzing Human Genome
Section 3: Biostatistical & Web
Section 4: Drug Metabolism
Section 5: Membrane Transporters
Section 6: Immune / Inflammatory
Section 7: CNS Drugs
Section 8: Oncology Agents
Section 9: Cardio
Section 12: Immunosuppressive Agents
Section 13: Drug Development

Pharmacogenomics and Biomarkers in On

by Felix W. Frueh, PhD

8

Section 8

Available December 2007

NEWS & EVENTS

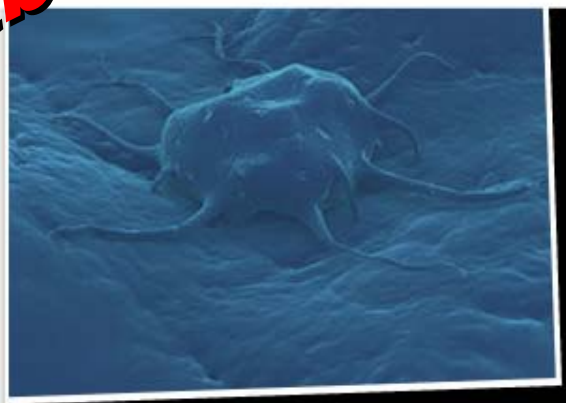
01.01.07: Article H

This is an example s article. This is the 1st slide.

The articles are short, punchy and can be updated quickly across the entire site.

LEGEND

- As you select a slide, this helix shows you which slide you are currently on.



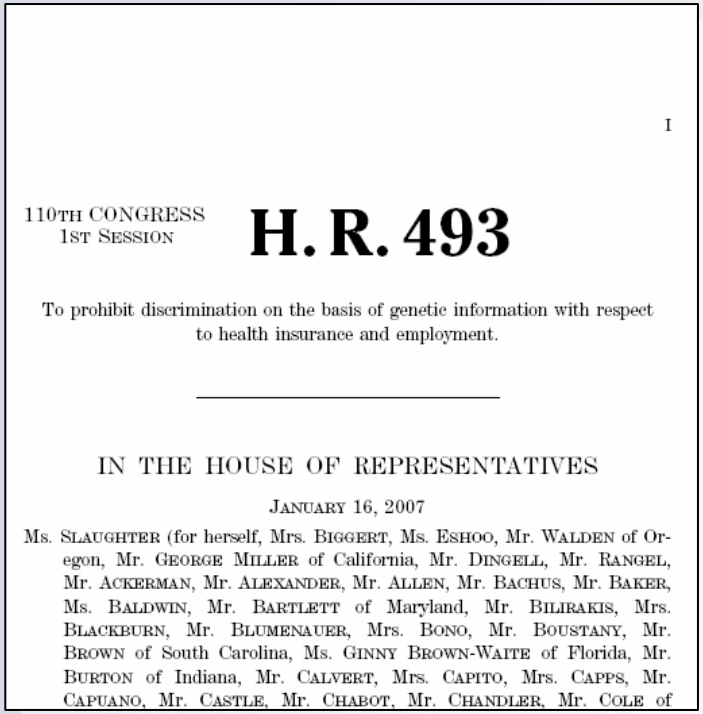
Progress in the translation of pharmacogenomic knowledge in drug development and clinical practice has been most rapid in the areas for which we understand, at least to some extent, the molecular mechanisms that lead to pathophysiology and, therefore, can be utilized to explore drug function.

Click Slides in order: 1 2 3 4 5 6 7 8

Lastly, New Legislation

For example:

- Genetic Information Nondiscrimination Act (GINA)
- Genomics and Personalized Medicine Act



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110TH CONGRESS
1ST SESSION

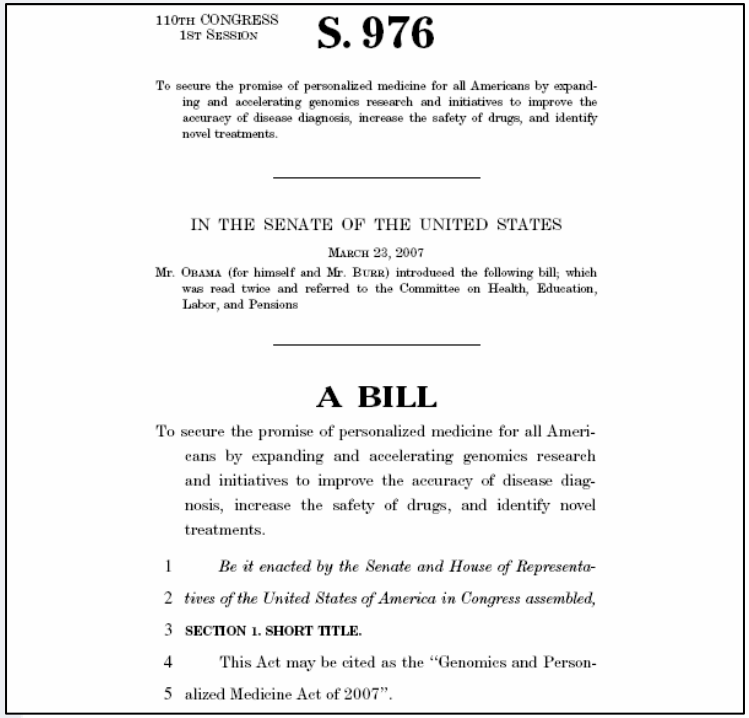
H. R. 493

To prohibit discrimination on the basis of genetic information with respect to health insurance and employment.

IN THE HOUSE OF REPRESENTATIVES

JANUARY 16, 2007

Ms. SLAUGHTER (for herself, Mrs. BIGGERT, Ms. ESHOO, Mr. WALDEN of Oregon, Mr. GEORGE MILLER of California, Mr. DINGELL, Mr. RANGEL, Mr. ACKERMAN, Mr. ALEXANDER, Mr. ALLEN, Mr. BACHUS, Mr. BAKER, Ms. BALDWIN, Mr. BARTLETT of Maryland, Mr. BILIRAKIS, Mrs. BLACKBURN, Mr. BLUMENAUER, Mrs. BONO, Mr. BOUSTANY, Mr. BROWN of South Carolina, Ms. GINNY BROWN-WAITE of Florida, Mr. BURTON of Indiana, Mr. CALVERT, Mrs. CAPITO, Mrs. CAPPS, Mr. CAPUANO, Mr. CASTLE, Mr. CHABOT, Mr. CHANDLER, Mr. COLE of



110TH CONGRESS
1ST SESSION

S. 976

To secure the promise of personalized medicine for all Americans by expanding and accelerating genomics research and initiatives to improve the accuracy of disease diagnosis, increase the safety of drugs, and identify novel treatments.

IN THE SENATE OF THE UNITED STATES

MARCH 23, 2007

Mr. OBAMA (for himself and Mr. BURR) introduced the following bill; which was read twice and referred to the Committee on Health, Education, Labor, and Pensions

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A BILL

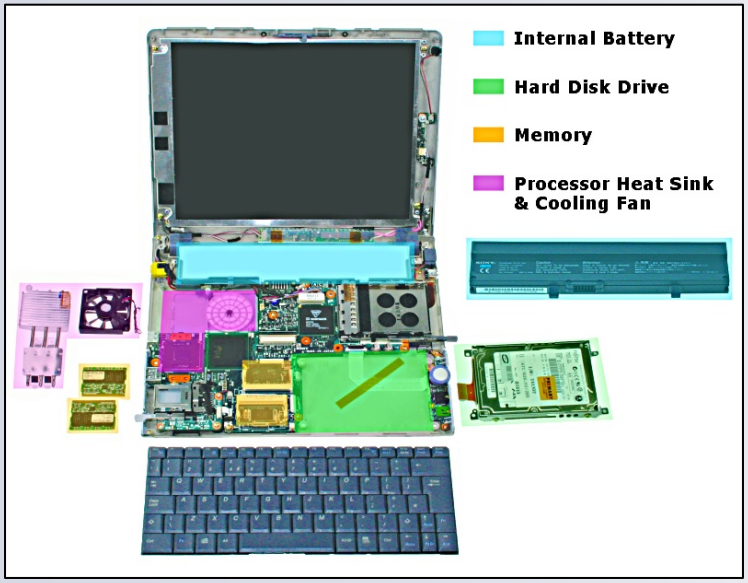
To secure the promise of personalized medicine for all Americans by expanding and accelerating genomics research and initiatives to improve the accuracy of disease diagnosis, increase the safety of drugs, and identify novel treatments.

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled,

SECTION 1. SHORT TITLE.

This Act may be cited as the "Genomics and Personalized Medicine Act of 2007".

Summary



All of the fruits of the tremendous explosion in innovation that's been occurring in biomedical research — which make the molecular metamorphosis possible — fulfill their purpose only when they are translated into interventions and solutions that are applied to patients.

Dr. A. von Eschenbach, April 6, 2006

THANK YOU !

www.fda.gov/cder/genomics

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Office of Clinical Pharmacology
FDA/CDER