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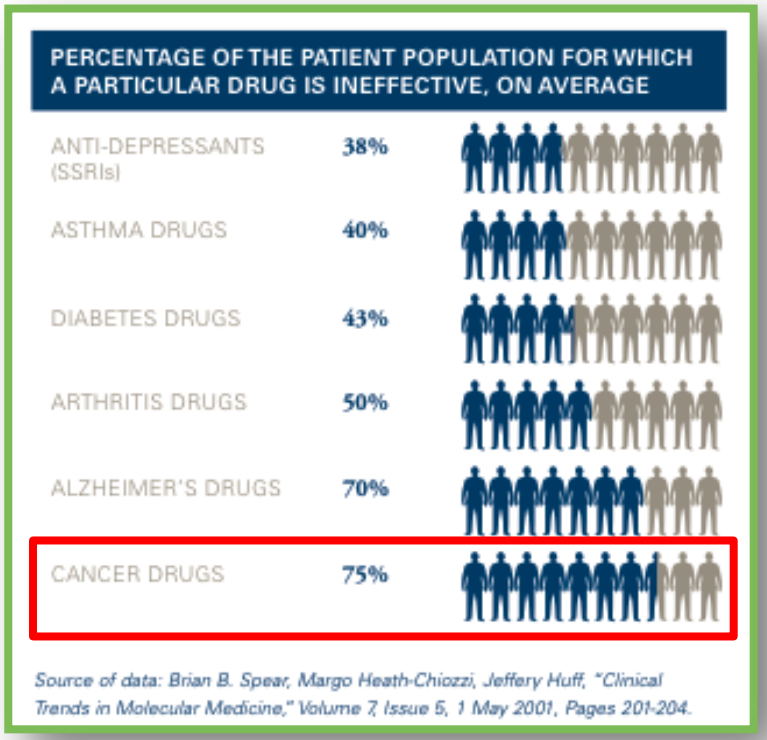
Technology To Make Personalized Cancer Therapy A Clinical Reality

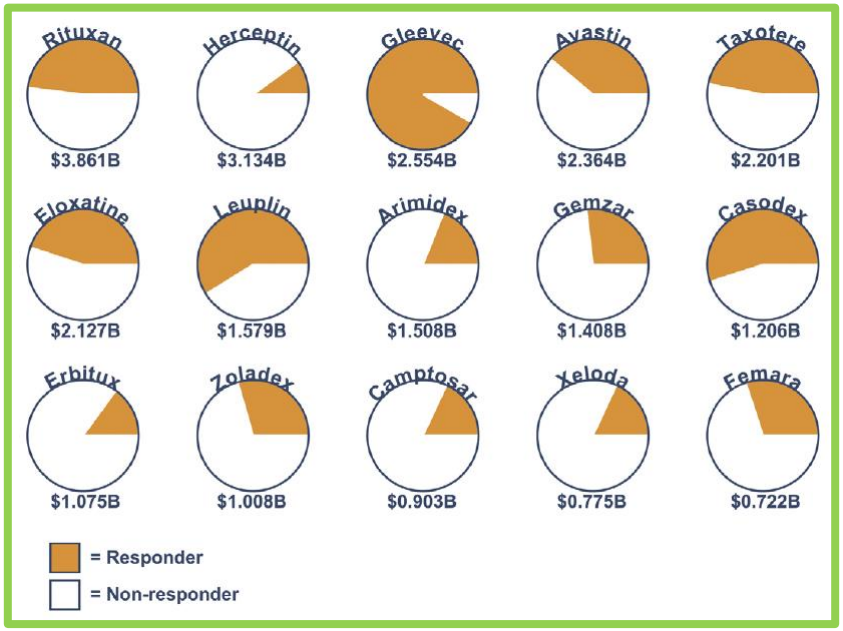
Eric M. Stone
Vice President, Sales & Marketing
May 7, 2013

Current Cancer Treatment Paradigm Leaves Room for Improvement

Cancer Drugs Effective in Only 25% of Patients



~$17.2B Spent on Non-Responders to Top 15 Cancer Drugs



Jackson, David. "Clinical and economic impact of the non-responder phenomenon – implications for systems based discovery," *Drug Discovery Today*. April 2009

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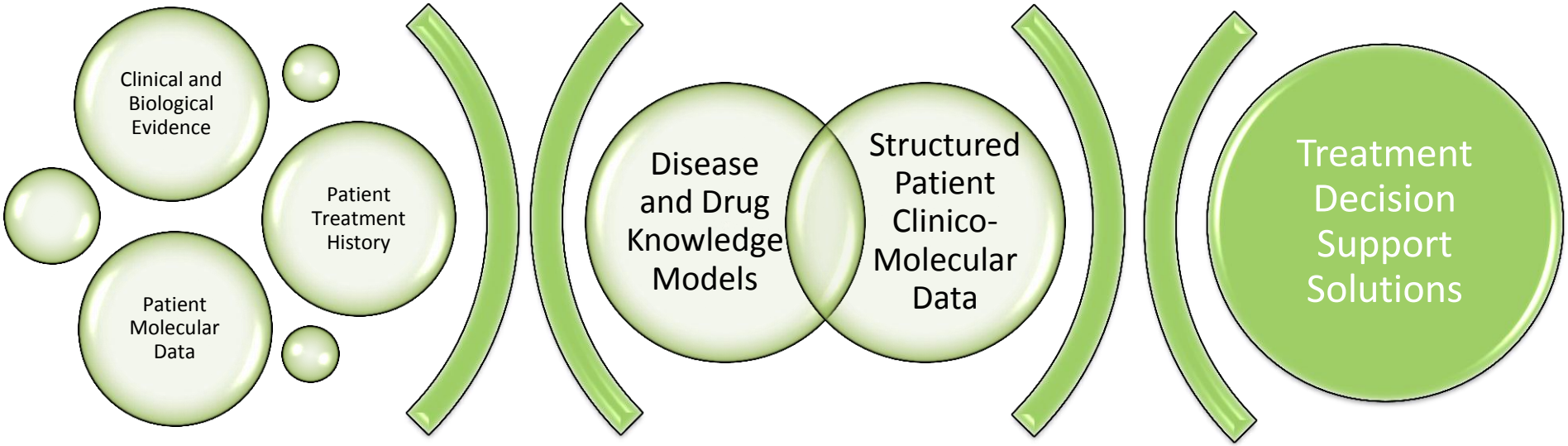
The Clinico-Molecular Paradigm



Disaggregate Data

Integration and Interpretation

Translation and Accessibility

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Genomic, Pharmacology and Cohort Evaluations Can Create a Complete Picture

Summary of Treatment Options | **Advanced Data Exploration**

Cancer Genome

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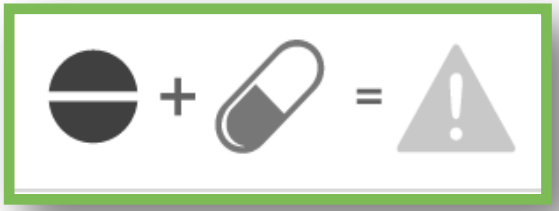
Clinical Pharmacology



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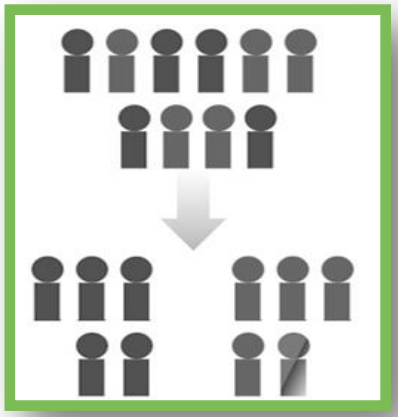
Cohort Study



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Technologies Are Emerging to Inform Treating Oncologist and Pathologist Decisions

Automatic Generation of Technical, Clinical Lab Report with Editing and Sign-out Functionality for Molecular Pathologist

Dr. X.J. Zhang, MD, Ph.D.

Cases

MOLECULARONCOLOGY

Log Out

SP00001, F, 39
Carcinoma, Non-Small-Cell Lung

Summary | Model | Variants | Details

Key findings

Print

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Revert

Whole exome sequence analysis was performed on a biopsy of NON-SMALL CELL LUNG CANCER from the right hilar lobe.

Sequence analysis revealed that **no clinically endorsed biomarkers** were present in the patients tumor. Nevertheless, **four clinically observed response biomarkers** were identified. Three independent predictors of **Gefitinib** responsiveness were detected (*EGFR*/L858R data; *KRAS*/G12F data; *BRAF*/V471F data), and the drug is approved for use in this indication. The *BRAF*/V471F mutation has also been observed to confer resistance to Gefitinib, but caution is urged as this effect was observed in pre-clinical models (data). Two independent predictors of **Erlotinib** responsiveness were also detected (*EGFR*/L858R data; *DDR2*/S768R data) and the drug is approved for use in this indication. A *DDR2*/S768R mutation has also been shown to predict responsiveness to Dasatinib, which is currently under Phase 2 investigation in this indication. In addition, the *KRAS*/G12F mutation has also been associated with responsiveness to Everolimus (data), which is currently under Phase 2 investigation in this indication.

Pharmacological analysis of the patient's current medications (Clarithromycin; Ambien; Aspirin; Crestor; Diovan; Hydrochlorothiazide; Metoprolol) also revealed potential for drug:drug interactions. Co-medication of Clarithromycin with Gefitinib or Erlotinib may increase the levels and toxicity of these anti-cancer agents. Consider appropriate modifications to anti-infective regimen if Gefitinib and/or Erlotinib are chosen for follow-on treatment.

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