A framework to evaluate the economic impact of pharmacogenomics

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Introduction: Pharmacogenomics and personalized medicine promise to improve health care by increasing drug efficacy and minimizing side effects. There may also be substantial savings realized by eliminating costs associated with failed treatment. This paper describes a framework using health claims data for analyzing the potential value of pharmacogenomic testing in clinical practice. Methods: We evaluated a model of alternate clinical strategies using asthma patients’ data from a retrospective health claims database to determine a potential cost offset. We estimated the likely cost impact of using a hypothetical pharmacogenomic test to determine a preferred initial therapy. We compared the annualized per patient costs distributions under two clinical strategies: testing all patients for a nonresponse genotype prior to treating and testing none. Results: In the Test All strategy, more patients fall into lower cost ranges of the distribution. In our base case (15% phenotype prevalence, US$200 test, 74% overall first-line treatment efficacy and 60% second-line therapy efficacy) the cost savings per patient for a typical run of the testing strategy simulation ranged from US$200 to US$767 (5th and 95th percentile). Genetic variant prevalence, test cost and the cost of choosing the wrong treatment are key parameters in the economic viability of pharmacogenomics in clinical practice. Conclusions: A general tool for predicting the impact of pharmacogenomic-based diagnostic tests on healthcare costs in asthma patients suggests that upfront testing costs are likely offset by avoided nonresponse costs. We suggest that similar analyses for decision making could be undertaken using claims data in which a population can be stratified by response to a drug.

Pharmacogenomics – the use of genomic markers to predict health status and drug response – promises safer and more effective drug treatment, biomarkers to guide drug discovery at its earliest stages, and a context for prioritizing future advances in medical care [1–4]. However, pharmacogenomics presents many challenges to our healthcare, drug development, clinical practice, regulatory and social systems [5,6].

One major challenge of pharmacogenomics is its translation from discovery to clinical practice [7,6]. Economic incentives for the pharmaceutical industry to develop diagnostics for clinical applications of pharmacogenomics are mixed, and the technology development is proceeding more slowly than proponents expected [8–10]. Clinical uptake of pharmacogenomics technology awaits evidence supporting its clinical utility. At the same time, further evidence supporting the exploration of its clinical utility through clinical trials or post-marketing studies on pharmacogenomic-correlated outcomes is needed [11–14].

A clinical area pharmacogenomics is expected to impact strongly is using genetic determinants of drug response to drive treatment choice [8,10,15,16]. Several published studies evaluating pharmacogenomics in clinical practice have measured the cost-effectiveness of screening patient populations using available pharmacogenomic-based test/treatment combinations. In some, but not all cases, the benefit of pharmacogenomic screening outweighs the cost. The results depend on the nature of the indication (monogenic or complex, common or rare), the test cost, treatment cost, treatment benefits with and without the test, and the prevalence of the pharmacogenomic variant [14,17–21].

This suggests that the economic viability of pharmacogenomic-based diagnostic screening will depend on specific circumstances of its use. Indeed, classic studies concluded that screening in high-risk populations [22,23]. Several groups have developed frameworks to help identify which specific circumstances will most likely benefit from pharmacogenomics-related clinical strategies [6,9,14,15,24,25]. Generally speaking, testing before treating is economically viable if the savings gained by avoiding ineffective treatment and adverse events are greater than the costs of testing. A method for using data already available to predict the circumstances of economic viability

before investing resources into pharmacogenomic marker discovery and diagnostic test development would be valuable to those involved in deciding how to allocate discovery, development and technology translation resources. Here we present an approach to evaluating the potential economic value of pharmacogenomics in clinical practice using data from retrospective claims databases. The health claims data are rich in information and outcomes that have not been previously utilized for a pharmacogenomics inquiry.

We hypothesize that the costs of treatment nonresponse reflect less desirable health outcomes, such as continued or exacerbated symptoms and adverse events. Were it possible to determine who would not respond to a given therapy through a diagnostic test, the costs of nonresponse could be reduced or eliminated, creating a potential cost offset. We expect the costs of nonresponse to be reflected in overall costs, so that overall costs, available from retrospective claims databases, can be used to gauge the potential economic viability of a diagnostic test.

We used retrospective health claims data and a stochastic model to predict the likely cost impact of using a hypothetical diagnostic test to aid in initial treatment choice. We illustrate our approach using characteristics of a population of patients with asthma. As we specified neither the genetic marker tested nor the drugs used in treatment, we anticipate that our framework is general. Any clinical indication where costs can be measured and patients can be grouped according to differential response rates using characteristics reported in health claims data could be analyzed similarly to the asthma case presented here. This method can highlight important parameters in the economic viability of future pharmacogenomic-based diagnostic tests. We suggest that this model may be of benefit in guiding decisions for pharmacogenomics development in indications for which there is health claims data available.

Methods & data

Approach

We modeled the use of a hypothetical genetic test predicting treatment efficacy under two alternative, general clinical scenarios:

- Test no one (No Test), offering the first-line treatment choice (Rx1) to all newly diagnosed patients
- Test all patients (Test All) for a genetic marker indicating lowered response to Rx1, offering a second-line therapy (Rx2) to those who test positive for the marker (Figure 1)

Model simulations

A cohort of 10,000 asthma patients was simulated for each clinical strategy by randomly drawing the value of each model parameter from its estimated distribution, calculating treatment response and annual cost under each scenario, and repeating these calculations 10,000 times. We treated test costs as constants. We compared the simulated cost distributions (No Test–Test All). Break-even analyses were conducted to assess the sensitivity of cost savings to a range of values for test cost and genotype prevalence.

Model parameters

We derived values for the model’s parameters, listed in Table 1, using data from published literature [14,18–20].

Marker characteristics

In simulation of both strategies, a prevalence parameter determined whether each patient had the genetic variant. The 15% prevalence for the base case is the same as the prevalence of a genetic variant of the \( \beta_2 \)-adrenergic receptor associated with adverse response to regular albuterol treatment [26,27].

Test characteristics

We assumed a 1% rate of false positives from the test. We varied the test cost from US$0–1600 and chose US$200 for the base case, based on reported test costs for similar DNA sequence tests [18–20].

Treatment characteristics

The response rate for Rx1 in the base case was 80% in the genotype-negative population and 40% for the population with the marker. An 80% response rate is high, but close to that found for inhaled beclamethasone in a clinical trial in which approximately 75% of trial participants receiving beclomethasone experienced improved lung function as measured by forced expiratory volume [28,29]. A 50% reduction in response for those with the hypothetical marker was a speculative assumption.

Cost data

Our cost data were derived from Medstat’s MarketScan® Commercial Claims and Encounters Databases for 1995–2000, comprised of longitudinal patient healthcare records based on insurance claims for medical services and prescription drugs covered by employer-sponsored health plans. In our analysis, total healthcare costs are actual payments to providers for all health services, including hospitalizations, outpatient...
services and filled prescriptions, for asthma patients aged 4–64 years. We followed patients’ resource utilization and costs for 24 months after their index date (the date on which the inclusion criteria were met):

- At least two outpatient claims with primary or secondary asthma diagnoses; or
- At least one emergency room claim with a primary asthma diagnosis and a drug transaction for an asthma drug 90 days prior or 7 days following emergency room claim; or
- At least one inpatient claim with primary asthma diagnosis; or
- A secondary asthma diagnosis and a primary respiratory infection diagnosis in the same claim

Patients were excluded if they had:

- Outpatient prescription drug coverage and continuous health plan enrollment for less than 12 months prior to or 24 months following their index date; or
- Chronic obstructive pulmonary disease (COPD) diagnosis; or
- Diagnosis or procedure codes indicating pregnancy or delivery

The population of asthma patients in the health claims database was subdivided into responders and nonresponders using a binary measure of medication complexity as a proxy for treatment response over the study period. Asthma patients were characterized as responders unless their claims exhibited the following criteria for high level of medical complexity, in which case they were characterized as nonresponders:

- At least four asthma medication dispensing events; or
- At least one emergency room visit with asthma as a primary diagnosis; or
- At least one inpatient visit with asthma as a primary diagnosis; or

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**Figure 1. Clinical strategies simulated.**

We simulated cost distributions for the two different clinical strategies shown. The first-line treatment, Rx1, is given to all patients in the No Test Strategy. The alternative treatment, Rx2, is given to patients who test positive for a marker predictive of reduced response to Rx1. In our stochastic model, each patient’s response to treatment is determined and a cost, extracted from the retrospective health claims database sample, is applied. Taking the costs for the whole population gives the cost distribution for the simulation.

PGx: Pharmacogenomics; Rx1: First-line treatment; Rx2: Second-line therapy.
At least four outpatient visits with asthma listed as one of the diagnoses and at least two asthma medication dispensing events. We identified 28,324 asthmatics in the sample. The mean costs for all asthmatics over the study period were US$7610, or an annualized rate of US$3805. We classified 66.7% as responders and 33.3% nonresponders with mean (± standard deviation) annualized costs of US$5132 (± 9188) and US$3140 (± 8272) per patient, respectively. These means are consistent with those from other recent studies of asthma costs using patient claims data. For example, Birnbaum and colleagues found that annual per-capita employer expenditures for asthmatic patients were 2.5 times higher than patients without asthma claims (US$5385 vs US$2121, respectively) [30]. Empirical cost distributions rather than estimated moments were used as model inputs.

For populating the healthcare costs distributions, patients were randomly allocated a responder cost or a nonresponder cost, depending on their response status, derived from the retrospective healthcare claims database described above. In the Test All strategy, the test cost was included in every patient’s cost, and the incremental cost of Rx2 versus Rx1 affected the costs for positive-test patients (given Rx2). In the No Test strategy, the costs were unmodified.

### Results

A population diagnosed with a certain indication for which a first-line pharmaceutical treatment is regularly prescribed can be divided into those for whom the treatment is quite effective and those for whom the treatment is less effective. We developed a model for evaluating the potential cost savings using a pharmacogenomic test to distinguish those groups using retrospective claims data when clinical data on outcomes from the pharmacogenomic strategy are not available. Based on model parameters, we used stochastic simulations to subdivide 10,000-patient populations into those responding and those not responding to therapy. We then applied cost data derived from Medstat’s MarketScan Commercial Claims and Encounters Databases for 1995–2000 to arrive at cost distributions for the alternate clinical strategies.

In the framework for the model (Figure 1), we envision a genetic marker for reduced response to the preferred first-line treatment for the indication (Rx1). In the Test All strategy, the two genotypes are assumed to yield different proportions of positive and negative diagnostic test results. A positive test result in our model indicates reduced Rx1 efficacy, and the patient is treated with a second-line therapy, Rx2. Response to Rx1 is conditional, in part, on the nonresponse genotype, while response to Rx2 is independent of it. A negative test result does not predict Rx1 response, but all patients with negative results are treated with Rx1. In the No Test strategy, all patients are treated with Rx1, and treatment response is determined by the patient’s simulated genotype and the Rx1 efficacy parameters. Nonresponders in the No Test strategy

### Table 1. Model parameters: parameters used in the model shown with their base case values and the range of the simulation.

<table>
<thead>
<tr>
<th></th>
<th>Base case</th>
<th>Range or points simulated</th>
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<tbody>
<tr>
<td><strong>Genomic data</strong></td>
<td></td>
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<tr>
<td>Prevalence of genetic nonresponse to Rx1 (phenotype)</td>
<td>15%</td>
<td>1–50%</td>
</tr>
<tr>
<td><strong>Proportion responding to therapy</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rx1 (patients without genotype)</td>
<td>80%</td>
<td>40–80%</td>
</tr>
<tr>
<td>Rx2 (all patients)</td>
<td>60%</td>
<td>50–80%</td>
</tr>
<tr>
<td>Rx1 (patients with genotype)</td>
<td>40%</td>
<td>From 0.25 to 0.75 the value of Rx1 for the responding patients</td>
</tr>
<tr>
<td><strong>Proportion testing positive for Rx1 nonresponsive gene, given:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Genotype absent (false positives)</td>
<td>1%</td>
<td>1–15%</td>
</tr>
<tr>
<td>Genotype present (true positives)</td>
<td>99%</td>
<td></td>
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<tr>
<td><strong>Costs</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Testing</td>
<td>US$200</td>
<td>US$0–1600</td>
</tr>
<tr>
<td>Asthma care, responders (sample mean)</td>
<td>US$3141</td>
<td></td>
</tr>
<tr>
<td>Asthma care, nonresponders (sample mean)</td>
<td>US$5133</td>
<td></td>
</tr>
<tr>
<td>Incremental costs of Rx2 vs Rx1 (per year)</td>
<td>US$0</td>
<td>US$0, 100, 1000 and 10,000</td>
</tr>
</tbody>
</table>
either have the genetic variant (reduced-response genotype) or do not respond to Rx1 for a different, undetermined reason.

We found that a Test All strategy can be cost-neutral or slightly cost-effective in the asthma population studies as it improves overall health outcomes with more patients responding to initial therapy. The genetic variant prevalence, test cost, and the cost of making an incorrect treatment choice in terms of additional disease burden and/or adverse events are the most important variables for determining whether testing before treating is a cost-effective clinical practice. Our base case represents 15% prevalence of the Rx1 nonresponse genotype, a US$200 test, 74% overall Rx1 efficacy and 60% average Rx2 efficacy. In a typical base case simulation, the expected value for treatment response is 77% for the Test All strategy compared with 68.2% for the No Test strategy (Table 2). The expected value for first-year per patient healthcare costs is slightly lower for the Test All strategy (US$3918 vs US$3921 for the No Test strategy). In the simulated first-year costs distribution, the range of cost savings (No Test cost–Test All cost) was US$200 (5th percentile) to US$767 (95th percentile) in a typical simulation run. The Test All strategy’s cost distribution is shifted slightly lower, as more patients fall into lower cost ranges (Figure 2).

Our results indicate that the prevalence of the genetic variant is a key parameter in the economic viability of the Test All strategy (Figure 3a). When the prevalence is lower, fewer patients in the population would have their responder status affected by the outcome of a diagnostic test. At lower prevalence, then, nonresponder costs are less significant to overall costs, the relative cost and relative effectiveness of the two therapies are more important to the economic outcome for the population, and testing a broad population is economically viable in more limited circumstances.

We investigated the base case using a set of drug response rate and drug incremental cost values to simulate two treatment conditions:

- That Rx2 is the second-line therapy because it is less effective (Rx2 efficacy 60%, incremental cost between Rx2 and Rx1 US$0 per year)
- That Rx2 is costly but equally effective (Rx2 efficacy 80%, incremental cost US$1000 per year)

From the results of these simulations, we could conclude that the potential cost savings is less sensitive to increased test cost under the condition where Rx1 and Rx2 cost the same (Figure 3b).

**Discussion**

Our analysis of health claims data suggests that avoiding the burden of ineffective treatment is likely to offset the costs of screening the population of primary care patients with asthma-like symptoms for treatment response. We chose asthma as a test case because it is highly prevalent and constitutes an important public health concern. While researchers have found genetic markers related to treatment response heterogeneity in asthma patients, current asthma practice guidelines include no pretreatment diagnostic test for response to aid initial treatment decisions [31–33]. Still, when the clinical practice guidelines for asthma are achieved, quality of life improves [34]. Our results, then, are nonobvious and draw attention to the potential value of pharmacogenomic-based diagnostics used broadly in clinical practice. Moreover, our results suggest that health claims data can be a valuable source of information used for pharmacogenomics modeling.

The results from our model are most strongly dependent on the genotype prevalence and the test cost. This corresponds with factors influencing potential cost effectiveness of pharmacogenomics identified by others, lending some validation to our approach [6,9,14,15,24,25]. However, a true validation of our model will emerge from its use by other researchers in other indications, and from any prospective studies encouraged by our results.

Finding the most effective therapy earlier in a disease course becomes more valuable when continuing symptoms or drug side effects are severe. A clinical trial has confirmed the anecdotal finding that β-receptor genotype affects β-agonist treatment response in mild asthmatics. The surprising result from this trial is that the β-agonist appears to worsen symptoms for those with the reduced-response genotype, although their mild asthma is still manageable without costly interventions such as emergency room visits [27]. So, in this case, patients with the nonresponse phenotype who are given a β-agonist for their mild asthma will experience some exacerbation of their symptoms in addition to not responding to their prescribed treatment, increasing the value of testing first.

There are limitations to simulating cost outcomes in general and there are limitations to our model as presented here. Ours is a simulation and not a formal cost-effectiveness or cost–benefit analysis [14,15,17]. Our evaluation can determine whether a pharmacogenomics clinical strategy would likely be favored economically and can distinguish small from large impacts on healthcare.
The results from the simulation in asthma reveal that the absolute numbers could not be used reliably, for example, to establish a diagnostic test price.

In this first illustration of our model we made some assumptions that limit our analysis, and we anticipate that our model will be refined and improved with ensuing use and follow-on analyses. It is unlikely that patients would remain on an ineffective medication for a year, as is a consequence of our model assumptions. However, we did not model a crossover treatment strategy that would take changing medication into account in this first assessment of our method. Our analysis does not take adverse events into account explicitly, but assumes that costs associated with adverse events are rolled into the overall costs. We inferred nonresponse from medical resource consumption as documented by health insurance claims, rather than from explicit medical assessments. Hence, it is possible that patients who complied rigorously with complex medication regimes and/or frequent follow-up assessments may have been misclassified as nonresponders, while others who suffered from uncontrolled asthma symptoms but avoided seeking care may have been misclassified as responders. In addition, annual costs were estimated from 24-month follow-up data. To the extent that costs for individual patients vary from year to year, regression to the mean may have diminished the variation that would be expected if only one year of cost was observed. How these data limitations might have affected estimates of annual cost conditional on response or nonresponse is uncertain.

Adjusting treatment based on a genotype has been recommended in some genotype–phenotype association studies related to drug response [27,35,36]. We designed a method using health claims data to examine when it may be justifiable to use a diagnostic test in treatment selection, and we tested it in the context of asthma. The method described here can provide a quantitative estimate of the potential economic viability of a clinical strategy that includes pharmacogenomics. The results can be used for:

- Determining healthcare service coverage and reimbursement policies
- Planning for and forecasting future healthcare costs
- Determining regulatory policy for pharmacogenomic-based diagnostic products
- Developing clinical practice guidelines
- Developing diagnostic testing products for commercialization

This prospective analysis of parameters influencing the economic viability of potential diagnostic tests could provide important information for all stakeholder groups developing policies for incorporating pharmacogenomic information into healthcare.

### Table 2. Results for the base case.

<table>
<thead>
<tr>
<th></th>
<th>Calculated expected values</th>
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</thead>
<tbody>
<tr>
<td><strong>Response to therapy</strong></td>
<td></td>
</tr>
<tr>
<td>No Test</td>
<td>67.5%</td>
</tr>
<tr>
<td>Test All</td>
<td>79.5%</td>
</tr>
<tr>
<td><strong>Annualized per patient cost</strong></td>
<td></td>
</tr>
<tr>
<td>No Test</td>
<td>US$3921</td>
</tr>
<tr>
<td>Test All</td>
<td>US$3918</td>
</tr>
<tr>
<td><strong>Average savings (No Test–Test All)</strong></td>
<td>US$3</td>
</tr>
</tbody>
</table>

Range in distribution of 10,000 random patients

<table>
<thead>
<tr>
<th>Range of cost savings</th>
<th>5th percentile</th>
<th>95th percentile</th>
</tr>
</thead>
<tbody>
<tr>
<td>-US$200</td>
<td>US$767</td>
<td></td>
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</table>

15% phenotype prevalence, US$200 test, 74 and 60% overall efficacy for Rx1 and Rx2, respectively, no incremental cost difference between Rx1 and Rx2. For first year response rates and first year cost, values shown are expected values calculated from the base case values of the simulation parameters. The range of cost savings is illustrated by the 5th and 95th percentile values taken from a typical simulation run where 10,000 random annual treatment costs were generated for each clinical scenario and compared (No Test–Test All).

Rx1: First-line treatment; Rx2: Second-line therapy.

**Outlook**

Well beyond our asthma example, a scientific revolution is underway. Information being accumulated from sequencing the genome and elucidating the proteome is being translated into knowledge about individual characteristics that...
are determinants of disease and response to therapy. Translating new technologies for assessing individual drug response into clinical practice awaits evidence of their clinical utility [11,15]. Economic models for evaluating the economic viability of emerging diagnostic tests to predict drug response are imperative if there are to be the systemic changes in healthcare required for clinical adoption of pharmacogenomics. A case in point is the US FDA-approved AmpliChip® CYP450, a microarray-based test from Roche Diagnostics (Basel, Switzerland) [37]. This pharmacogenomic-based diagnostic test determines the polymorphism fingerprint of an individual’s two major drug-metabolizing enzymes, and is intended for use in pre-prescription clinical testing. Despite being approved by the US FDA, several payers have called for clinical and economic outcomes studies prior to its widespread utilization and reimbursement.

A clinical strategy to select an initial therapy on the basis of a diagnostic test linked to a biomarker is increasingly viable, occasioned by the increasing availability of targeted therapies. Clearly, the use of such tests, though elegant scientifically, comes with a cost. We are now in a period of transition into genomics-based clinical decision-making paradigms that will allow greater individualization of therapies. During this transition, therapy selection will need to proceed via a mix of old and new approaches, and cost considerations will critically influence how personalized medicine is incorporated into common medical practice. What is needed as we go forward are methods to analyze under what circumstances such an alternative strategy makes sense, especially in economic terms.

Conflict of interest statement
Dr Ginsburg had access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the analysis. There are no conflicts of interest to report.

This work was supported by grants from Millennium Pharmaceuticals, Inc. (MA, USA) and the Alfred P Sloan Foundation (NY, USA) through the MIT Program (MA, USA) on the Pharmaceutical Industry. Authors from Millennium Pharmaceuticals, Inc. were involved in the study design. Sponsors had no role in the data collection, interpretation or analysis.
Figure 3a. The role of test cost and phenotype prevalence on economic viability of pharmacogenomic testing.

Break-even points illustrate the model parameter values where the two clinical strategies are cost neutral. Shown are the break-even points for the parameters of test cost and genetic variant prevalence under two different treatment characteristic conditions – one where the alternative treatment (Rx2) is less effective but no more expensive than the first-line treatment (Rx1), and one where Rx2 is more costly but equally effective. For a specific test cost in each condition, the Test All strategy is economically viable in our model when the genetic prevalence has any value above that condition’s line. For example, when Rx2 is equally effective but significantly more costly (the purple line), there is no reasonable value of genotype prevalence that would allow the Test All strategy to be cost neutral or cost effective once the test cost exceeds US$300. Therefore, development of a diagnostic that is inexpensive could be prioritized over one requiring more costly technology.

Highlights

- Clinical adoption of pharmacogenomics-based treatment strategies requires robust models for evaluating the economic viability of emerging diagnostic tests to predict drug response.
- We developed a model for using retrospective health claims data to predict the likely cost impact of using a hypothetical pharmacogenomic test to determine a preferred initial therapy. Our stochastic model uses health claims data to stratify a population by response and determine costs distributions for two different speculative clinical strategies.
- We tested our model first on a population of primary care patients with asthma-like symptoms. We compared two speculative clinical strategies: 1) test all patients first to determine a preferred initial therapy, or 2) test none and treat all with the same first-line treatment.
- Our analysis suggests upfront testing costs are likely offset by avoiding nonresponse costs in asthma. The prevalence of the genetic variant is a key parameter for economic viability of a test-first clinical strategy.
- We designed the model to be a generalizable tool for predicting the healthcare costs impact of pharmacogenomic-based diagnostic testing using claims data. Analyses of similar data sets could be expanded to other indications for guiding decisions around pharmacogenomic test development.
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Figure 3b. Break-even analysis for the Test All and No Test clinical strategies.

Cost differences calculated using base case values for all parameters except test cost and genetic variant prevalence are presented to highlight their effects on the predicted savings from a pharmacogenomic clinical strategy. Predicted cost savings (No Test–Test All) are shown on the vertical (Y) axis. Test cost values are shown on the X axis. Savings results from using prevalence values of 1 and 50% outline the range of the effect prevalence has on savings. If the genotype prevalence is only 1%, there is No Test cost that would result in cost savings from a pharmacogenomic strategy in our model.

Rx1: First-line treatment; Rx2: Second-line therapy.

Bibliography
Papers of special note have been highlighted as either of interest (*) or of considerable interest (**) to readers.

This paper reviews cost-effectiveness analysis in valuing pharmacogenomics and cogently lays out a framework for its use to aid decision-making.


A clear and thorough review of different economics evaluation techniques and their potential application in pharmacogenomics.


References 19 and 20 represent prime examples of using cost-effectiveness analysis to assess pharmacogenomic-based clinical strategies for indications where a test has already been developed.


This prospective clinical trial of a genotype-stratified population, suggested by a retrospective analysis of clinical trials data, is a standard for the data needed to fully assess the impact of pharmacogenomics on outcomes.


37. AmpliChip™ CYP450, Test product detail. Roche, Basel, Switzerland.