Introduction

This slide presentation contains forward-looking statements which are subject to change based on various important factors, including without limitation, competitive actions in the marketplace and adverse actions of governmental and other third-party payors.

Actual results could differ materially from those suggested by these forward-looking statements. Further information on potential factors that could affect the Company’s financial results is included in the Company’s Form 10-K for the year ended December 31, 2007, and subsequent SEC filings.
The US Healthcare & Clinical Laboratory Testing Market

2007 Projected US Healthcare Spend $2.3 Trillion

- Total market size—$50 billion
- Industry CAGR of 5%-7%
- Market Segments:
  - Routine—$30-$35 billion
  - Esoteric—$4-$5 billion
  - Anatomic pathology—$6-$10 billion

Source: CMS, Office of the Actuary, G-2, and Company Estimates
Revenue Growth Drivers

Industry Forces
• Focus on Outcomes and Cost Containment (Medical & Drug)
• Increased emphasis on drug efficacy, proper dosage and adverse effects
• Advances in science and genomics

- Expansion of Managed Care partnerships
- Aging Population
  - Increased utilization for older patients
- Industry Consolidation
- Hospital Opportunity
- More Esoteric Testing
  - Cardiovascular Disease
  - Cancer
- Outcome Management Program
  - Litholink Model
- Companion Diagnostics
  - ARCA
  - Warfarin

LabCorp Assets
• Standardized Data
• Clinical Trials
• Dianon, USLabs, Esoterix, NGI & Viromed
Strategic Focus Areas

Scientific Leadership
- Cancer diagnostics and monitoring
- Advanced cardiovascular disease testing
- Advancement through acquisitions and licensing

Managed Care
- Lab data enables better treatment and outcomes
- Partner to control high cost leakage
- Recognize value of lab services through appropriate pricing

Customer Focus
- Quality and service driven culture
- First-time problem resolution
- Continuous enhancements in customer connectivity
The Enemy

- Homicide 17,732
- Accidents 109,277
  - Auto Accidents (45,000)
- Suicide 31,484
- Chronic lower-respiratory disease 126,382
- Diabetes 74,219
- Stroke 157,689
- Heart Disease 685,089
- Cancer 556,902
- Diseases 2.3M
- All other deaths 8,364
The Value of Lab Testing

Sources of Growth in Projected Federal Spending on Medicare and Medicaid (Percentage of GDP)

We have to slow this growth

Effect of Cost Growth Faster Than GDP and Aging of Population

Effect of Aging of Population

Source: Congressional Budget Office, November 2007
The Healthcare Conundrum

- Healthcare cost the United States 2.3 trillion dollars in 2007
- Lab tests cost $50 Billion
- Imaging is about the same.
- 90% of the medical decisions are made from information derived at a small % of the cost.
- We bring the most value!
DNA is the Blueprint
RNA is the Contractor
Ribosomes are the Workmen
Proteins make up the house
Individual genetic variation effects drug response

Pharmacokinetics – what the body does to the drug
Pharmacodynamics – what the drug does to the body

All patients with same diagnosis

Standard therapy
Responders and Patients
Not Predisposed to Toxicity

Alternate therapy
non-responders
and toxic responders

Personalized medicine:
Pharmacogenetics
Biomarker studies
- markers of disease state or drug effect

<table>
<thead>
<tr>
<th>Biomarker Discovery</th>
<th>Biomarker Verification</th>
<th>Biomarker Validation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of Analytes</td>
<td>Number of Samples</td>
<td></td>
</tr>
<tr>
<td>1,000</td>
<td>100</td>
<td>10</td>
</tr>
<tr>
<td>10</td>
<td>100</td>
<td>1,000</td>
</tr>
</tbody>
</table>

Failure rate of biomarker candidates expected to be similar to failure rate of drug candidates
Genome Wide Association Studies (GWAS)

Unbiased genome wide approach using 1000’s of individuals across very high density SNP chip arrays

Illumina
370k
550k/650kY
1 million ~95%

Affymetrix
100k
500k
1 million ~93%
The SNP Chip

Green = Homozygous G or C
Red = Homozygous A or T
Yellow = Heterozygous
2007: The year of GWAS

A Common Allele at 9p21 Affects Myocardial Infarction

The NEW ENGLAND JOURNAL of MEDICINE

Established in 1812 AUGUST 30, 2007 VOL. 357 NO. 9

Risk Alleles for Multiple Sclerosis Identified by a Genomewide Study

A Whole-Genome Association Study Identifies New Susceptibility Loci for Crohn Disease and Implicates Autophagy in Disease Pathogenesis

Genome-wide association study identifies new susceptibility loci for Crohn disease and implicates autophagy in disease pathogenesis

Risk Alleles for Multiple Sclerosis Identified by a Genomewide Study

The International Multiple Sclerosis Genetics Consortium

Genome-wide association study identifies new susceptibility loci for Crohn disease and implicates autophagy in disease pathogenesis

Genome-wide association study identifies new susceptibility loci for Crohn disease and implicates autophagy in disease pathogenesis

Discovery of tag SNPs identifies colorectal cancer at 8q24.21
Proteomics

Black Swallowtail – larvae and butterfly same DNA

Same DNA but very different proteome
- One cannot understand the biology without understanding the proteome
US molecular diagnostic testing market

Pharmacogenetic tests aren’t expected to see aggressive revenue growth until around 2010.
<table>
<thead>
<tr>
<th>Partner</th>
<th>Clinical Area</th>
</tr>
</thead>
<tbody>
<tr>
<td>ARCA Discovery</td>
<td>Companion Diagnostics (CVD) (exclusive)</td>
</tr>
<tr>
<td>Celera Diagnostics</td>
<td>Breast Cancer</td>
</tr>
<tr>
<td>Duke University</td>
<td>Lung Cancer (exclusive)</td>
</tr>
<tr>
<td>Exact Sciences</td>
<td>Colon Cancer</td>
</tr>
<tr>
<td>Intema Ltd.</td>
<td>Prenatal Testing</td>
</tr>
<tr>
<td>Ipsogen</td>
<td>Molecular Diagnostics</td>
</tr>
<tr>
<td>Medco Health Solutions</td>
<td>Companion Diagnostics (Research)</td>
</tr>
<tr>
<td>OMS</td>
<td>Companion Diagnostics (Oncology) (exclusive)</td>
</tr>
<tr>
<td>Siemens Health Solutions</td>
<td>Companion Diagnostics (Oncology and CVD)</td>
</tr>
<tr>
<td>SmartGene</td>
<td>Bioinformatics Tools</td>
</tr>
<tr>
<td>Third Wave Technologies</td>
<td>Companion Diagnostics (CVD)</td>
</tr>
<tr>
<td>Vanda Pharmaceuticals</td>
<td>Companion Diagnostics (Oncology) (exclusive)</td>
</tr>
<tr>
<td>Veridex</td>
<td>Prostate Cancer</td>
</tr>
<tr>
<td>Yale University</td>
<td>Ovarian Cancer (exclusive)</td>
</tr>
</tbody>
</table>
Congestive Heart Failure

Bucindololol and New Thinking

Pathophysiologial Definition:
A condition in which the heart is no longer able to pump an adequate supply of blood to meet the metabolic needs of tissues.

Clinical Definition:
A condition in which ventricular dysfunction causes reduced exercise capacity.
BEST trial, all-cause mortality full model (covariate adjusted, transplant censored)

HR = 0.87 (0.76, 1.00)
841 Ev, p = 0.053
SYNERGISTIC POLYMORPHISMS OF $\beta_1$- AND $\alpha_{2C}$-ADRENERGIC RECEPTORS AND THE RISK OF CONGESTIVE HEART FAILURE

Kersten M. Small, Ph.D., Lynne E. Wagoner, M.D., Albert M. Levin, M.P.H., Sharon L.R. Kardia, Ph.D., and Stephen B. Liggett, M.D.
## Adrenergic receptor $\beta_1$ 389 Arg/Gly and $\alpha_{2c}$ Wt/Del genotype combinations

### Gene Variants

<table>
<thead>
<tr>
<th>$\beta_1$ 389 Arg/Arg + $\alpha_{2c}$ 322-325 Del or Wt</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\beta_1$ 389 Gly carrier + $\alpha_{2c}$ 322-325 Wt/Wt</td>
</tr>
<tr>
<td>$\beta_1$ 389 Gly carrier + $\alpha_{2c}$ 322-325 Del carrier</td>
</tr>
</tbody>
</table>

### Bucindolol Rx interaction

- **$\beta_1$ 389 Arg/Arg + $\alpha_{2c}$ 322-325 Del or Wt**
  - (47% of BEST, 51% U.S.)
  - **Much higher efficacy in $\beta_1$ Arg/Arg overcomes $\alpha_{2c}$ Del adverse effects**
- **$\beta_1$ 389 Gly carrier + $\alpha_{2c}$ 322-325 Wt/Wt**
  - (40% of BEST, 39% of U.S.)
  - Efficacy from mild NE lowering adds to some efficacy in $\beta_1$ 389 Gly
- **$\beta_1$ 389 Gly carrier + $\alpha_{2c}$ 322-325 Del carrier**
  - (13% of BEST, 10% of U.S.)
  - Adverse effects of $\alpha_{2c}$ Del neutralizes low efficacy of $\beta_1$ 389 Gly

### Net Effects

- **“Very Favorable genotype”**
  - (HF EP effect sizes 34-48%)
- **“Favorable genotype”**
  - (HF EP effect sizes 19-40%)
- **“Unfavorable genotype”**
  - (No efficacy)
**All-cause Mortality by $\beta_1$ 389/$\alpha_{2c}$ 322-325 genotypes**

**Adjusted Analysis**

**Very Favorable**
($\beta_1$ 389 Arg/Arg + $\alpha_{2c}$ Wt/Wt or Del carrier)

- **Placebo**: HR = 0.62 (0.39, 0.99)
- **Bucindolol**: HR = 1.04 (0.43, 2.54)

**Favorable**
($\beta_1$ 389 Gly carrier + $\alpha_{2c}$ Wt/Wt)

- **Placebo**: HR = 0.75 (0.48, 1.17)
- **Bucindolol**: HR = 1.04 (0.43, 2.54)

**Unfavorable**
($\beta_1$ 389 Gly carrier + $\alpha_{2c}$ Del carrier)

- **Placebo**: HR = 0.75 (0.48, 1.17)
- **Bucindolol**: HR = 1.04 (0.43, 2.54)

**No. At Risk**

<table>
<thead>
<tr>
<th></th>
<th>Placebo</th>
<th>Bucindolol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>236</td>
<td>257</td>
</tr>
<tr>
<td>Bucindolol</td>
<td>229</td>
<td>250</td>
</tr>
</tbody>
</table>

**2 Months After Randomization**

- **Placebo**: 236 229 190 140 115 82 49
- **Bucindolol**: 257 250 224 181 144 106 61

**4 Months After Randomization**

- **Placebo**: 214 205 183 145 109 71 29
- **Bucindolol**: 199 193 163 136 108 68 41

**8 Months After Randomization**

- **Placebo**: 12 3
- **Bucindolol**: 15 6

**24 Ev, p = 0.042**

**85 Ev, p = 0.21**

**24 Ev, p = 0.93**

**HR = 0.62 (0.39, 0.99)**

**HR = 0.75 (0.48, 1.17)**

**HR = 1.04 (0.43, 2.54)**
Stroke Warfarin - Safety

- Over-anticoagulation associated with bleeding
- Bleeding events most likely within the first 90 days of therapy
- One-third of INR values exceed target range in first month of therapy
- 7% of patients suffer a major hemorrhage
- Relative risk of fatal extracranial bleeds 4.8%
- Rate of major bleed within six months range 5.6% to 12%
- Near top in most surveys of adverse events
- Average cost per patient of a bleeding episode $15,988 with a mean hospital stay of 6 days

Warfarin sodium can cause major or fatal bleeding. Bleeding is more likely to occur during the starting period and with a higher dose (resulting in a higher INR). Risk factors for bleeding include high intensity of anticoagulation (INR >4.0), age ≥65, highly variable INRs, history of gastrointestinal bleeding, hypertension, cerebrovascular disease, serious heart disease, anemia, malignancy, trauma, renal insufficiency, concomitant drugs (see PRECAUTIONS), and long duration of warfarin therapy. Regular monitoring of INR should be performed on all treated patients. Those at high risk of bleeding may benefit from more frequent INR monitoring, careful dose adjustment to desired INR, and a shorter duration of therapy. Patients should be instructed about prevention measures to minimize risk of bleeding and to report immediately to physicians signs and symptoms of bleeding (see PRECAUTIONS: Information for Patients).
Warfarin: Optimal Dose

The graph shows the relationship between the International Normalized Ratio (INR) and the odds ratio of ischemic stroke and intracranial bleeding. The x-axis represents the INR, ranging from 1.0 to 8.0. The y-axis represents the odds ratio, ranging from 0 to 20. The solid line represents ischemic stroke, and the dotted line represents intracranial bleeding. The data points indicate that as the INR increases, the odds ratio for ischemic stroke decreases, and the odds ratio for intracranial bleeding increases.
Genetic Factors and Warfarin Dosing

- 2 genes → 3 SNPs → Reduced Activity
- Two genes play key role in the response to warfarin
- Variants significantly impact the rate of warfarin metabolism and amount of drug target available
- Pharmacokinetics – CYP2C9
- Pharmacodynamics – VKORC1

PHARMACOGENOMICS

CYP2C9 gene variants, drug dose, and bleeding risk in warfarin-treated patients: A HuGEnet™ systematic review and meta-analysis

VKORC1 and CYP2C9 genes are known to be involved in the response to warfarin treatment. Variants in these genes significantly impact the rate of warfarin metabolism and the amount of drug target available.

Pharmacokinetics – CYP2C9

Pharmacodynamics – VKORC1

A Genetic Component to Coumarin Sensitivity

In addition to this variation in dose-response within individuals, the individual dose range required to achieve adequate anticoagulation may vary widely.

Combined genetic profiles of components and protein-dependent 9-carboxylation system affect individual response to warfarin.

Summary

HEMOSTASIS, THROMBOSIS, AND VASCULAR BIOLOGY

Cytochrome P450 2C9 (CYP2C9) and vitamin K epoxide reductase (VKORC1) genotypes as determinants of acenocoumarol sensitivity

Pharmacokinetics

- CYP2C9 metabolizes S-warfarin and terminates the drug activity
- Genetic variations in CYP2C9 alter S-warfarin clearance
- CYP2C9*2 and CYP2C9*3 alleles significantly reduce S-warfarin metabolism

- **2C9 = Sets the rate**
- **VKORC1 = Sets the amount**

- Significantly associated with lower maintenance doses
- Significantly associated with increased time to stable dose

Li et al., 2004 Nature vol 427:541-544; Rost et al., 2004 Nature Vol 427:537-541; Reider et al., 2005 NEJM 352:2285-93; Takahashi & Echizen 2003 Pharmacogenomics J 3:202-211.
In a large collaborative study 31,567 asymptomatic people were screened for lung cancer using low dose CT.

- 821 suspicious lesions were detected.
- 412 turned out to be stage 1 lung cancer.
- 409 turned out to be benign.
- It takes very dangerous biopsy or PET scan to tell the difference.
The Duke Lung Cancer markers are serum proteins which differentiate between benign lesions and true cancers with a simple serum based test.
Fig 1. Classification and Regression Tree analysis of the training set selected four proteins with seven terminal nodes. The three terminal cancer nodes have a bold outline. CEA, carcinoembryonic antigen; RBP, retinol binding protein; SCC, squamous cell carcinoma antigen; AAT, α1-antitrypsin.
5-year survival rates

70-80% among the

25-30% of patients
diagnosed with stage I
or II

20-30% survival
among the >70% of
patients diagnosed
with stage III or IV

VALIDATION

**Microarray**

**Leptin**

**Prolactin**

**OPN**

**IGF-II**

---

**ELISA**

**Leptin**

**Prolactin**

**Osteopontin**

**Insulin-like Growth Factor-II**
What About Prevention?

- "The time to repair the roof is when the sun is shining."
  -- John F. Kennedy

- Difficult job because you need to influence individual behavior.
Measuring The Balance of DNA Damage and Repair

In order to find out which way to go you have to know where you are.

8-Hydroxy-2’-deoxyguanosine

Antioxidants

Comet Assay

- Oxidative Damage
- DNA Repair
- DNA
- Protective Enzymes

mRNA Levels for Repair Enzymes
ReiCa, 8OH2'dG profile

Merry Christmas, Happy New Year!
They were at the same parties.
DNA damage may be calculated using different measurements:

- **Tail Length**: Distance from center of comet head to end of tail
- **Tail Extent Moment**: \( \text{Tail length} \times \% \text{tail fluorescence} \)
- **Olive Tail Moment**: \( \text{Avg distance of DNA migration} \times \% \text{tail fluorescence} \)
Color Enhanced Comet Assay Photo
We can tell you your Real DNA Age

60 Year Old

40 Year Old

20 Year Old
Titrating H$_2$O$_2$ to induce DNA damage

<table>
<thead>
<tr>
<th></th>
<th>Tail Extent Moment</th>
<th>Olive Tail Moment</th>
<th>Tail Length</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Negative Control</strong></td>
<td>8.4</td>
<td>2.2</td>
<td>28.7</td>
</tr>
<tr>
<td><strong>200 μM H$_2$O$_2$</strong></td>
<td>51.8</td>
<td>17.7</td>
<td>75.7</td>
</tr>
<tr>
<td><strong>600 μM H$_2$O$_2$</strong></td>
<td>58.6</td>
<td>21.4</td>
<td>79.8</td>
</tr>
<tr>
<td><strong>1000 μM H$_2$O$_2$</strong></td>
<td>64.8</td>
<td>23.8</td>
<td>85.2</td>
</tr>
</tbody>
</table>

Jurkat E6-1 cell line
DNA Repair Capacity Analysis Assay

1. Patient Blood Sample (white blood cells)

2. Split into 3 equal cell populations

3. Induce DNA damage (H₂O₂)
   - Positive control - assayed immediately after damage induction
   - Negative control - no damage induced - assayed directly

4. Test Sample - incubate cells in culture medium @ 37°C for ≥ 1 hr. to allow DNA damage repair

Perform Comet Assay on all cell populations to quantify damage
DNA Repair Enzymes

OGG1 8-oxoG DNA Glycosylase
MTH1 MutT Homologue-1
NEIL1 nei endonuclease VIII-like 1 protein
ERCC1 Excision Repair Cross-Complementing gene
MYH MutY Homologue
HOX 1 Heme Oxygenase 1
NTH1 Nth Homolog 1
APE 1 AP Endonuclease 1

Antioxidant Enzyme
SOD-1 Super Oxide Dismutase

Housekeeping Gene
UBC Ubiquitin C

- Housekeeping gene is quantified alongside enzymes via multiplex PCR
- Enzyme concentrations are reported as a ratio relative to UBC
- Resulting ratios are compared between test samples and controls to indicate degree of up-regulation, if any
Rapid, short exposure to $\text{H}_2\text{O}_2$ followed by 6 hour recovery incubation induces some enzyme up-regulation.
Cancer is linked to changes in the genome in a more direct way than the other major diseases.

This gives an opportunity to try something really special.

We could sequence the cancer genome.
Tumor Biopsy & Blood Sample Provided

Laboratory

Bioinformatics

Interpretation Relative To Published Literature

Personalized Web Based Genome Browser
2001
- First human genome assembled (85% complete) for about $200 million, huge teams, and years of work from about 24 billion bases of raw sequence.

2006
- 454 introduces massively parallel sequencer: 40 million bases of raw sequence in 1 day for $10,000: Declare sequencing of Jim Watson (~4-5x coverage though) for about $1 million. Genome sequence would cost about $6 million at 20x and require 1500 days of machine time.

2007
- Illumina introduces ‘sequencing by synthesis’ generates about 1 billion based of raw sequence in 4 days for $4000. Genome sequence possible at $240,000 in about 240 days of machine time.

2008
- ABI Solid introduces sequencing by ligation generates about 4 billion bases of raw sequence in 5 days for $7000. Sequences Yoruban individual at 10x coverage for $60,000 in reagent costs ($120,000 to generate sufficient sequence for complete genome) and requires 75 days of machine time.

2008
- Release of Helicos will probably not change pricing much.

2009-2011
ABI Solid Sequencer can generate 5 billion bases every 5 days, and is advancing rapidly.

12 runs of a single machine (60 days) generates sufficient sequence to Cover the whole genome.
We have forded relationships with:

- Duke
- Harvard/MIT
- UCLA

We will do this in the next two years!
Revenue CAGR of 8.5% – Diluted EPS CAGR of 18.6%

1. Excluding the $0.09 per diluted share impact in 2005 of restructuring and other special charges, and a non-recurring investment loss.
2. Excluding the $0.06 per diluted share impact in 2006 of restructuring and other special charges.
3. Excluding the $0.25 per diluted share impact in 2007 of restructuring and other special charges.
Five-Year OCF and EBITDA Margin Trend

OCF CAGR of 6% – EBITDA Margin Growth of 210 bps

1. Includes approximately $50 million of benefit from one-time tax credits recorded in 2003.
2. Excluding the impact in 2005 of restructuring and other special charges and a non-recurring investment loss.
3. Excluding the impact in 2006 and 2007 of restructuring and other special charges.
4. As a result of adopting FASB 123(R) in 2006, the Company recorded incremental stock compensation expense of $23.3 and $26.7 in 2006 and 2007, respectively.
# Second Quarter Results

(In millions, except per share data)

<table>
<thead>
<tr>
<th></th>
<th>6/30/2007</th>
<th>6/30/2008</th>
<th>+/(-)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Revenue</td>
<td>$1,043.1</td>
<td>$1,147.8</td>
<td>10.0%</td>
</tr>
<tr>
<td>EBITDA (^{(1)})</td>
<td>$279.6</td>
<td>$301.1</td>
<td>7.7%</td>
</tr>
<tr>
<td>EBITDA Margin</td>
<td>26.8%</td>
<td>26.2%</td>
<td>(60) bp</td>
</tr>
<tr>
<td>Diluted EPS (^{(2)})</td>
<td>$1.09</td>
<td>$1.24</td>
<td>13.8%</td>
</tr>
</tbody>
</table>

\(^{(1)}\) Excludes restructuring and other special charges of $4.1 and $61.0 million recorded by the Company in the second quarter of 2007 and 2008, respectively.  

\(^{(2)}\) Excludes the $0.04 and $0.32 per diluted share impact of the restructuring and other special charges recorded in the second quarter of 2007 and 2008, respectively.
### YTD Second Quarter Results
*(In millions, except per share data)*

<table>
<thead>
<tr>
<th></th>
<th>6/30/2007</th>
<th>6/30/2008</th>
<th>+/-(-)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Revenue</td>
<td>$2,041.8</td>
<td>$2,251.0</td>
<td>10.2%</td>
</tr>
<tr>
<td>EBITDA&lt;sup&gt;(1)&lt;/sup&gt;</td>
<td>$540.1</td>
<td>$586.6</td>
<td>8.6%</td>
</tr>
<tr>
<td>EBITDA Margin</td>
<td>26.5%</td>
<td>26.1%</td>
<td>(40) bp</td>
</tr>
<tr>
<td>Diluted EPS&lt;sup&gt;(2)&lt;/sup&gt;</td>
<td>$2.06</td>
<td>$2.38</td>
<td>15.5%</td>
</tr>
</tbody>
</table>

<sup>(1)</sup> Excludes restructuring and other special charges of $4.1 and $61.0 million recorded by the Company through the second quarter of 2007 and 2008, respectively.

<sup>(2)</sup> Excludes the $0.03 and $0.32 per diluted share impact of the restructuring and other special charges recorded through the second quarter of 2007 and 2008, respectively.
2008 Second Quarter Financial Achievements

- **Diluted EPS of $1.24**\(^{(1)}\)
- **EBITDA margin of 26.2% of net sales**\(^{(2)}\)
- **Operating cash flow of $194.7 million**
- **Increased revenues**
  - 10.0% (9.0% volume; 1.0% price)
  - Excl. Canada 3.6% (1.3% volume, 2.3% price)
- **Repurchased approximately $10.8 million of LabCorp stock**

---

(1) Excludes the $0.32 per diluted share impact of the restructuring and other special charges recorded in the second quarter of 2008.

(2) Excludes the restructuring and other special charges of $61 million recorded by the company in the second quarter of 2008.
2008 YTD Second Quarter
Financial Achievements

- Diluted EPS of $2.38 \(^{(1)}\)
- EBITDA margin of 26.1% of net sales\(^{(2)}\)
- Operating cash flow of $371.2 million
- Increased revenues
  - 10.2% (8.8% volume; 1.4% price)
  - Excl. Canada 3.8% (1.4% volume, 2.4% price)
- Repurchased approximately $66.5 million of LabCorp stock

\(^{(1)}\) Excludes the $0.32 per diluted share impact of the restructuring and other special charges recorded through the second quarter of 2008.

\(^{(2)}\) Excludes the restructuring and other special charges of $61 million recorded by the company through the second quarter of 2008.
Revenue by Payer - US
YTD Q2 2008
(In millions)

- Patient: $190.8 (9%)
- Managed Care Capitated: $88.1 (4%)
- Medicare & Medicaid: $403.6 (19%)
- Managed Care Fee-for-service: $851.5 (40%)
- Client: $585.9 (28%)
Revenue by Business Area - US
YTD Q2 2008
(In millions)

- Core: $1391.1 (66%)
- Histology (Non-Pap): $161.7 (8%)
- Other Esoteric: $245.9 (11%)
- Genomic: $321.2 (15%)
1) EBITDA represents earnings before interest, income taxes, depreciation and amortization, and includes the Company’s proportional share of the underlying EBITDA of the income from joint venture partnerships. The Company uses EBITDA extensively as an internal management performance measure and believes it is a useful, and commonly used measure of financial performance in addition to earnings before taxes and other profitability measurements under generally accepted accounting principles (“GAAP”). EBITDA is not a measure of financial performance under GAAP. It should not be considered as an alternative to earnings before income taxes (or any other performance measure under GAAP) as a measure of performance or to cash flows from operating, investing or financing activities as an indicator of cash flows or as a measure of liquidity. The following table reconciles earnings before income taxes, representing the most comparable measure under GAAP, to EBITDA for the three-month period ended March 31, 2008 and 2007:

<table>
<thead>
<tr>
<th></th>
<th>Three Months</th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Ended March 31,</td>
<td>2008</td>
<td>2007</td>
</tr>
<tr>
<td>Earnings before income taxes</td>
<td></td>
<td>$221.9</td>
<td>$208.9</td>
</tr>
<tr>
<td>Add (subtract):</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interest expense</td>
<td></td>
<td>19.9</td>
<td>12.6</td>
</tr>
<tr>
<td>Investment income</td>
<td></td>
<td>(0.5)</td>
<td>(2.1)</td>
</tr>
<tr>
<td>Other (income) expense, net</td>
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<td>0.6</td>
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