UPDATE ON OUR GENERAL MEDICINE PORTFOLIO

NOVEMBER 7, 2022
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No forward-looking statement can be guaranteed and actual results may differ materially from those we project. Our results may be affected by our ability to successfully market both new and existing products domestically and internationally, clinical and regulatory developments involving current and future products, sales growth of recently launched products, competition from other products including biosimilars, difficulties or delays in manufacturing our products and global economic conditions. In addition, sales of our products are affected by pricing pressure, political and public scrutiny and reimbursement policies imposed by third-party payers, including governments, private insurers and managed care providers and may be affected by regulatory, clinical and guideline developments and domestic and international trends toward managed care and healthcare cost containment. Furthermore, our research, testing, pricing, marketing and other operations are subject to extensive regulation by domestic and foreign government regulatory authorities. We or others could identify safety, side effects or manufacturing problems with our products, including our devices, after they are on the market. Our business may be impacted by government investigations, litigation and product liability claims. In addition, our business may be impacted by the adoption of new tax legislation or exposure to additional tax liabilities. If we fail to meet the compliance obligations in the corporate integrity agreement between us and the U.S. government, we could become subject to significant sanctions. Further, while we routinely obtain patents for our products and technology, the protection offered by our patents and patent applications may be challenged, invalidated or circumvented by our competitors, or we may fail to prevail in present and future intellectual property litigation. We perform a substantial amount of our commercial manufacturing activities at a few key facilities, including in Puerto Rico, and also depend on third parties for a portion of our manufacturing activities, and limits on supply may constrain sales of certain of our current products and product candidate development. An outbreak of disease or similar public health threat, such as COVID-19, and the public and governmental effort to mitigate against the spread of such disease, could have a significant adverse effect on the supply of materials for our manufacturing activities, the distribution of our products, the commercialization of our product candidates and our clinical trial operations, and any such events may have a material adverse effect on our business, outcomes, progress, and other such estimates and results. Forward-looking statements involve significant risks and uncertainties, including those discussed below and more fully described in the Securities and Exchange Commission reports filed by Amgen, including our most recent annual report on Form 10-K and any subsequent periodic reports on Form 10-Q and current reports on Form 8-K. Unless otherwise noted, Amgen is providing this information as of the date of this presentation and does not undertake any obligation to update any forward-looking statements contained in this document as a result of new information, future events or otherwise.

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<table>
<thead>
<tr>
<th>Topic</th>
<th>Presenter</th>
</tr>
</thead>
</table>
| Introduction                                               | David Reese, MD  
Executive Vice President, Research and Development, Amgen                                                                     |
| FOURIER AND FOURIER-OLE (Open-Label Extension) Studies     | Marc S. Sabatine, MD, MPH  
Chair of TIMI Study Group  
Distinguished Chair in Cardiovascular Medicine, Brigham and  
Women’s Hospital  
Professor of Medicine, Harvard Medical School            |
| Reduction of Lipoprotein(a) With Small Interfering RNA     | Narimon Honarpour MD, PhD  
Vice President, Clinical Development, Amgen                                                                     |
| AMG 133 Program Update                                     |                                                                                                                                          |
| Concluding Remarks                                         |                                                                                                                                          |
| Q&A                                                        | All                                                                                                                                     |
TARGETING MECHANISMS OF RESIDUAL RISK THROUGH INNOVATIVE USE OF HUMAN DATA TO INFORM TARGET DISCOVERY, BIOMARKERS, AND PATIENT SEGMENTATION

High CV and Metabolic Risk Patients

Untreated Risk

- Lp(a)
- Olpasiran
- AMG 133
- OBESITY AND ITS CONSEQUENCES
- INFLAMMATION
- OTHER MECHANISMS

Lower LDL Cholesterol

Additional programs coming into the clinical pipeline for metabolic/obesity, heart failure and vascular disease

LDL= low-density lipoprotein; Lp(a)= Lipoprotein(a); CV= cardiovascular.

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FOURIER AND FOURIER-OLE (Open-Label Extension) Studies

Marc S. Sabatine, MD, MPH

This study was funded by Amgen Inc.
Background

• In the FOURIER trial, 27,564 patients with stable ASCVD were randomized to the PCSK9 inhibitor evolocumab vs. placebo.

• Evolocumab reduced the risk of MACE, but there was no observed effect on CV mortality.

• However, the median follow-up was only 2.2 years.

• Pivotal statin trials had median follow-up of 4-5 years and demonstrated both a lag effect (clinical benefit grew over time) and legacy effect (clinical benefit persisted in extended follow-up after the parent trial ended).

• Thus, very long-term data on safety and efficacy of LDL-C lowering with PCSK9 inhibition are needed.

ASCVD = atherosclerotic cardiovascular disease; PCSK9 = proprotein convertase subtilisin/kexin type 9; MACE = Major adverse cardiac events; CV = cardiovascular; LDL-C = low-density lipoprotein cholesterol.
Patients with stable ASCVD and LDL-C ≥70 mg/dl (~1.8mM) or non-HDL-C ≥100 mg/dl (~2.6mM) on optimized statin Rx

Evolocumab Q2 or Q4 wks

Matching placebo

Open-Label Evolocumab Q2 or Q4 wks

Parent FOURIER Trial
Median follow-up 2.2 years
N=27,564

FOURIER OLE Program
Median follow-up 5.0 years
N=6635

US & Eastern Europe: NCT02867813
Western Europe: NCT03080935
Effect on LDL-C

Parent FOURIER
Evolocumab vs placebo

FOURIER-OLE
Open-label Evolocumab

Placebo transition
to evolocumab

Median LDL cholesterol (95% CI, mmol/L)

Placebo  ➔  Evolocumab
Evolocumab  ➔  Evolocumab

Median LDL-C at Week 260:
0.75 mmol/L (IQR 0.44-1.29)
29 mg/dl (IQR 17-50)

LDL-C = low-density lipoprotein cholesterol; OLE = open label extension; IQR = interquartile range.

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Long-Term Safety

SERIOUS ADVERSE EVENTS
- Placebo Phase FOURIER: 13%
- Evolocumab Phase FOURIER: 13%
- Evolocumab Phase FOURIER & OLE: 10%

INJECTION SITE REACTIONS
- Placebo Phase FOURIER: 0.7%
- Evolocumab Phase FOURIER: 0.8%
- Evolocumab Phase FOURIER & OLE: 0.4%

DRUG-RELATED ALLERGIC REACTION
- Placebo Phase FOURIER: 1.1%
- Evolocumab Phase FOURIER: 1.1%
- Evolocumab Phase FOURIER & OLE: 0.6%

MUSCLE-RELATED EVENT
- Placebo Phase FOURIER: 1.9%
- Evolocumab Phase FOURIER: 2.1%
- Evolocumab Phase FOURIER & OLE: 1.2%

NEW ONSET DIABETES
- Placebo Phase FOURIER: 2.3%
- Evolocumab Phase FOURIER: 1.8%
- Evolocumab Phase FOURIER & OLE: 1.2%

HEMORRHAGIC STROKE
- Placebo Phase FOURIER: 0.05%
- Evolocumab Phase FOURIER: 0.00%
- Evolocumab Phase FOURIER & OLE: 0.04%

IR = Incidence rate; OLE = open label extension

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Efficacy during FOURIER-OLE

**Primary Endpoint:** CV death, MI, stroke, unstable angina or coronary revascularization

- **Placebo**
  - HR 0.85 (95% CI 0.75-0.96)
  - P=0.008
  - 15% reduction

- **Evolocumab**
  - HR 0.80 (95% CI 0.68-0.93)
  - P=0.003
  - 20% reduction

**Key Secondary Endpoint:** CV death, MI or stroke

- **Placebo**
  - HR 0.80 (95% CI 0.68-0.93)
  - P=0.003
  - 20% reduction

- **Evolocumab**
  - HR 0.77 (95% CI 0.60-0.99)
  - P=0.04
  - 23% reduction

**CV death**

- **Placebo**
  - 3.32%

- **Evolocumab**
  - 4.45%

CV = cardiovascular; MI = myocardial infarction; OLE = open label extension; HR = hazard ratio.
Analyses On Very Low LDL-C

• The optimal achieved LDL-C level with regards to cardiovascular and safety outcomes in the long-term remains unclear.

• We explored the relationship between achieved LDL-C levels and the occurrence of long-term adverse cardiovascular and safety outcomes, down to very low (<20 mg/dL) achieved LDL-C levels.

LDL-C = low-density lipoprotein cholesterol.
Primary endpoint: CV death, MI, stroke, coronary revascularization or hospitalization for unstable angina.

Key secondary endpoint: CV death, MI, or stroke.

Adj P-value <0.0001
Safety and Achieved LDL-C

An Academic Research Organization of Brigham and Women’s Hospital and Harvard Medical School

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Summary & Conclusions

• Long-term use of evolocumab with median follow-up of more than 7 years appears both safe and well-tolerated.

• Earlier initiation of evolocumab is associated with continued accrual of cardiovascular benefit, including cardiovascular mortality, over the next several years.

• Monotonic relationship between lower achieved LDL-C levels, down to very low LDL-C levels <20 mg/dL, and a lower risk of cardiovascular events up to 8.6 years of follow-up.

• “There appears to be no LDL-C level below which benefit ceases”.

(2022 ACC Expert Consensus Decision Pathway on the Role of Non-statin Therapies for LDL-Cholesterol Lowering)
Reduction of Lipoprotein(a) With Small Interfering RNA: Results of the OCEAN(a)-DOSE (TIMI 67) Trial

Marc S. Sabatine MD MPH: On Behalf of the OCEAN(a)-DOSE Trial Investigators

Clinicaltrials.gov: NCT04270760
The study was funded by Amgen
Lp(a) and Coronary Heart Disease Risk

Lp(a) is associated with atherosclerotic cardiovascular disease risk independent of traditional risk factors

Lp(a) distribution in individuals with established atherosclerotic cardiovascular disease

- Lp(a) ≥ 150 nmol/L
- 20.3%

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Lp(a) = Lipoprotein(a)
Patients aged 18-80 years with atherosclerotic disease & Lp(a) >150 nmol/L

N=281

RANDOMIZE 1:1:1:1:1

DOUBLE BLIND

Olpasiran 10mg Q12W
Olpasiran 75mg Q12W
Olpasiran 225mg Q12W
Olpasiran 225mg Q24W
Placebo

Primary Endpoint: % Change in Lp(a) from Baseline to Week 36
Key Secondary Endpoint: % Change in Lp(a) from Baseline to Week 48

Lp(a)= Lipoprotein(a); Q12W = every 12 weeks; Q24W = every 24 weeks.

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O'Donoghue ML et al., Am Heart J 2022;251:61-69
## Baseline Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Olpasiran Pooled Dose Arms (N=227)</th>
<th>Placebo Q12W SC (N=54)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age in years, mean (SD)</strong></td>
<td>61.6 (9.6)</td>
<td>63.4 (8.9)</td>
</tr>
<tr>
<td><strong>Female sex</strong></td>
<td>32%</td>
<td>33%</td>
</tr>
<tr>
<td><strong>Coronary Artery Disease</strong></td>
<td>91%</td>
<td>93%</td>
</tr>
<tr>
<td><strong>Myocardial Infarction</strong></td>
<td>27%</td>
<td>37%</td>
</tr>
<tr>
<td><strong>Peripheral Artery Disease</strong></td>
<td>12%</td>
<td>6%</td>
</tr>
<tr>
<td><strong>Cerebrovascular Disease</strong></td>
<td>20%</td>
<td>22%</td>
</tr>
<tr>
<td><strong>Selected lipid lowering therapy use at baseline</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Statin</td>
<td>89%</td>
<td>83%</td>
</tr>
<tr>
<td>Ezetimibe</td>
<td>55%</td>
<td>41%</td>
</tr>
<tr>
<td>PCSK9 inhibitor</td>
<td>24%</td>
<td>22%</td>
</tr>
<tr>
<td><strong>Baseline laboratory values, median (IQR)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lipoprotein(a), (nmol/L)</td>
<td>261 (197, 360)</td>
<td>246 (200, 343)</td>
</tr>
<tr>
<td>LDL-C, (mg/dl)</td>
<td>68 (52, 87)</td>
<td>65 (48, 81)</td>
</tr>
</tbody>
</table>

LDL-C = low-density lipoprotein cholesterol; SC = subcutaneous; Q12W = every 12 weeks.
Changes in Lp(a) Through Follow-Up

![Graph showing changes in Lp(a) over weeks for different treatments.]

- **Olpasiran 10mg Q12W**
- **Olpasiran 225mg Q24W**
- **Placebo**

Means reported as least-square means.

Lp(a) = Lipoprotein(a); Q12W = every 12 weeks; Q24W = every 24 weeks.

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Primary Endpoint:
% Change in Lp(a) at 36 weeks

Olpasiran Doses

Placebo-adjusted least square means % change in Lp(a)

-70.5%  -97.4%  -101.1%  -100.5%

10 mg Q12W  75 mg Q12W  225 mg Q12W  225 mg Q24W

Lp(a)= Lipoprotein(a); Q12W = every 12 weeks; Q24W = every 24 weeks.
Interindividual Variability in Lp(a) Response at Week 36

PLACEBO Q12W

10 mg Q12W

75 mg Q12W

225 mg Q12W

225 mg Q24W

% change from baseline Lp(a)

Lp(a) = Lipoprotein(a); Q12W = every 12 weeks; Q24W = every 24 weeks.
# Safety & Tolerability

<table>
<thead>
<tr>
<th></th>
<th>Olpasiran 10 mg Q12W SC (N=58)</th>
<th>Olpasiran 75 mg Q12W SC (N=58)</th>
<th>Olpasiran 225 mg Q12W SC (N=56)</th>
<th>Olpasiran 225 mg Q24W SC (N=55)</th>
<th>Placebo Q12W SC (N=54)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Treatment Emergent Adverse Events</strong></td>
<td>78%</td>
<td>79%</td>
<td>84%</td>
<td>85%</td>
<td>83%</td>
</tr>
<tr>
<td><strong>Serious adverse events</strong></td>
<td>5.2%</td>
<td>5.2%</td>
<td>11%</td>
<td>7.3%</td>
<td>15%</td>
</tr>
<tr>
<td><strong>Reported as related to study drug</strong></td>
<td>12%</td>
<td>22%</td>
<td>29%</td>
<td>25%</td>
<td>20%</td>
</tr>
<tr>
<td><strong>Leading to discontinuation of study drug</strong></td>
<td>1.7%</td>
<td>1.7%</td>
<td>1.8%</td>
<td>1.8%</td>
<td>1.9%</td>
</tr>
<tr>
<td><strong>Myalgia</strong></td>
<td>5.2%</td>
<td>1.7%</td>
<td>7.1%</td>
<td>7.3%</td>
<td>7.4%</td>
</tr>
<tr>
<td><strong>Liver-related adverse events</strong></td>
<td>1.7%</td>
<td>3.4%</td>
<td>1.8%</td>
<td>1.8%</td>
<td>3.7%</td>
</tr>
<tr>
<td><strong>Hyperglycemia, new-onset or worsening diabetes mellitus</strong></td>
<td>8.6%</td>
<td>5.2%</td>
<td>8.9%</td>
<td>5.5%</td>
<td>5.6%</td>
</tr>
<tr>
<td><strong>Injection site reactions</strong></td>
<td>5.2%</td>
<td>19%</td>
<td>21%</td>
<td>24%</td>
<td>11%</td>
</tr>
<tr>
<td><strong>Hypersensitivity reactions</strong></td>
<td>1.7%</td>
<td>6.9%</td>
<td>5.4%</td>
<td>9.1%</td>
<td>1.9%</td>
</tr>
</tbody>
</table>

*Injection site reactions were generally mild
**Hypersensitivity reactions were generally described as mild injection site pain

Q12W = every 12 weeks; Q24W = every 24 weeks.
Conclusions

• Lp(a) is associated with atherosclerotic cardiovascular disease risk independent of traditional risk factors

• Olpasiran, an siRNA, dosed 75 mg or higher every 12 weeks, reduces Lp(a) concentration by more than 95% in patients with established atherosclerotic cardiovascular disease.

• Olpasiran appears both safe and well-tolerated in this study.

• These findings set the foundation for Phase 3 testing scheduled to commence later this year (NCT05581303).

siRNA = small interfering ribonucleic acid; Lp(a) = Lipoprotein(a).

Small Interfering RNA to Reduce Lipoprotein(a) in Cardiovascular Disease

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Baris Gencer, M.D., J. Antonio G. López, M.D., Norman E. Lepor, M.D.,
Seth J. Baum, M.D., Elmer Stout, M.D., Daniel Gaudet, M.D., Ph.D.,
Beat Knusel, Ph.D., Julia F. Kuder, M.A., Xinhui Ran, M.S.,
Sabina A. Murphy, M.P.H., Huei Wang, Ph.D., You Wu, Ph.D.,
Helina Kassahun, M.D., and Marc S. Sabatine, M.D., M.P.H.,
for the OCEAN(a)-DOSE Trial Investigators*
PHASE 3 CARDIOVASCULAR OUTCOMES TRIAL SCHEMA

Primary endpoint: time to first CHD, MI or urgent revascularization

N~6000

- Established ASCVD (prior MI or high-risk prior PCI)
- Lp(a) ≥200 nmol/L

Olpasiran Q12W

Placebo Q12W

End of Study

Estimated study start in December 2022

ASCVD = atherosclerotic cardiovascular disease; PCI = percutaneous coronary intervention; Lp(a) = Lipoprotein(a); Q12W = every 12 weeks; CHD = coronary heart disease; MI = myocardial infarction.
AMG 133 PROGRAM UPDATE

NARIMON HONARPOUR
VICE PRESIDENT CLINICAL DEVELOPMENT
AMG 133, A FIRST-IN-CLASS ANTIBODY-PEPTIDE CONJUGATE TARGETING OBESITY

GIPR INHIBITORY ANTIBODY

AMG 133

GLP-1R activation

GLP-1R activation and GIPR inhibition

GLP-1 activation and GIPR inhibition

STUDY POPULATIONS OF GIPR VARIANTS

Japanese
Genome-wide association study\(^1\)

European
Genome-wide association study\(^2,3\)

Whole exome sequencing
in UK-Biobank and N. America\(^4\)

GIPR= Gastric Inhibitory Polypeptide Receptor; GLP-1= Glucagon-like peptide-1; GLP-1R= Glucagon-like peptide-1 receptor

\(^{1}\) Nature Genetics 2012; 44 (3):302-6

\(^{2}\) Nature Genetics 2010; 42 (11):937-48 (deCODE is collaborator)

\(^{3}\) Nature Genetics 2013; 45 (5):501-12 (deCODE is collaborator)

\(^{4}\) Science 2021; 373 (6550)
AMG 133 HAS DEMONSTRATED EARLY CLINICAL EFFICACY IN OBESE PATIENTS

Mean change from baseline in weight (kg)

Single Subcutaneous Dose of AMG133

- Placebo
- Low
- High

Day

kg= kilogram
Data source: Amgen internal data

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SINCE OUR UPDATE IN FEBRUARY, WE’VE CONTINUED TO PROGRESS AMG 133 IN PHASE 1

- Completed single ascending dose and multiple ascending dose cohorts
  - Dosed Q4W, subcutaneously.
- Dose-dependent reductions in mean body weight from baseline.
  - Multi-dose mean percent change in BW by day 85: -7.19% (low dose) to -14.52% (high dose) in AMG 133-treated vs 1.49% in placebo.
  - Total of three doses, each 4 weeks apart.
  - Plateau on weight reduction not observed.
- No notable safety concerns were identified; most TEAEs were mild and transient.
- The pharmacokinetics were dose-proportional with an extended half-life.
- Data from the initial cohorts will be presented at the 20th World Congress on Insulin Resistance, Diabetes and Cardiovascular Disease (WCIRDC) Hybrid Conference in Dec.

Planning underway to rapidly initiate a Phase 2 trial

Q4W= every 4 weeks; BW = body weight; TEAE = treatment emergent adverse event
Data source: Amgen internal data

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CONCLUSIONS
ROBUST CARDIOMETABOLIC PORTFOLIO PROVIDES POTENTIAL LONG-TERM GROWTH OPPORTUNITIES FOR THE COMPANY

- Portfolio strategy focuses on genetically validated drivers of risk such as PCSK9, Lp(a) and GIPR (for obesity); results are encouraging.
- Repatha FOURIER OLE findings argue for early initiation of a significant and sustained LDL-C reduction to maximize clinical benefit.
- Olpasiran treatment resulted in rapid and sustained reduction in Lp(a); plan to initiate Phase 3 in December 2022.
- AMG 133 demonstrated significant dose-dependent weight-loss of up to ~14.5% @ d85 in obese patients with Q4W dosing; plan to initiate Phase 2 early 2023.

PCSK9 = proprotein convertase subtilisin/kexin type 9; Lp(a) = Lipoprotein(a); GIPR = Gastric Inhibitory Polypeptide Receptor; OLE = open label extension; LDL-C = low-density lipoprotein cholesterol; Q4W = every 4 weeks

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QUESTIONS?

DAVID REESE, MD