



Pioneering science delivers vital medicines™

November 6, 2012

Amgen Investor Event 2012 AHA Scientific Sessions

Sean E. Harper, MD

Executive Vice President, Research and Development

Scott M. Wasserman, MD, FACC

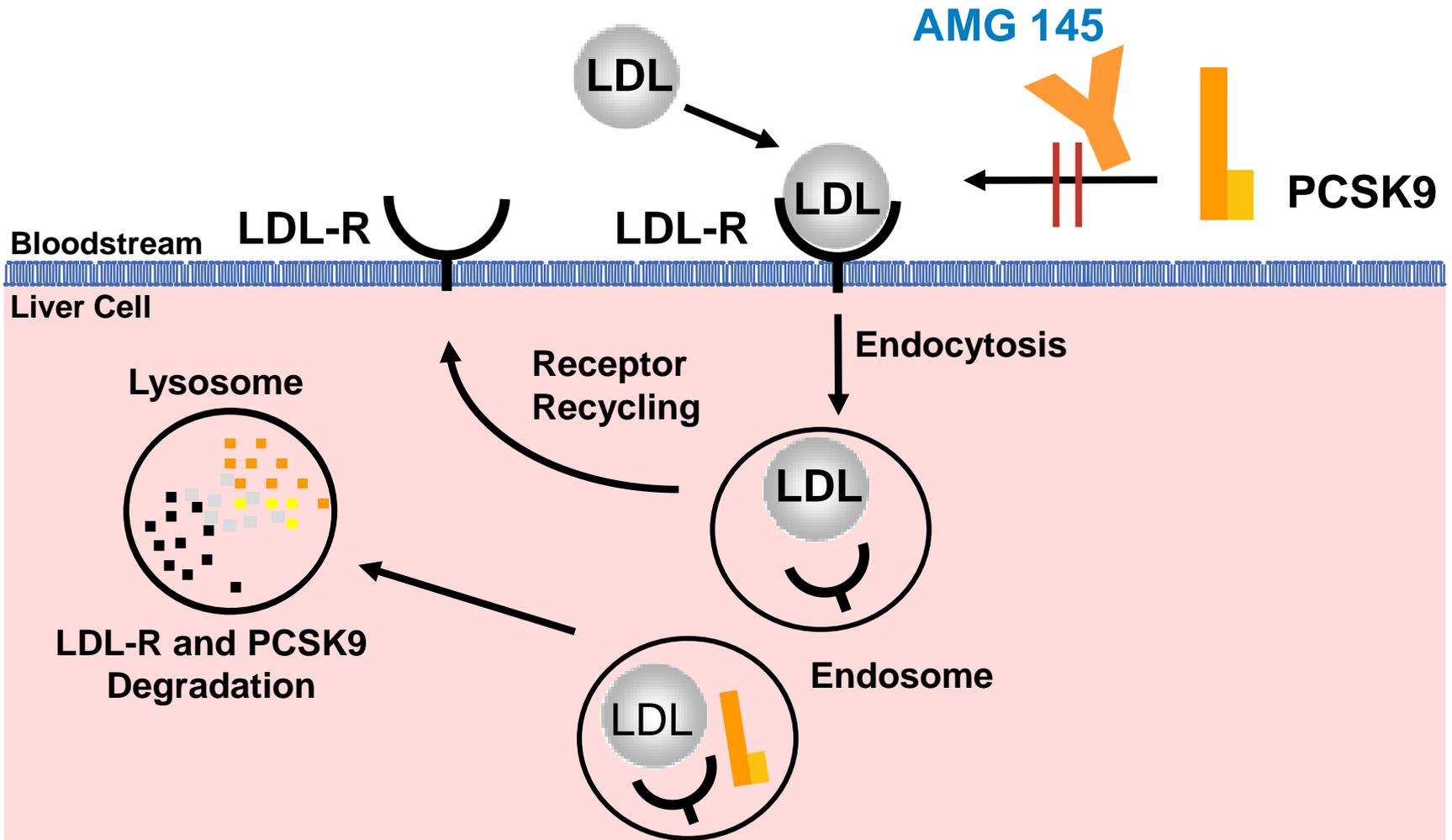
Executive Medical Director, Clinical Development

Safe Harbor Statement

This presentation contains forward-looking statements that are based on management's current expectations and beliefs and are subject to a number of risks, uncertainties and assumptions that could cause actual results to differ materially from those described. All statements, other than statements of historical fact, are statements that could be deemed forward-looking statements, including statements about estimates of revenues, operating margins, capital expenditures, cash, other financial metrics, expected legal, arbitration, political, regulatory or clinical results or practices, customer and prescriber patterns or practices, reimbursement activities and outcomes 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 (SEC) reports filed by Amgen, including Amgen's most recent annual report on Form 10-K and most recent periodic reports on Form 10-Q and Form 8-K. Please refer to Amgen's most recent Forms 10-K, 10-Q and 8-K for additional information on the uncertainties and risk factors related to our business. Unless otherwise noted, Amgen is providing this information as of November 6, 2012 and expressly disclaims any duty to update information contained in this presentation.

No forward-looking statement can be guaranteed and actual results may differ materially from those we project. The Company's results may be affected by our ability to successfully market both new and existing products domestically and internationally, clinical and regulatory developments (domestic or foreign) involving current and future products, sales growth of recently launched products, competition from other products (domestic or foreign) and difficulties or delays in manufacturing our products. In addition, sales of our products are affected by reimbursement policies imposed by third-party payers, including governments, private insurance plans 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 as well as U.S. legislation affecting pharmaceutical pricing and reimbursement. Government and others' regulations and reimbursement policies may affect the development, usage and pricing of our products. 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 after they are on the market. Our business may be impacted by government investigations, litigation and products liability claims. 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. We depend on third parties for a significant portion of our manufacturing capacity for the supply of certain of our current and future products and limits on supply may constrain sales of certain of our current products and product candidate development. In addition, we compete with other companies with respect to some of our marketed products as well as for the discovery and development of new products. Discovery or identification of new product candidates cannot be guaranteed and movement from concept to product is uncertain; consequently, there can be no guarantee that any particular product candidate will be successful and become a commercial product. Further, some raw materials, medical devices and component parts for our products are supplied by sole third-party suppliers. Our business performance could affect or limit the ability of our Board of Directors to declare a dividend or our ability to pay a dividend or repurchase our common stock.

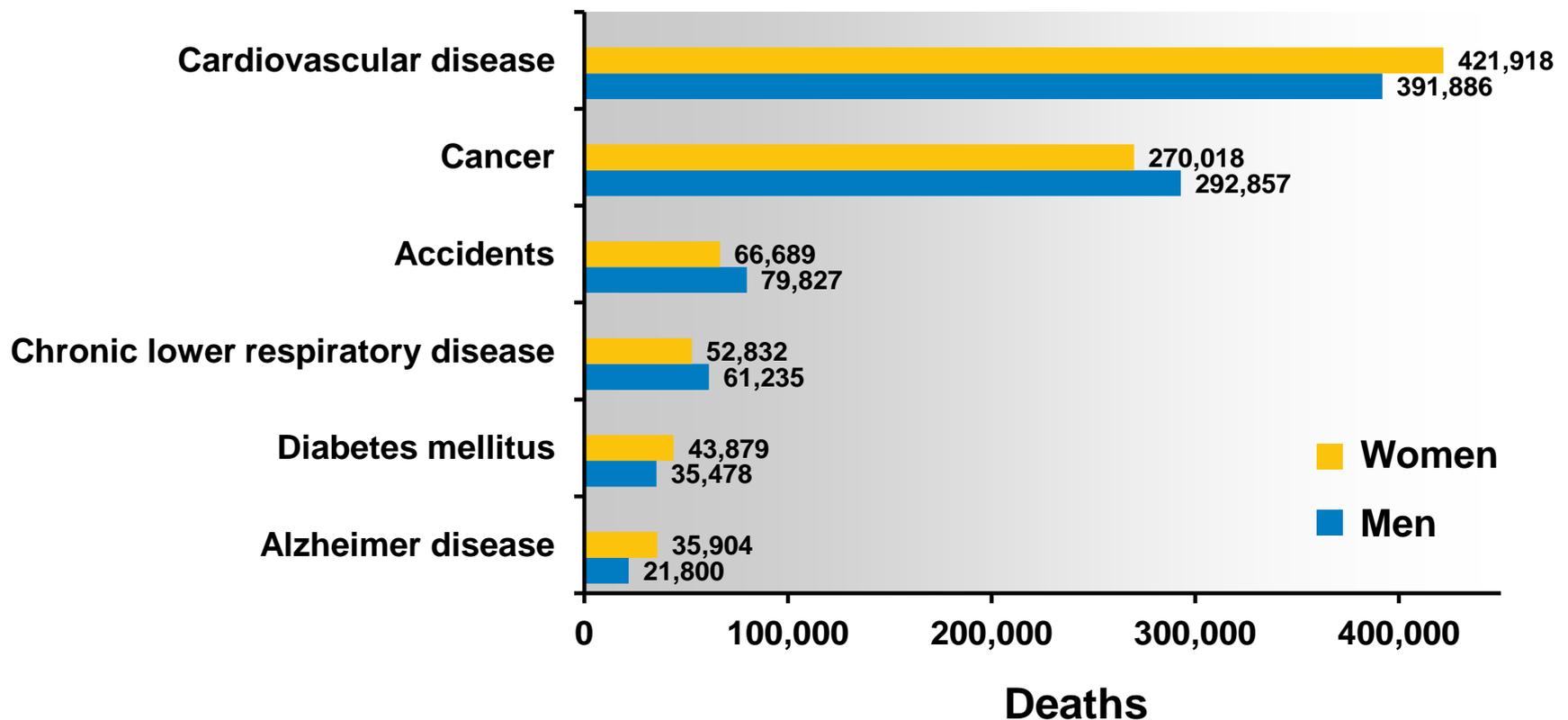
AMG 145 Inhibits PCSK9 and Increases LDL Receptor Expression



LDL = low-density lipoprotein; LDL-R = LDL receptor
Provided November 6, 2012 as part of an oral presentation and is qualified by such, contains forward-looking statements, actual results may vary materially; Amgen disclaims any duty to update.

Cardiovascular Disease Is the Leading Cause of Death in the US

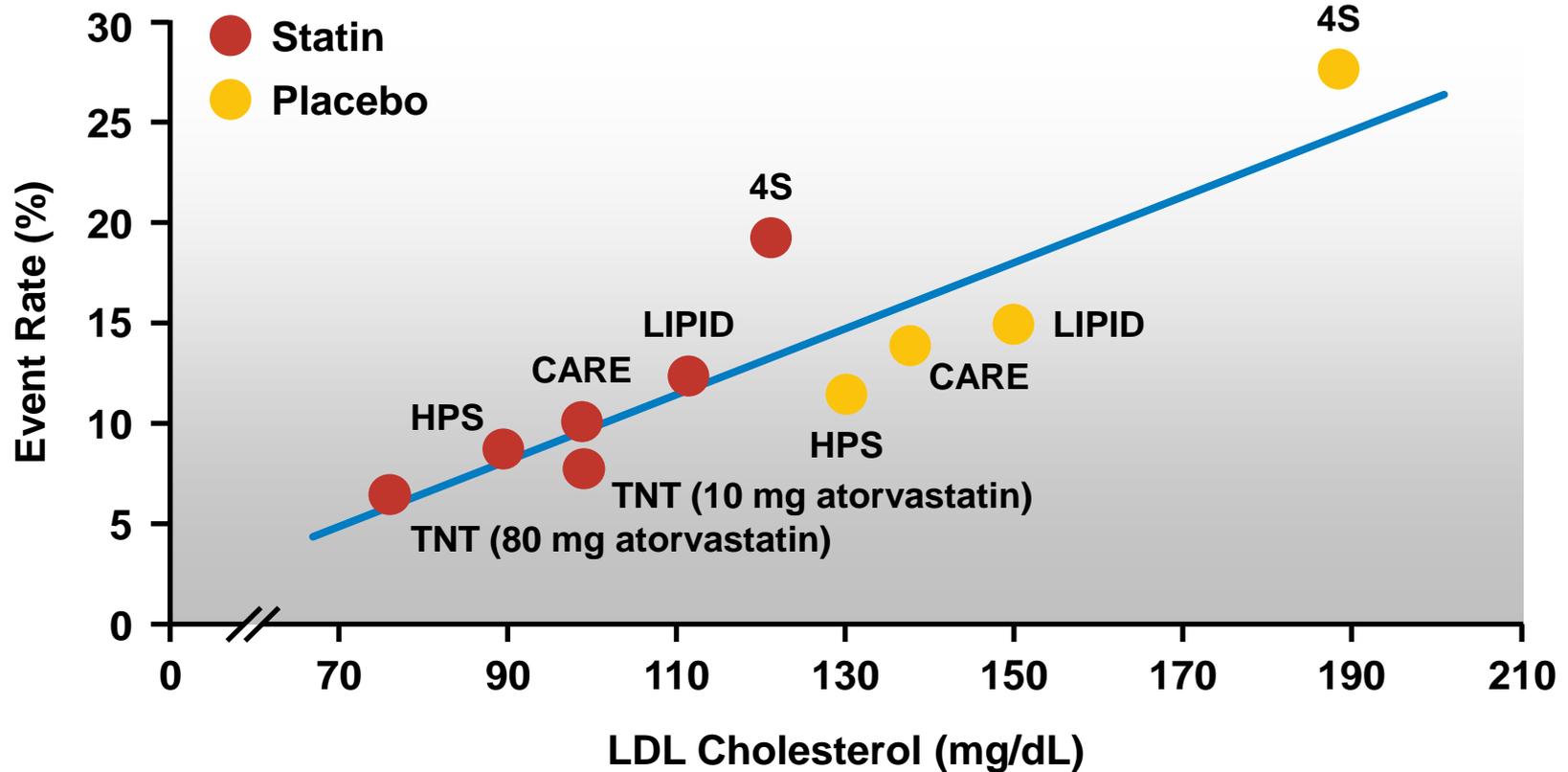
Cardiovascular disease and other major causes of death for all males and females (United States: 2007)



Circulation. 2011;123: e18-e203

Provided November 6, 2012 as part of an oral presentation and is qualified by such, contains forward-looking statements, actual results may vary materially; Amgen disclaims any duty to update.

Lowering LDL Cholesterol Is a Proven Intervention to Reduce Cardiovascular Events



Every 1 mg/dL decrease in LDL-C decreases relative risk for CHD by ~1%–2%

LDL-C = LDL cholesterol; CHD = coronary heart disease
Provided November 6, 2012 as part of an oral presentation and is qualified by such, contains forward-looking statements, actual results may vary materially; Amgen disclaims any duty to update.

Significant Unmet Need Remains in the Treatment of Hypercholesterolemia

- There are more than 200 million cases of dyslipidemia in the US and Europe¹
- Two-thirds of people with dyslipidemia have elevated levels of LDL-C¹
- In the US alone, it is estimated that hypercholesterolemia affects ~70 million adults²
- Of these, roughly half receive therapy, and 11 million are not adequately controlled²
- ~27% of treated patients do not achieve CPG LDL-C goals³
- Approximately two-thirds of treated high-risk patients do not achieve CPG LDL-C goals³

CPG = clinical practice guidelines

1. *The Lancet*. 2003;362: 717-31; 2. 2005–2008 NHANES; 3. *Circulation*. 2009;120: 28-34

Provided November 6, 2012 as part of an oral presentation and is qualified by such, contains forward-looking statements, actual results may vary materially; Amgen disclaims any duty to update.

Four Key Scientific Questions in Phase 2

Question	Study	Description
What is the safety/efficacy as monotherapy ?	MENDEL	Monotherapy
What is the safety/efficacy with statins ?	LAPLACE-TIMI 57	Combination therapy
What is the safety/efficacy with statins in patients with LDL-R mutations ?	RUTHERFORD	HeFH
What is the safety/efficacy in statin-intolerant patients?	GAUSS	Statin-intolerant

HeFH = heterozygous familial hypercholesterolemia

Provided November 6, 2012 as part of an oral presentation and is qualified by such, contains forward-looking statements, actual results may vary materially; Amgen disclaims any duty to update.

AMG 145 Phase 2 Program

	Without Statins*	With Statins ± Ezetimibe
Every 2 and 4 week dosing	MENDEL (Study 20101154) Monotherapy N = 405	LAPLACE-TIMI 57 (Study 20101155) Combination therapy N = 600
Every 4 week dosing	GAUSS** (Study 20090159) Statin-intolerant N = 150	RUTHERFORD (Study 20090158) Heterozygous FH N = 150

*Include ezetimibe comparator

**16% of subjects in GAUSS (phase 2 statin-intolerant study) were on/able to tolerate a low or atypical dose of a statin

Provided November 6, 2012 as part of an oral presentation and is qualified by such, contains forward-looking statements, actual results may vary materially; Amgen disclaims any duty to update.

Efficacy and Safety of a Fully Human Monoclonal Antibody Against PCSK9 as Monotherapy for Hypercholesterolemia: Results from the MENDEL Study, a Global Phase 2 Trial of AMG 145

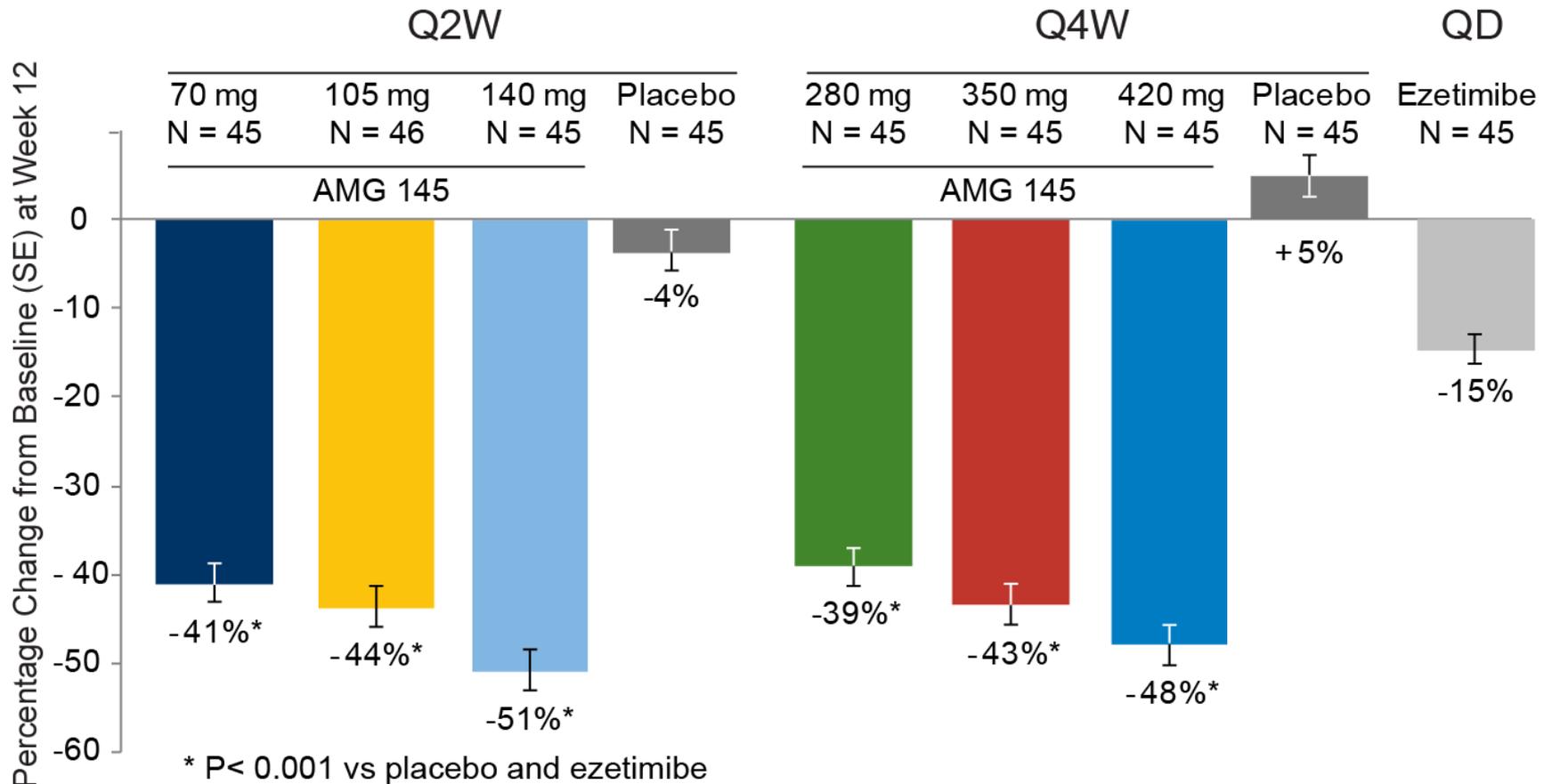
Michael J. Koren¹, Rob Scott², Jae B. Kim², Beat Knusel²,
Thomas Liu², Lei Lei², Michael Bolognese³, Scott M. Wasserman²

¹Jacksonville Center for Clinical Research, Jacksonville, FL, USA; ²Amgen Inc.,
Thousand Oaks, CA, USA; ³Bethesda Health Research Center, Bethesda, MD, USA

November 6, 2012, Session CS.03

American Heart Association Scientific Sessions, Los Angeles, CA

MENDEL: Effects of AMG 145 Versus Placebo and Ezetimibe on LDL-C



LDL-C values at baseline and week 12 were measured using preparative ultracentrifugation. Q2W = every 2 weeks; Q4W = every 4 weeks; QD = daily; SE = standard error

MENDEL: Safety and Tolerability

Adverse Events, Patient Incidence, n (%)	Q2W				Q4W				Ezetimibe 10 mg QD N=45
	Placebo N=45	AMG 145			Placebo N=45	AMG 145			
		70 mg N=45	105 mg N=46	140 mg N=45		280 mg N=45	350 mg N=45	420 mg N=45	
Treatment-emergent AEs	19 (42)	24 (53)	22 (48)	26 (58)	22 (49)	22 (49)	23 (51)	19 (42)	26 (58)
Serious AEs	0 (0)	0 (0)	1 (2.2)	1 (2.2)	0 (0)	0 (0.0)	1 (2.2)	0 (0)	0 (0)
Deaths	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Treatment-related AEs	3 (7)	4 (9)	7 (15)	9 (20)	5 (11)	5 (11)	4 (9)	2 (4)	3 (7)
Most common AEs									
Upper respiratory tract infection	5 (11)	3 (7)	2 (4)	2 (4)	2 (4)	3 (7)	3 (7)	4 (9)	5 (11)
Back pain	0 (0)	1 (2)	1 (2)	2 (4)	4 (9)	1 (2)	1 (2)	3 (7)	1 (2)
Diarrhea	0 (0)	2 (4)	0 (0)	2 (4)	3 (7)	0 (0)	3 (7)	3 (7)	1 (2)
AEs leading to discontinuation	1 (2)	0 (0)	0 (0)	0 (0)	1 (2)	0 (0)	0 (0)	0 (0)	0 (0)
Adjudicated muscle-related AEs	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Injection-site reactions	1 (2)	1 (2)	6 (13)	2 (4)	3 (7)	3 (7)	3 (7)	0 (0)	0 (0)

AE = Adverse event

Some patients experienced more than 1 AE.



LAPLACE-TIMI 57 Primary Results

A Double-blind, Randomized, Placebo-controlled, Dose-ranging Study to Evaluate the Efficacy, Safety, and Tolerability of a Monoclonal Antibody to PCSK9 in Combination with a Statin in Patients with Hypercholesterolemia

Robert P. Giugliano, MD, SM, FAHA, FACC

TIMI Study Group, Cardiovascular Division

Brigham and Women's Hospital

Harvard Medical School, Boston, MA

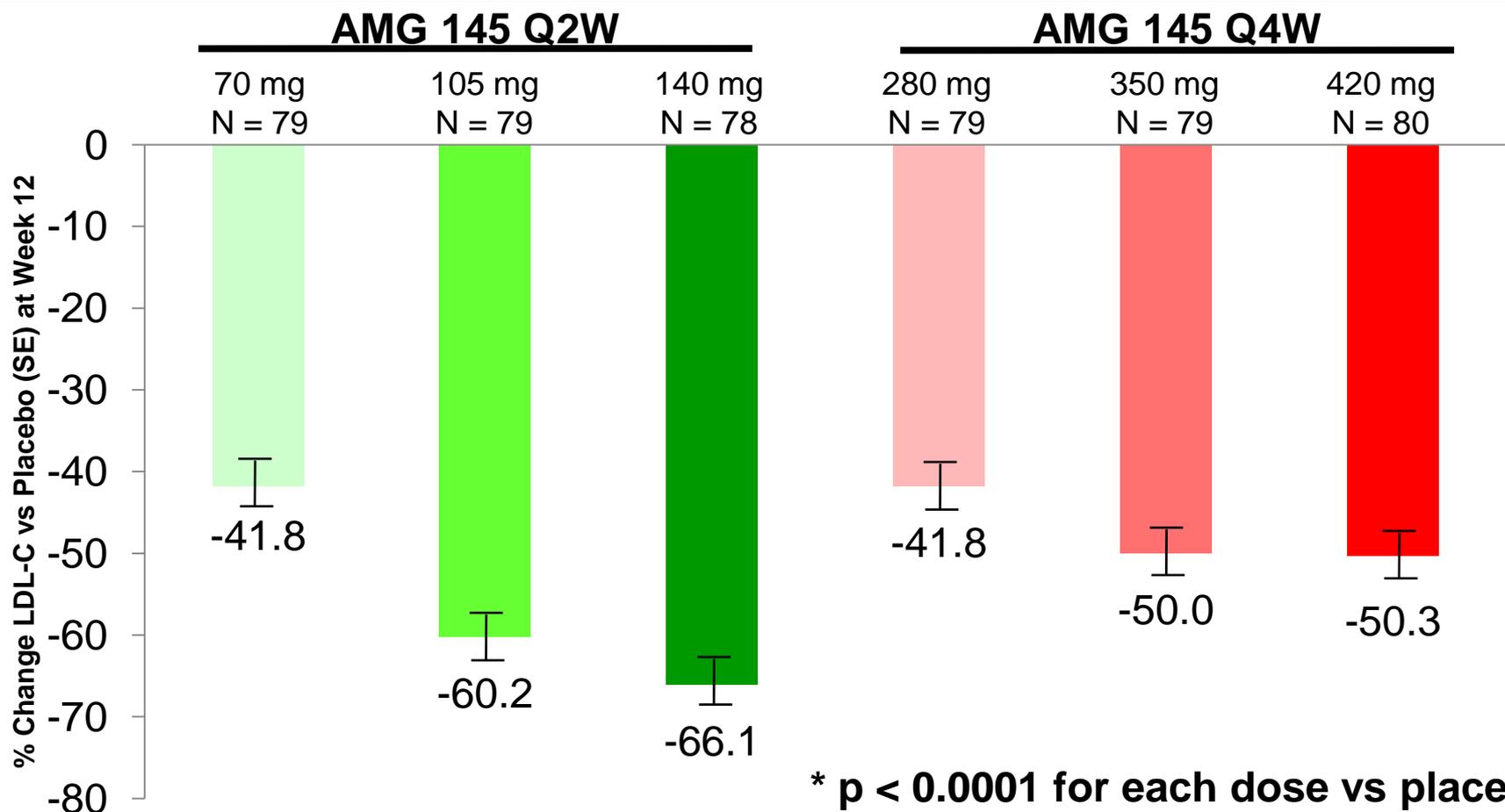
Supported by research grant from Amgen, Inc.



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



Primary Endpoint: AMG 145 Reduced LDL-C at 12 wks



LDL-C at 12 wks						
Mean (mg/dL)	73	53	44	69	60	58
(SD)	(25)	(21)	(25)	(28)	(23)	(26)



Safety



Adverse Events, Patient Incidence, n	Q2W Dose Groups				Q4W Dose Groups				Total N = 629
	Placebo N = 78	AMG 145			Placebo N = 77	AMG 145			
		70 mg N = 79	105 mg N = 79	140 mg N = 78		280 mg N = 79	350 mg N = 79	420 mg N = 80	
Adverse events	33	41	52	43	38	45	48	48	348
Serious AE	4	0	1	4	0	2	2	2	15
Lead to drug DC	0	0	0	2*	0	0	0	0	2
Drug related AEs	7	4	9	4	4	6	7	9	50 [†]
Lead to drug DC	0	0	0	0	0	0	0	0	0
Injection site rxn	2	1	1	0	1	2	3	1	11
AST or ALT >3x ULN	1	0	0	0	0	0	0	0	1
CPK >5X ULN	0	1	1	1	0	0	0	1	4**
CV events‡	1	1	0	4	0	1	1	0	8
Death	0	0	0	1	0	0	0	0	1

*Both events were reported as non-serious by the investigators.

†All 50 were reported as non-serious by the investigator and none led to discontinuation of drug

** All were asymptomatic ‡Acute coronary syndrome, coronary revascularization, TIA, congestive heart failure requiring hospitalization, or death



Goal Achievement after Utilizing an Anti-PCSK9 Antibody in Statin-Intolerant Subjects (GAUSS): Results from a Randomized, Double-blind, Placebo and Ezetimibe Controlled Study

Evan A. Stein¹, David Sullivan², Anders G. Olsson³, Rob Scott⁴, Jae B. Kim⁴, Allen Xue⁴, Thomas Liu⁴, Scott M. Wasserman⁴

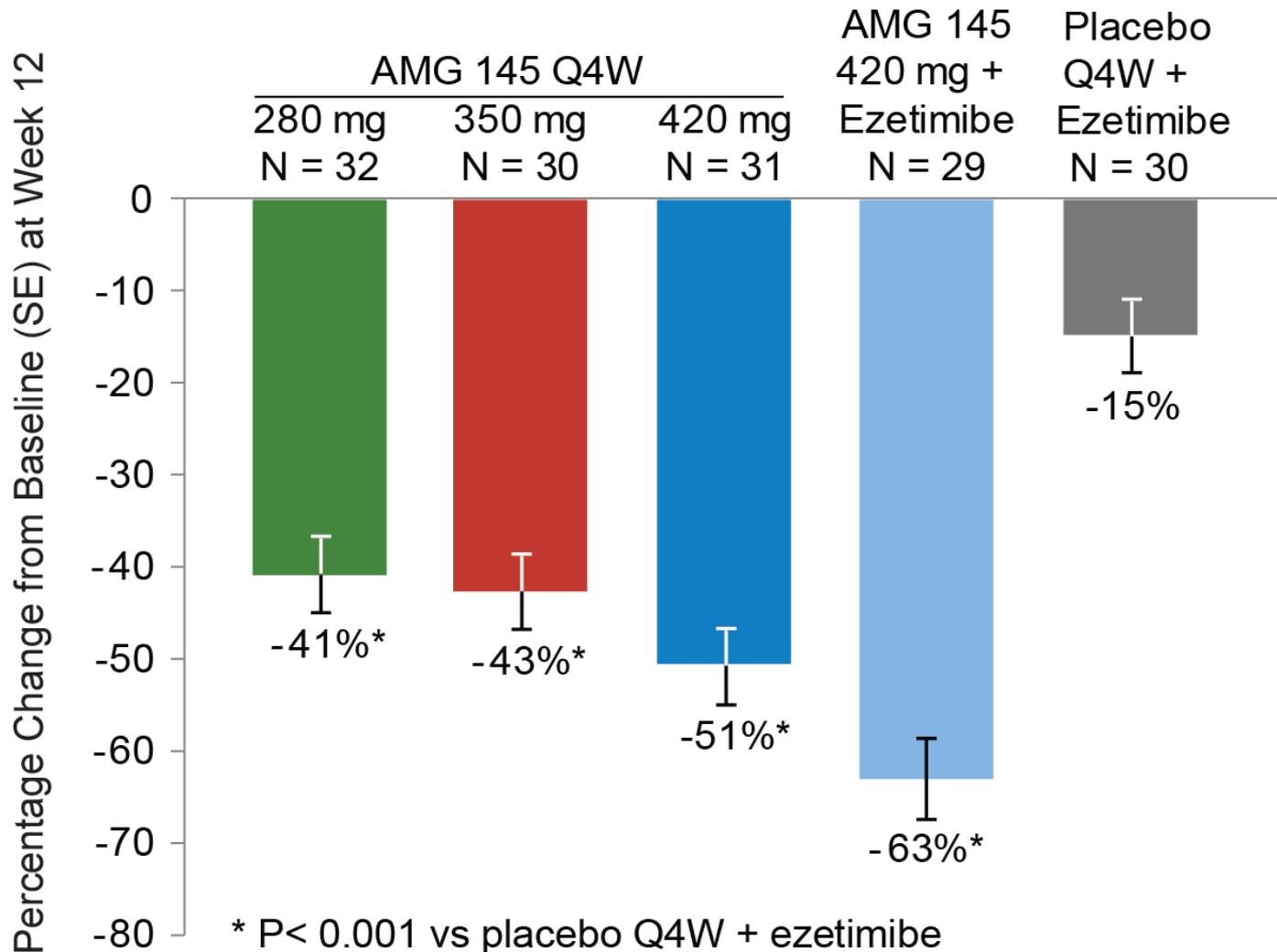
¹Metabolic and Atherosclerosis Research Center, Cincinnati, OH, USA; ²Department of Clinical Biochemistry, Royal Prince Alfred Hospital, Camperdown, NSW, Australia;

³Stockholm Heart Center, Stockholm, Sweden; ⁴Amgen Inc., Thousand Oaks, CA, USA

November 5, 2012, Session LBCT.04

American Heart Association Scientific Sessions, Los Angeles, CA

GAUSS: % Change in LDL-C, by UC, from Baseline at Week 12



LDL-C values at baseline and week 12 were measured using preparative ultracentrifugation.

Q4W = every 4 weeks; QD = daily; SE = standard error

GAUSS: Safety and Tolerability

Adverse Events, Patient Incidence, n (%)	AMG 145			AMG 145 420 mg + Ezetimibe 10 mg N = 30	Placebo Q4W + Ezetimibe N = 32
	280 mg N = 32	350 mg N = 31	420 mg N = 32		
Treatment-emergent AEs	22 (68.8)	15 (48.4)	18 (56.3)	20 (66.7)	19 (59.4)
Serious AEs*	2 (6.3)	1 (3.2)	1 (3.1)	0 (0.0)	0 (0.0)
Deaths	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
Treatment-related AEs	8 (25.0)	3 (9.7)	6 (18.8)	5 (16.7)	7 (21.9)
Muscle-related AEs					
Myalgia	5 (15.6)	1 (3.2)	1 (3.1)	6 (20.0)	1 (3.1)
Muscle fatigue	2 (6.3)	0 (0.0)	0 (0.0)	0 (0.0)	1 (3.1)
Muscle spasms	1 (3.1)	2 (6.5)	0 (0.0)	0 (0.0)	3 (9.4)
AEs leading to discontinuation	0 (0.0)	1 (3.2)	1 (3.1)	1 (3.3)	2 (6.3)
Other most commonly reported AEs					
Nasopharyngitis	2 (6.3)	2 (6.5)	1 (3.1)	3 (10.0)	5 (15.6)
Nausea	2 (6.3)	1 (3.2)	1 (3.1)	0 (0.0)	1 (3.1)
Fatigue	4 (12.5)	0 (0.0)	0 (0.0)	0 (0.0)	2 (6.3)

*Four serious adverse events were reported for AMG 145: acute pancreatitis, coronary artery disease, hip fracture, and syncope. **None were considered treatment related.**

Reduction of LDL-C with PCSK9 Inhibition in Heterozygous Familial Hypercholesterolemia Disorder (RUTHERFORD): Results from a Phase 2, Randomized, Double-Blind, Placebo-Controlled Trial

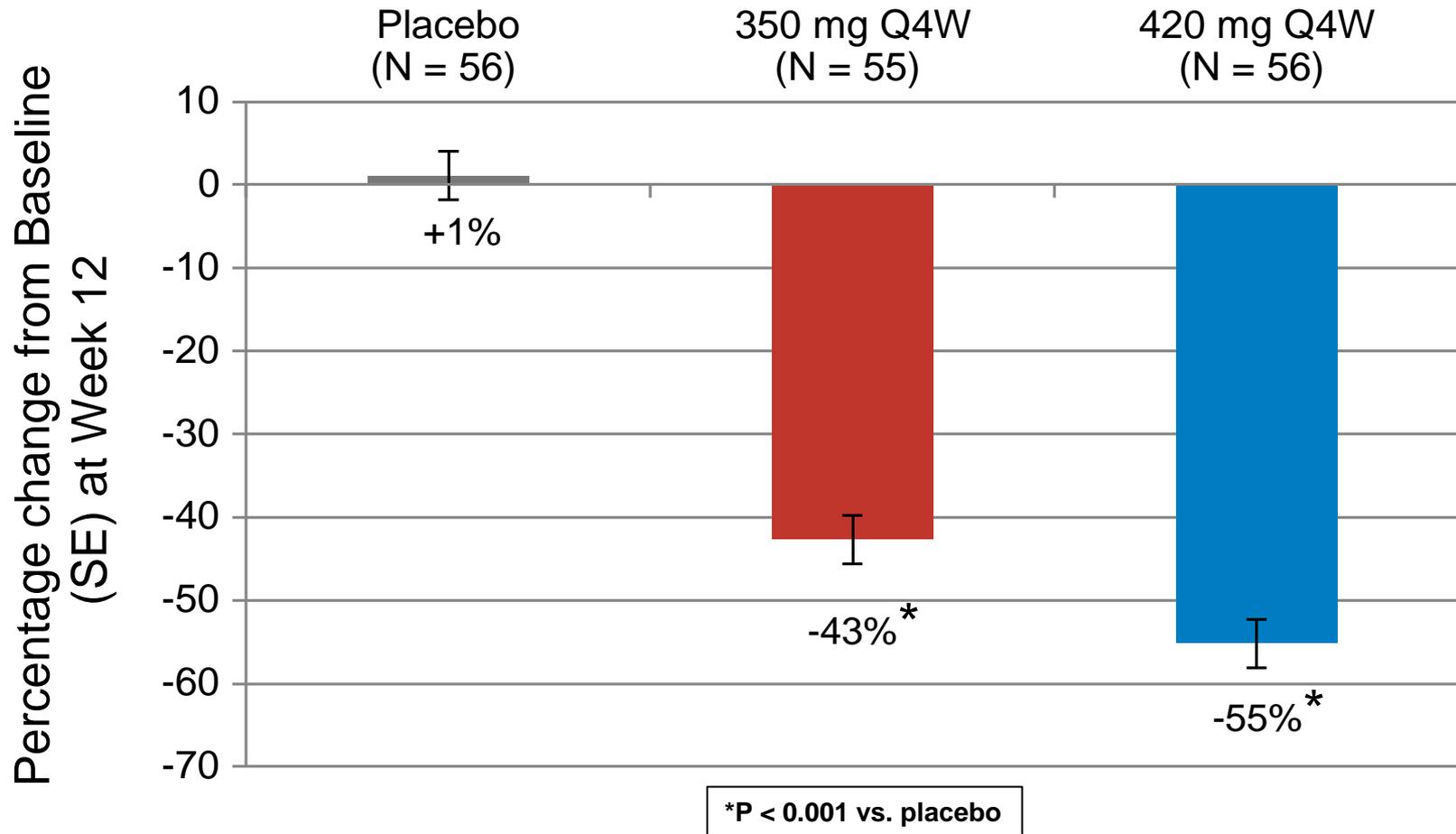
Frederick Raal¹, Rob Scott², Ransi Somaratne², Ian Bridges³, Thomas Liu², Scott M. Wasserman², Evan A. Stein⁴

¹Carbohydrate & Lipid Metabolism Research Unit, University of Witwatersrand, Johannesburg, South Africa; ²Amgen Inc., Thousand Oaks, CA, USA; ³Amgen Ltd., Uxbridge, UK; ⁴Metabolic and Atherosclerosis Research Center, Cincinnati, OH, USA

November 5, 2012, Session: LBCT.04

American Heart Association Scientific Sessions, Los Angeles, CA

RUTHERFORD: % Change in LDL-C, by UC, from Baseline to Week 12



Q4W = every 4 weeks; SE = standard error; UC = ultracentrifugation

LDL-C values at baseline and week 12 were measured using preparative UC.

Least Square Means are presented from the ANCOVA model including treatment and stratification factors as covariates.

Missing UC LDL-C values at week 12 were imputed using last observation carried forward and calculated LDL-C. A Hochberg adjustment was used to control the family wise error rate at ≤ 0.05 .

RUTHERFORD: Safety and Tolerability

Adverse Events, Patient Incidence, n (%)	Placebo N = 56	AMG 145	
		350 mg N = 55	420 mg N = 56
Treatment-emergent AEs	33 (58.9)	32 (58.2)	37 (66.1)
Most common AEs			
Nasopharyngitis	6 (10.7)	7 (12.7)	7 (12.5)
Injection site pain	1 (1.8)	5 (9.1)	2 (3.6)
Headache	5 (8.9)	3 (5.5)	3 (5.4)
Serious AEs	0 (0)	0 (0)	2 (3.6)
Deaths	0 (0)	0 (0)	0 (0)
Treatment-related AEs	6 (10.7)	13 (23.6)	8 (14.3)
AEs leading to discontinuation	1 (1.8)	1 (1.8)	1 (1.8)
Muscle-related AEs	2 (3.6)	2 (3.6)	4 (7.1)
Injection-site reactions	3 (5.4)	6 (10.9)	2 (3.6)

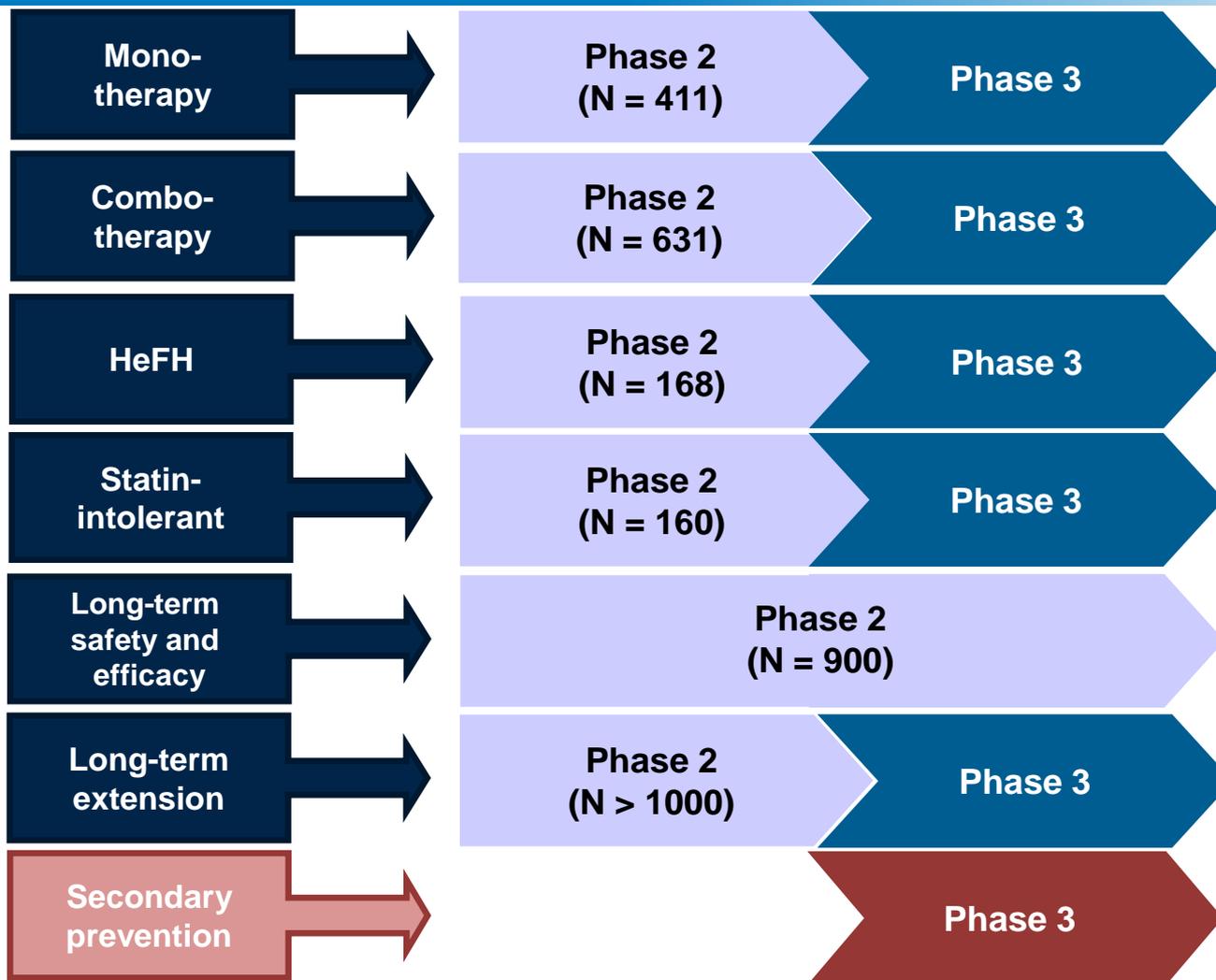
AE = Adverse event

Some patients experienced more than 1 AE.

Phase 2 Program Summary

- **AMG 145 therapy significantly reduced LDL-C compared to the control arms at 12 weeks in hypercholesterolemic subjects**
 - **As monotherapy**
 - **In combination with a stable regimen of statin ± ezetimibe**
 - **With statin-intolerance**
 - **With heterozygous familial hypercholesterolemia (on statin ± ezetimibe)**
- **AMG 145 had an acceptable safety profile and also resulted in improvements in other lipid and lipoprotein parameters**
- **These results in over 1300 subjects suggest that AMG 145 may offer a new effective treatment option to further reduce LDL-C in patients unable to achieve optimal LDL-C targets on current medications**

Development Plan Supports LDL-C and Secondary Prevention Submissions



Provided November 6, 2012 as part of an oral presentation and is qualified by such, contains forward-looking statements, actual results may vary materially; Amgen disclaims any duty to update.



Pioneering science delivers vital medicines™

November 6, 2012

Amgen Investor Event 2012 AHA Scientific Sessions

Sean E. Harper, MD

Executive Vice President, Research and Development

Scott M. Wasserman, MD, FACC

Executive Medical Director, Clinical Development