New Detailed Data from Three Phase 3 Pivotal Studies Show Amgen's Novel Investigational Cholesterol-Lowering Medicine Evolocumab Significantly Reduced LDL Cholesterol By 55-66 Percent Compared To Placebo In Patients With High Cholesterol

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Data From Three Separate Phase 3 Studies of Evolocumab (AMG 145), a PCSK9 Inhibitor, Presented as Featured Clinical Research at ACC.14

Positive Results From the Long-Term Safety and Efficacy Study DESCARTES Simultaneously Published in the New England Journal of Medicine and the Monotherapy Study MENDEL-2 Results Simultaneously Published in the Journal of the American College of Cardiology

Data From Two Additional Phase 3 Studies on Evolocumab to be Presented in Late-Breaking Session Tomorrow at ACC.14

THOUSAND OAKS, Calif., March 29, 2014 /PRNewswire/ -- Amgen (NASDAQ: AMGN) today announced new detailed data from three Phase 3 studies that showed treatment with its novel investigational cholesterol-lowering medication, evolocumab (AMG 145), resulted in a statistically significant reduction of 55-66 percent in low-density lipoprotein cholesterol (LDL-C) compared to placebo in patients with high cholesterol. Results from the three separate Phase 3 studies, MENDEL-2, DESCARTES and RUTHERFORD-2, were presented today as Featured Clinical Research in a Special Session at the American College of Cardiology's 63rd Annual Scientific Session (ACC.14). Data from DESCARTES, the long-term safety and efficacy study, were simultaneously published in the *New England Journal of Medicine* and data from MENDEL-2, the monotherapy study, were simultaneously published in the *Journal of the American College of Cardiology*.

Evolocumab is an investigational fully human monoclonal antibody that inhibits proprotein convertase subtilisin/kexin type 9 (PCSK9), a protein that reduces the liver's ability to remove LDL-C from the blood.[1]

The three Phase 3 studies evaluated evolocumab in different patient populations: as monotherapy in patients with high cholesterol (MENDEL-2); as a long-term 52-week therapy in patients with high cholesterol on risk-based lipid-lowering therapy (DESCARTES); and in combination with statins and other lipid-lowering therapies in patients with heterozygous familial hypercholesterolemia (HeFH), a genetic disorder characterized by elevated LDL-C levels (RUTHERFORD-2).

In MENDEL-2, the most common adverse events (AEs) (≥2 percent in evolocumab combined group) were headache, diarrhea, nausea and urinary tract infection. The most common AEs (>5 percent in evolocumab) in the DESCARTES study were nasopharyngitis, upper respiratory tract infection, influenza and back pain. In RUTHERFORD-2, the most common AEs (≥2 percent in the combined evolocumab group) were nasopharyngitis, headache, contusion (i.e., bruise), back pain, nausea, arthralgia, upper respiratory tract infection, influenza, myalgia and pain in extremity.

"We are pleased to report positive detailed findings from three of our pivotal studies in key patient populations at risk for cardiovascular disease," said Sean E. Harper, M.D., executive vice president of Research and Development at Amgen. "These results add to the growing body of evidence from our comprehensive Phase 3 clinical trial program. We look forward to working with regulatory authorities on our global filing plan in the hopes of bringing this new treatment option to patients with high cholesterol who have an unmet medical need."

MENDEL-2, DESCARTES and RUTHERFORD-2 are three of five Phase 3 evolocumab studies being presented at ACC.14. Data from two other trials, LAPLACE-2 and GAUSS-2, will be featured in a Late-Breaking Clinical Trials session on Sunday, March 30, at 8 a.m. EDT.

"Positive results from these first Phase 3 studies provide encouragement that evolocumab will find use as a treatment for a range of at-risk patients," said Michael Koren, M.D., clinical investigator for the MENDEL-2 and DESCARTES studies and the chief executive officer of the Jacksonville Center for Clinical Research. "The newly released data support the potential of evolocumab in patients with high cholesterol who struggle to keep their LDL-C levels under control despite currently available treatments."

MENDEL-2 (Monoclonal Antibody Against PCSK9 to Reduce Elevated LDL-C in Subjects Currently Not Receiving Drug Therapy for Easing Lipid Levels-2) Primary Results

- The MENDEL-2 study showed that in 614 patients with high cholesterol (LDL-C ≥100 mg/dL and <190 mg/dL) who were not receiving lipid-lowering therapy, treatment with subcutaneous evolocumab significantly reduced mean LDL-C by 55-57 percent from baseline compared to placebo and 38-40 percent from baseline compared to ezetimibe (p<0.001).
    - Results of the study showed the mean percent reduction from baseline in LDL-C at weeks 10 and 12 were 57 percent for evolocumab 140 mg every two weeks and 57 percent for evolocumab 420 mg monthly compared to placebo; and 39 percent for evolocumab 140 mg every two weeks and 40 percent for evolocumab 420 mg monthly compared to ezetimibe.
    - At week 12, the percent reduction from baseline in LDL-C was 57 percent for evolocumab 140 mg every two weeks and 55 percent for evolocumab 420 mg monthly compared to placebo; and 39 percent for evolocumab 140 mg every two weeks and 38 percent for evolocumab 420 mg monthly compared to ezetimibe.
- The most common AEs (≥2 percent in evolocumab combined group) were headache (3.3 percent evolocumab; 3.2 percent

ezetimibe; 2.6 percent placebo), diarrhea (2.9 percent evolocumab; 1.9 percent ezetimibe; 3.9 percent placebo), nausea (2.6 percent evolocumab; 1.9 percent ezetimibe; 0.6 percent placebo) and urinary tract infection (2.3 percent evolocumab; 1.9 percent ezetimibe; 1.3 percent placebo).

DESCARTES (Durable Effect of PCSK9 Antibody CompARed wiTh PlacEbo Study) Primary Results

- The DESCARTES study showed that in 901 patients with high LDL-C and a range of cardiovascular risk, evolocumab 420 mg subcutaneous monthly reduced mean LDL-C by 57 percent from baseline at week 52 compared to placebo (p<0.001).
    - LDL-C reduction for evolocumab at week 12 was consistent with the long-term efficacy at week 52.
    - Compared to placebo, the mean percent LDL-C reductions from baseline with evolocumab at week 52 are 56 percent, 62 percent, 57 percent and 49 percent in diet alone, atorvastatin 10 mg, atorvastatin 80 mg, and atorvastatin 80 mg plus ezetimibe 10 mg groups, respectively.
- The most common AEs (>5 percent in evolocumab) were nasopharyngitis (10.5 percent evolocumab; 9.6 percent placebo), upper respiratory tract infection (9.3 percent evolocumab; 6.3 percent placebo), influenza (7.5 percent evolocumab; 6.3 percent placebo) and back pain (6.2 percent evolocumab; 5.6 percent placebo).

RUTHERFORD-2 (RedUction of LDL-C with PCSK9 InhibiTion in HEteRozygous Familial HypercholesteRolemia Disorder Study-2) Primary Results

- The RUTHERFORD-2 study showed that in 329 HeFH patients on a stable dose of statin and other lipid-lowering therapies, treatment with subcutaneous evolocumab significantly reduced mean LDL-C by 59-66 percent from baseline compared to placebo (p<0.001).
    - Data show the mean percent reduction from baseline in LDL-C at weeks 10 and 12 were 60 percent for evolocumab 140 mg every two weeks and 66 percent for evolocumab 420 mg monthly compared to placebo.
    - At week 12, the percent reduction from baseline in LDL-C was 59 percent for evolocumab 140 mg every two weeks and 61 percent for evolocumab 420 mg monthly compared to placebo.
- The most common AEs (≥2 percent in the combined evolocumab group) were nasopharyngitis (8.6 percent evolocumab; 4.6 percent placebo), headache (4.1 percent evolocumab; 3.7 percent placebo), contusion (i.e., bruise) (4.1 percent evolocumab; 0.9 percent placebo), back pain (3.6 percent evolocumab; 0.9 percent placebo), nausea (3.6 percent evolocumab; 0.9 percent placebo), influenza (3.2 percent evolocumab; 0 percent placebo), myalgia (2.7 percent evolocumab; 0 percent placebo) and pain in the extremity (2.3 percent evolocumab; 2.8 percent placebo).

High cholesterol is the most common form of dyslipidemia, which is an abnormality of lipids in the blood.[2,3] There are approximately 300 million cases of dyslipidemia in the U.S., Japan and Western Europe.[4] According to the Centers for Disease Control and Prevention, more than 71 million American adults have high LDL-C[5], or "bad" cholesterol, and elevated LDL-C is recognized as a major risk factor for cardiovascular disease.[6]

Patients with familial hypercholesterolemia, an inherited condition that causes high levels of LDL-C beginning at birth, are at high-risk for cardiovascular events at an early age.[7] Heterozygous familial hypercholesterolemia is one of the most common genetic disorders, affecting approximately one out of every 200 to 500 people worldwide.[8,9]

Amgen will also host a webcast investor meeting at ACC.14 on Sunday, March 30, at 7 p.m. EDT. Sean E. Harper, M.D., executive vice president of Research and Development at Amgen, along with members of Amgen's clinical development team and clinical investigators, will participate at the investor meeting to discuss Amgen's cardiovascular program, including the primary analyses of five Phase 3 evolocumab studies being presented at ACC.14.

Live audio of the investor meeting will be simultaneously broadcast over the Internet and will be available to members of the news media, investors and the general public.

The webcast, as with other selected presentations regarding developments in Amgen's business given by management at certain investor and medical conferences, can be found on Amgen's website, www.amgen.com, under Investors. Information regarding presentation times, webcast availability and webcast links are noted on Amgen's Investor Relations Events Calendar. The webcast will be archived and available for replay for at least 90 days after the event.

MENDEL-2 Study Design
MENDEL-2 (Monoclonal Antibody Against PCSK9 to Reduce Elevated LDL-C in Subjects Currently Not Receiving Drug Therapy For Easing Lipid Levels-2) is a Phase 3 randomized, multicenter, double-blind, double-dummy, placebo- and ezetimibe-controlled parallel group study designed to evaluate the efficacy and safety of evolocumab in 614 hyperlipidemic patients with a 10-year Framingham risk score of 10 percent or less who were not receiving lipid-lowering therapy. Patients were randomized to one of six treatment groups to compare two dosing regimens of evolocumab (140 mg every two weeks or 420 mg monthly) with placebo and ezetimibe (10 mg daily). The co-primary endpoints were the percent reduction from baseline in LDL-C at week 12 and the mean percent reduction from baseline in LDL-C at weeks 10 and 12. Co-secondary efficacy endpoints included means at weeks 10 and 12 and at week 12 for the following: absolute change from baseline in LDL-C; LDL-C <70 mg/dL; and the percentage change from baseline in non-high-density lipoprotein cholesterol (non-HDL-C), apolipoprotein B (ApoB), total cholesterol (TC)/HDL-C ratio, ApoB/apolipoprotein A1 (ApoA1) ratio, lipoprotein(a), triglycerides, HDL-C and very low-density lipoprotein cholesterol (VLDL-C).

DESCARTES Study Design
DESCARTES (Durable Effect of PCSK9 Antibody CompARed wiTh PlacEbo Study) is a Phase 3 randomized, multicenter, double-blind, placebo-controlled study designed to evaluate the long-term (52-week) safety, tolerability and efficacy of evolocumab in patients with hyperlipidemia at risk for cardiovascular disease. Background lipid-lowering therapy was optimized to one of four treatment groups (diet alone; diet plus atorvastatin 10 mg; diet

plus atorvastatin 80 mg; and diet plus atorvastatin 80 mg plus ezetimibe 10 mg) for individual patients based on their LDL-C and cardiovascular risk according to the National Cholesterol Education Program Adult Treatment Panel (NCEP ATP) III risk categories. After optimization, patients were maintained on therapy for at least four weeks. A total of 901 patients with a fasting LDL-C ≥75 mg/dL were then randomized and received monthly subcutaneous evolocumab 420 mg or placebo in combination with background lipid-lowering therapy.

The primary endpoint was percent change from baseline in LDL-C, measured by the accepted standard, preparative ultracentrifugation, after 52 weeks of treatment. Secondary efficacy endpoints included the absolute change from baseline in LDL-C and LDL-C response (LDL-C <70 mg/dL [1.8 mmol/L]) at week 52, percent change from baseline in LDL-C and total cholesterol (TC) at week 12, and percent change from baseline at week 52 in TC, non-high-density lipoprotein cholesterol (non-HDL-C), apolipoprotein B (ApoB), TC/HDL-C ratio, ApoB/apolipoprotein A1 (ApoA1) ratio, lipoprotein(a), triglycerides, HDL-C and very low density lipoprotein cholesterol (VLDL-C).

RUTHERFORD-2 Study Design
RUTHERFORD-2 (RedUction of LDL-C with PCSK9 InhibiTion in HEteRozygous Familial HypercholesteRolemia Disorder Study-2) is a Phase 3 randomized, multicenter, double-blind, placebo-controlled trial designed to evaluate the safety, tolerability and efficacy of evolocumab in 329 patients with HeFH and an LDL-C ≥100 mg/dL who were on a stable dose of statin therapy and lipid-lowering medication. Patients were randomized to one of four treatment groups to compare subcutaneous evolocumab (140 mg every two weeks or 420 mg monthly) with subcutaneous placebo (every two weeks or monthly). The co-primary endpoints were the percent reduction from baseline in LDL-C at week 12 and the mean percent reduction from baseline in LDL-C at weeks 10 and 12. Co-secondary efficacy endpoints included means at weeks 10 and 12 and at week 12 for the following: absolute change from baseline in LDL-C; LDL-C <70 mg/dL; and the percentage change from baseline in non-high-density lipoprotein cholesterol (non-HDL-C), apolipoprotein B (ApoB), total cholesterol (TC)/HDL-C ratio, ApoB/apolipoprotein A1 (ApoA1) ratio, lipoprotein(a), triglycerides, HDL-C and very low-density lipoprotein cholesterol (VLDL-C).

About Evolocumab
Evolocumab is a fully human monoclonal antibody that inhibits proprotein convertase subtilisin/kexin type 9 (PCSK9).[1] PCSK9 is a protein that targets LDL receptors for degradation and thereby reduces the liver's ability to remove LDL-C, or "bad" cholesterol, from the blood.[10] Evolocumab, being developed by Amgen scientists, is designed to bind to PCSK9 and inhibit PCSK9 from binding to LDL receptors on the liver surface. In the absence of PCSK9, there are more LDL receptors on the surface of the liver to remove LDL-C from the blood.[1]

About PROFICIO: The Evolocumab Clinical Trial Program
PROFICIO, which stands for the Program to Reduce LDL-C and Cardiovascular Outcomes Following Inhibition of PCSK9 In Different POpulations, is a large and comprehensive clinical trial program evaluating evolocumab in 20 clinical trials, with a combined planned enrollment of nearly 30,000 patients.

The Phase 3 program includes 14 trials to evaluate evolocumab administered every two weeks and monthly in multiple patient populations, including in combination with statins in patients with hyperlipidemia (LAPLACE-2 and YUKAWA-2); in patients with hyperlipidemia who cannot tolerate statins (GAUSS-2 and GAUSS-3); as a stand-alone treatment in patients with hyperlipidemia (MENDEL-2); in patients whose elevated cholesterol is caused by genetic disorders called heterozygous (RUTHERFORD-2 and TAUSSIG) and homozygous (TESLA and TAUSSIG) familial hypercholesterolemia; as well as the administration of evolocumab (THOMAS-1 and THOMAS-2).

Five studies in the evolocumab Phase 3 program will provide long-term safety and efficacy data. These include FOURIER (Further Cardiovascular OUtcomes Research with PCSK9 Inhibition in Subjects with Elevated Risk), which will assess whether treatment with evolocumab in combination with statin therapy compared to placebo and statin therapy reduces recurrent cardiovascular events in approximately 22,500 patients with cardiovascular disease; DESCARTES (Durable Effect of PCSK9 Antibody CompARed wiTh PlacEbo Study) in patients with hyperlipidemia at risk for cardiovascular disease; OSLER-2 (Open Label Study of Long TERm Evaluation Against LDL-C Trial-2) in patients with high cholesterol who completed any of the Phase 3 studies; GLAGOV (GLobal Assessment of Plaque ReGression with a PCSK9 AntibOdy as Measured by IntraVascular Ultrasound), which will determine the effect of evolocumab on coronary atherosclerosis in approximately 950 patients undergoing cardiac catheterization; and TAUSSIG (Trial Assessing Long Term USe of PCSK9 Inhibition in Subjects with Genetic LDL Disorders), which will assess the long-term safety and efficacy of evolocumab on LDL-C in patients with severe familial hypercholesterolemia.

About Amgen's Commitment to Cardiovascular Disease
Amgen is dedicated to addressing important scientific questions in order to advance care and improve the lives of patients with cardiovascular disease. Through its own research and development efforts and innovative partnerships, Amgen has built a robust cardiology pipeline consisting of several investigational molecules in an effort to address a number of today's important unmet patient needs, such as high cholesterol and heart failure.

About Amgen
Amgen is committed to unlocking the potential of biology for patients suffering from serious illnesses by discovering, developing, manufacturing and delivering innovative human therapeutics. This approach begins by using tools like advanced human genetics to unravel the complexities of disease and understand the fundamentals of human biology.

Amgen focuses on areas of high unmet medical need and leverages its biologics manufacturing expertise to strive for solutions that improve health outcomes and dramatically improve people's lives. A biotechnology pioneer since 1980, Amgen has grown to be the world's largest independent biotechnology company, has reached millions of patients around the world and is developing a pipeline of medicines with breakaway potential.

For more information, visit www.amgen.com and follow us on www.twitter.com/amgen.

Forward-Looking Statements
This news release 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 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 any subsequent 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 March 29, 2014, and expressly disclaims any duty to update information contained in this news release.

No forward-looking statement can be guaranteed and actual results may differ materially from those Amgen Inc. and its subsidiaries (which are collectively referred to as we, or us) project. Discovery or identification of new product candidates or development of new indications for existing products cannot be guaranteed and movement from concept to product is uncertain; consequently, there can be no guarantee that any particular product candidate or development of a new indication for an existing product will be successful and become a commercial product. Further, preclinical results do not guarantee safe and effective performance of product candidates in humans. The complexity of the human body cannot be perfectly, or sometimes, even adequately modeled by computer or cell culture systems or animal models. The length of time that it takes for us and our partners to complete clinical trials and obtain regulatory approval for product marketing has in the past varied and we expect similar variability in the future. We develop product candidates internally and through licensing collaborations, partnerships and joint ventures. Product candidates that are derived from relationships may be subject to disputes between the parties or may prove to be not as effective or as safe as we may have believed at the time of entering into such relationship. Also, 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 product liability claims. 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. 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, sales of our products (including products of our wholly-owned subsidiaries) are affected by the 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. 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. We believe that some of our newer products, product candidates or new indications for existing products, may face competition when and as they are approved and marketed. Our products may compete against products that have lower prices, established reimbursement, superior performance, are easier to administer, or that are otherwise competitive with our products. In addition, while we and our partners routinely obtain patents for products and technology, the protection of our products offered by patents and patent applications may be challenged, invalidated or circumvented by our competitors and there can be no guarantee of our or our partners' ability to obtain or maintain patent protection for our products or product candidates. We cannot guarantee that we will be able to produce commercially successful products or maintain the commercial success of our existing products. Our stock price may be affected by actual or perceived market opportunity, competitive position, and success or failure of our products or product candidates. Further, the discovery of significant problems with a product similar to one of our products that implicate an entire class of products could have a material adverse effect on sales of the affected products and on our business and results of operations. Our efforts to integrate the operations of companies we have acquired may not be successful.

The scientific information discussed in this news release related to our product candidates is preliminary and investigative. Such product candidates are not approved by the U.S. Food and Drug Administration (FDA), and no conclusions can or should be drawn regarding the safety or effectiveness of the product candidates.

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U.S. Burden of Cardiovascular Disease Infographic

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