

argenx announces publication of efgartigimod Phase 2 myasthenia gravis trial results in Neurology

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Breda, the Netherlands / Ghent, Belgium – argenx (Euronext & Nasdaq: ARGX), a clinical-stage biotechnology company developing a deep pipeline of differentiated antibody-based therapies for the treatment of severe autoimmune diseases and cancer, today announced the publication in *Neurology* of the results from the completed Phase 2 clinical trial of FcRn-antagonist efgartigimod (ARGX-113) in patients with generalized myasthenia gravis (gMG). The *Neurology* publication can be accessed here.

"We are pleased to have the full results of our Phase 2 trial of efgartigimod in gMG published today in the esteemed, peer-reviewed journal, *Neurology*. Through this publication, the unique properties of our drug candidate and its potential in gMG are now available to the broader neurology community and show that by reducing pathogenic autoantibodies, we have observed rapid and sustained clinical activity in a tolerable way," said Hans de Haard, CSO of argenx. "Based on these clinical data, we launched a global Phase 3 registration trial in gMG in September 2018."

Efgartigimod is currently being evaluated as a treatment for severe autoimmune indications with programs ongoing in gMG, primary immune thrombocytopenia (ITP), pemphigus vulgaris (PV), and chronic inflammatory demyelinating polyneuropathy (CIDP). Data reported to-date have shown that efgartigimod is well-tolerated, with reductions in pathogenic autoantibodies correlating with improvements in clinical scores. The Phase 3 ADAPT trial was launched in September 2018 evaluating intravenously (IV) administered efgartigimod in gMG and data are expected in 2020. A second Phase 3 program of efgartigimod is expected to launch this year in ITP and will evaluate both the IV and a subcutaneous formulation. A Phase 2 proof-of-concept trial of IV efgartigimod is ongoing in PV and data are expected in 2020. argenx is planning to launch a Phase 2 proof-of-concept trial in CIDP before the end of 2019.

About efgartigimod

Efgartigimod (ARGX-113) is an IgG Fc fragment engineered to optimally antagonize the neonatal Fc Receptor (FcRn) for the treatment of IgG-mediated autoimmune diseases. FcRn plays a central role in rescuing IgG from degradation in the lysosome through a recycling pathway. Through inhibition of FcRn, efgartigimod leads to fast depletion of the autoimmune disease-causing IgG autoantibodies. Efgartigimod binds in the same way as endogenous IgG, the natural ligand of FcRn, and has been engineered with ABDEG™ mutations to increase its affinity for FcRn while preserving the characteristic pH-dependent binding, contributing to its long serum half-life, pharmacodynamic effect and potentially enhanced tissue penetration. The development work on efgartigimod is conducted in close collaboration with Prof. E. Sally Ward (University of Texas Southwestern Medical and Texas A&M University Health Science Center, a part of Texas A&M University (TAMHSC)).

About argenx

argenx is a clinical-stage biotechnology company developing a deep pipeline of differentiated antibody-based therapies for the treatment of severe autoimmune diseases and cancer. The company is focused on developing product candidates with the potential to be either first-in-class against novel targets or best-in-class against known, but complex, targets in order to treat diseases with a significant unmet medical need. argenx's ability to execute on this focus is enabled by its suite of differentiated technologies. The SIMPLE Antibody™ Platform, based on the powerful llama immune system, allows argenx to exploit novel and complex targets, and its three complementary Fc engineering technologies are designed to expand the therapeutic index of its product candidates.

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Forward-looking Statements

The contents of this announcement include statements that are, or may be deemed to be, "forward-looking statements." These forward-looking statements can be identified by the use of forward-looking terminology, including the terms "believes," "estimates," "anticipates," "expects," "intends," "may," "will," or "should," and include statements argenx makes concerning the intended results of its strategy and argenx's advancement of, and anticipated clinical development and regulatory milestones and plans, including the timing of planned clinical trials and expected data readouts, related to efgartigimod. By their nature, forward-looking statements involve risks and uncertainties and readers are cautioned that any such forward-looking statements are not guarantees of future performance, argenx's actual results may differ materially from those predicted by the forward-looking statements as a result of various important factors, including argenx's expectations regarding its the inherent uncertainties associated with competitive developments, preclinical and clinical trial and product development activities and regulatory approval requirements: argenx's reliance on collaborations with third parties; estimating the commercial potential of argenx's product candidates; argenx's ability to obtain and maintain protection of intellectual property for its technologies and product candidates; argenx's limited operating history; and argenx's ability to obtain additional funding for operations and to complete the development and commercialization of its product candidates. A further list and description of these risks, uncertainties and other risks can be found in argenx's U.S. Securities and Exchange Commission (SEC) filings and reports, including in argenx's most recent annual report on Form 20-F filed with the SEC as well as subsequent filings and reports filed by argenx with the SEC. Given these uncertainties, the reader is advised not to place any undue reliance on such forward-looking statements. These forward-looking statements speak only as of the date of publication of this document, argenx undertakes no obligation to publicly update or revise the information in this press release, including any forward-looking statements, except as may be required by law.