PRESS RELEASE

Affimed Presents Data on Innate Cell Engagers AFM24 and AFM28 at 19th Meeting of the Society for Natural Immunity

- Correlative science data from AFM24-101 clinical study support the rationale to develop AFM24 as monotherapy and in combination therapies
- First preclinical data set on the combination of AFM28 with natural killer cells, both in co-administration or pre-complexed, demonstrates cytotoxic activity against CD123-positive tumor cells

Heidelberg, Germany, May 16, 2022 – Affimed N.V. (Nasdaq: AFMD) ("Affimed", or the "Company"), a clinical-stage immuno-oncology company committed to giving patients back their innate ability to fight cancer, has announced the presentation of new data on AFM24 and AFM28 in two posters at the 19th Meeting of the Society for Natural Immunity (NK2022).

The AFM24 presentation showed correlative science data of the exposure and pharmacodynamic effects of the compound in patients with epidermal growth factor receptor (EGFR)-expressing solid tumors from the ongoing phase 1/2a study. The poster featured an analysis of the longitudinal effects of AFM24, a CD16A/EGFR-targeting bispecific innate cell engager (ICE®), in patients treated in the AFM24-101 phase 1/2a clinical study, confirming the mechanism of action of AFM24 on the innate immune system.

The correlative science data further supports the rationale for combining different therapeutic approaches in patients with EGFR-expressing solid tumors. AFM24 engages CD16A on natural killer (NK) cells and macrophages with higher affinity than monoclonal antibodies, and triggers antibody-dependent cellular cytotoxicity (ADCC) and antibody-dependent cellular phagocytosis (ADCP), respectively, directed at EGFR-expressing cancer cells. Preclinical data have shown that AFM24 can induce NK cell-mediated killing of EGFR-positive solid tumor cell lines, independent of EGFR mutational status.

The analysis also showed activation of cytotoxic T cells in the periphery, and infiltration of T cells into the tumor bed, suggesting stimulation of anti-cancer immunity beyond the innate immune system and the possible engagement of the adaptive immune system. These data support the rationale for AFM24 as monotherapy and the two combinations that are currently under way in separate phase 1/2a studies – with autologous NK cell therapy and with immune checkpoint inhibition.

The AFM28 poster featured preclinical data on the anti-leukemic activity of the compound when pre-complexed and co-administered with allogeneic NK cells.

AFM28 is a novel ICE® binding to CD16A on NK cells, and CD123 on acute myeloid leukemia (AML) and myelodysplastic syndrome (MDS) tumor cells. The novel bispecific engager binds with high affinity to NK cells stimulating them to destroy CD123-positive tumor cells via ADCC. In addition, AFM28 exhibits greater cell surface retention than conventional monoclonal antibodies, including Fc-enhanced IgG1.

Furthermore, the data presented at the conference demonstrate that AFM28 stimulates lysis of CD123-positive tumor cells in both formats, pre-complexed or when co-administered with NK cells. The poster also demonstrated the feasibility of cryopreserving AFM28 pre-complexed with NK cells whilst maintaining anti-tumor activity suggesting the promise for an off-the-shelf therapy targeting leukemic blasts and leukemic stem cells in patients with AML and MDS.

Poster details:

Title: Analysis of the Longitudinal Effects of AFM24, a CD16A/Epidermal Growth Factor Receptor-Targeting (EGFR) Bispecific Innate Cell Engager, Confirms the Mechanism of Action and Supports the Rationale for Combination Approaches in Patients with EGFR-Expressing Solid Tumors

Authors: Gabriele Hintzen, Susanne Wingert, Michael Emig, Kerstin Pietzko, Uwe Reusch, Melissa M. Berrien-Elliott, Todd A. Fehniger, Mark Foster, Paolo Nuciforo, Tyler Burns, Paulien Ravenstijn, Stefan Knackmuss, Bettina Rehbein, Joachim Koch, Arndt Schottelius, and Erich Rajkovic

Title: Novel Bispecific Innate Cell Engager AFM28 in Combination with Allogeneic NK Cells for the Treatment of CD123^{+} Acute Myeloid Leukemia and Myelodysplastic Syndrome

Authors: Jens Pahl, Jana-Julia Siegler, Armin Beez, Rebecca Hussong, Sabrina Purr, Lena Wagner, Nicole Schulze, Tatjana Kosbar, Uwe Reusch, Joachim Koch, Arndt Schottelius, Thorsten Ross, Christian Merz and Sheena Pinto

About AFM24

AFM24 is a tetravalent, bispecific innate cell engager (ICE®) that activates the innate immune system by binding to CD16A on innate immune cells and EGFR, a protein widely expressed on solid tumors, to kill cancer cells. Generated by Affimed's fit-for-purpose ROCK® platform, AFM24 represents a distinctive mechanism of action that uses EGFR as a docking site to engage innate immune cells for tumor cell killing through antibody-dependent cellular cytotoxicity and antibody-dependent cellular phagocytosis.

Affimed is evaluating AFM24 in patients with advanced EGFR-expressing solid malignancies whose disease has progressed after treatment with previous anticancer therapies as monotherapy and in combinations with other cancer treatments. AFM24-101, a monotherapy, first-in-human phase 1/2a open-label, is a non-randomized, multi-center, multiple ascending dose escalation and expansion study. Additional details may be found at www.clinicaltrials.gov using the identifier NCT04259450. Furthermore, AFM24 is being evaluated in a phase 1/2a study in combination with Roche's anti-PD-L1 checkpoint inhibitor atezolizumab (AFM24-102, NCT05109442). Affimed and NKGen Biotech have initiated a phase 1/2a study (AFM24-103), investigating AFM24 in combination with SNK01, NKGen Biotech's NK cell product (NCT05099549).

About AFM28

AFM28 is a tetravalent, bispecific innate cell engager (ICE®) that activates the innate immune system by binding to CD16A on innate immune cells and CD123-positive cells on myeloid malignancies.

Developed on Affimed's ROCK® platform, it is designed to bring a new immunotherapeutic approach to patients with CD123-positive myeloid malignancies, including acute myeloid leukemia and myelodysplastic syndrome (MDS). AFM28 engages NK cells to initiate tumor cell killing via antibody-dependent cellular cytotoxicity (ADCC), and binds CD123-positive cancer cells even at low expression levels.

Clinical development is planned as both monotherapy and in combination with allogeneic NK cells in patients with relapsed/refractory CD123-positive leukemias.

About Affimed N.V.

Affimed (Nasdaq: AFMD) is a clinical-stage immuno-oncology company committed to give patients back their innate ability to fight cancer by actualizing the untapped potential of the innate immune system. The Company's proprietary ROCK® platform enables a tumor-targeted approach to recognize and kill a range of hematologic and solid tumors, enabling a broad pipeline of wholly-owned and partnered single agent and combination therapy programs.

The ROCK® platform predictably generates customized innate cell engager (ICE®) molecules, which use patients' immune cells to destroy tumor cells. This innovative approach enabled Affimed to become the first company with a clinical-stage ICE®. Headquartered in Heidelberg, Germany, with offices in New York, NY, Affimed is led by an experienced team of biotechnology and pharmaceutical leaders united by a bold vision to stop cancer from ever derailing patients' lives. For more about the Company's people, pipeline and partners, please visit: www.affimed.com.

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