UNITED STATES SECURITIES AND EXCHANGE COMMISSION Washington, D.C. 20549

FORM 6-K

Report of Foreign Private Issuer Pursuant to Rule 13a-16 or 15d-16 of the Securities Exchange Act of 1934

For the month of May, 2015

Commission File Number: 001-36619

Affimed N.V.

Im Neuenheimer Feld 582, 69120 Heidelberg, Germany (Address of principal executive offices)

Indicate by check mark whether the registrant files or will file annual reports under cover of Form 20-F or Form 40-F.

Form 20-F ⊠ Form 40-F □

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(1): 🗆

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(7): 🗆

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized, in Heidelberg, Germany, May 29, 2015.

AFFIMED N.V.

By: /s/ Adi Hoess

Name:	Adi Hoess
Title:	Chief Executive Officer

<u>By:</u> /s/ Florian Fischer

Name:Florian FischerTitle:Chief Financial Officer

EXHIBIT INDEX

Exhibit	Description of Exhibit
1	Affimed N.V. Press Release dated May 29, 2015 Announcing ASCO Data for the CD33/CD3-TandAb Program
2	Affimed N.V. Press Release dated May 29, 2015 Announcing ASCO Data Demonstrating Consistent Potency of NK-Cell-Engaging Combination Therapy with Checkpoint Modulators
	Combination Therapy with Checkpoint Modulators



FOR IMMEDIATE RELEASE

Affimed ASCO Data for the CD33/CD3-TandAb Program Demonstrate that CD33 and CD3 binding affinities correlate with potent T-cell activation and cytotoxicity

-- TandAb platform enables reliable and rapid candidate selection --

Heidelberg, Germany, May 29, 2015 - Affimed N.V. (Nasdaq: AFMD), a clinical stage biopharmaceutical company focused on discovering and developing highly targeted cancer immunotherapies based on its proprietary TandAb platform, today announced that first data on AMV-564 (formerly T564), the product candidate currently in IND-enabling studies, from the Company's Amphivena/Janssen collaboration will be presented on Saturday, May 30, and Sunday, May 31 at the American Society of Clinical Oncology (ASCO) 2015 Annual Meeting being held May 29 – June 2, 2015 in Chicago, IL.

Data from three posters by Affimed and its partners Amphivena and Janssen on its collaborative CD33/CD3 program for the treatment of acute myeloid leukemia (AML) validates the robustness of Affimed's proprietary TandAb technology platform. Overall, using various combinations of 10 human anti-CD33 variable domains, 4 human anti-CD3 variable domains and different middle linkers, the platform has enabled generating more than 150 unique CD33/CD3 TandAbs for further evaluation.

In Abstract #7071, titled "Development of a bispecific tetravalent CD33/CD3 TandAb for the treatment of AML" (by Amphivena and Affimed, on Sunday, May 31), 22 lead TandAbs were selected based on expression titers, homodimer content, melting temperature, thermal stability, and high-affinity CD33 binding or to preserve diversity of CD33 domain or linker, and subsequently were produced and purified to >90% purity. Notably, bivalent high affinity binding did not elicit significant cytokine release in the absence of CD33+ cells.
Abstract #7067, titled "Construction characterization of novel CD33/CD3 tandem diabodies (TandAbs) for the treatment of acute myeloid leukemia (AML)" (by Dr. Roland Walter of the Fred Hutchinson Cancer Research Center, on Sunday, May 31), demonstrated that CD33/CD3-targeted TandAbs exerted potent and specific cytotoxicity in CD33+ leukemia cells that is independent of disease stage and cytogenetic risk. Moreover, CD33 and CD3 binding affinities correlated with T-cell

activation and cytotoxicity, but no correlation between TandAb-induced specific cytotoxicity and CD33 expression level was observed. Abstract #3057, titled "In vitro *and* in vivo *killing of AML using tetravalent bispecific CD33/CD3 TandAbs*" (by Dr. John DiPersio of Washington University in St. Louis, on Saturday, May 30), showed that TandAbs specifically lysed human CD33+ target cell lines, but not human cells lacking the antigen, at concentrations as low as 0.001 and 1pM. Also, preclinical mouse data revealed that even though very few patient T cells (2%-4%) may be present, TandAbs could still clear all AML blasts.

"The power of Affimed's TandAb technology is evident in its ability to deliver a candidate rapidly – with the TandAb technology platform, it is possible to file an IND within 3 years," said Martin Treder, CSO of Affimed. "Immune cell engagers redirecting NK- or T-cells possess significant promise in eliminating tumor cells. Importantly, to maximize response rates, it is critical that even those tumor cells with very low target expression can be efficiently eliminated. These posters show that affinity to T-cells is highly relevant in the destruction of primary cancer cells derived from AML patients and that TandAbs with high affinity to T-cells demonstrate superior *in vitro* cytotoxicity as compared to those with lower affinity to T-cells."

TandAbs are currently the only 4-domain bispecific immune cells engagers worldwide that are in clinical investigations. This four domain (tetravalent) antibody structure is the underlying scaffold that enables generation of molecules with a binding affinity that can exploit the so-called avidity effect, a principle that relies on dual binding to cell surface antigens, to more effectively destroy tumor cells.

About NK-Cell TandAbs, T-Cell TandAbs and Trispecific Abs

Affimed develops TandAbs and Trispecific Abs to substantially increase the efficacy, specificity and/or extend the therapeutic window of current therapeutics. TandAbs and Trispecific Abs are a new generation of proprietary, tumor-cell engaging antibodies with a tetravalent architecture characterized by four binding domains. These tetravalent molecules bind to tumor and immune cells with high affinity. Although generation of such complex antibodies is very challenging, Affimed has succeeded in producing them economically and at high quality.

Leveraging this expertise, Affimed has implemented three platform technologies:

- · Bispecific TandAbs engaging NK-cells (via CD16A)
- Bispecific TandAbs engaging T-cells (via CD3)
- · Trispecific Abs engaging either NK- or T- cells

About Affimed N.V.

Affimed (Nasdaq: AFMD) is a clinical-stage biopharmaceutical company focused on discovering and developing highly targeted cancer immunotherapies. Affimed's product candidates are being developed in the field of immuno-oncology, which represents an innovative approach to cancer treatment that seeks to harness the body's own immune defenses to fight tumor cells. The most potent cells of the human defense arsenal are types of white blood cells called natural killer cells, or NK-cells, and T-cells. Affimed's proprietary, next-generation bispecific antibodies, called TandAbs for their tandem antibody structure, are designed to direct and establish a bridge between either NK-cells or T-cells and cancer cells, triggering a signal cascade that leads to the destruction of cancer cells. Affimed has focused its research and development efforts on three proprietary TandAb programs for which it retains global commercial rights. For more information, please visit <u>www.affimed.com</u>.

FORWARD-LOOKING STATEMENTS

This press release contains forward-looking statements. All statements other than statements of historical fact are forward-looking statements, which are often indicated by terms such as "anticipate," "believe," "could," "estimate," "expect," "goal," "intend," "look forward to", "may," "plan," "potential," "predict," "project," "should," "will," "would" and similar expressions. Forward-looking statements are based on management's beliefs and assumptions and on information available to management only as of the date of this press release. These forward-looking statements include, but are not limited to, statements regarding the risk of cessation or delay of any of the ongoing or planned clinical studies and/or development of our product candidates. Our actual results could differ materially from those anticipated in these forward-looking statements for many reasons, including, without limitation, risks associated with our clinical development activities, regulatory oversight, product commercialization, intellectual property claims, and the risks, uncertainties and other factors described under the heading "Risk Factors" in Affimed's filings with the Securities and Exchange Commission. Given these risks, uncertainties and other factors, you should not place undue reliance on these forward-looking statements, and we assume no obligation to update these forward-looking statements, even if new information becomes available in the future.

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FOR IMMEDIATE RELEASE

Affimed ASCO Data Demonstrate Consistent Potency of NK-Cell-Engaging Combination Therapy with Checkpoint Modulators

--Affimed's lead NK-cell engager AFM13 shows highly synergistic efficacy with PD-1 inhibitor--

Heidelberg, Germany, May 29, 2015 - Affimed N.V. (Nasdaq: AFMD), a clinical stage biopharmaceutical company focused on discovering and developing highly targeted cancer immunotherapies, today provided details on preclinical data from a combination study of Affimed's lead candidate AFM13 with checkpoint modulators, including checkpoint inhibitor PD-1. These data will be presented on Saturday, May 30, at the American Society of Clinical Oncology (ASCO) 2015 Annual Meeting being held May 29 – June 2, 2015 in Chicago, IL.

The poster titled "*CD137 co-stimulation and blocking PD-1 enhances NK-cell-mediated target lysis by CD30/CD16A TandAb AFM13*" (Abstract #3050) outlines the results of four preclinical studies developed by Dr. Holbrook Kohrt at Stanford University in Patient-Derived Tumor Graft (PDX) mice to analyze Affimed's AFM13 in combination with checkpoint modulating agents. In this model, which develops actual human tumors, AFM13, a first-in-class natural killer (NK-) cell engager, demonstrated significant synergy in combination with a PD-1 inhibitor. Importantly, these preclinical results were consistent among the four individual studies.

Specifically, tumor samples were surgically obtained from four newly diagnosed CD30+ Hodgkin lymphoma patients. These tumors were xenografted into mice and allowed to grow for 28 days before the mice were infused with the respective patient's peripheral blood mononuclear cells (PBMCs) and treated on a weekly basis with either monotherapy (IgG control, AFM13, anti-CTLA-4, anti-PD-1, or anti-CD137) or a combination regimen (AFM13 plus anti-CTLA-4, anti-PD-1, or anti-CD137). The mice received three weekly treatments via intraperitoneal injection and tumor size was compared between groups on day 58. The mean results for all patients demonstrated:

- · Of all single agents investigated, AFM13 showed the most potent anti-tumor efficacy.
- As single agents, AFM13 and anti-PD-1 were able to demonstrate tumor shrinkage.
- Treatment with the combination of AFM13 with an anti-PD-1 resulted in an impressive decrease in tumor volume, in some cases with complete tumor eradication.
- As a single agent, AFM13 led to substantial tumor-infiltration of NK-cells, which was enhanced by the combination with anti-CTLA-4, anti-CD137, and anti-PD-1.
- Increased number of tumor-infiltrating cytotoxic T-cells, especially with the combination of AFM13 with anti-PD-1 and anti-CTLA-4.

"These striking preclinical results, in particular the combination of AFM13 with a PD-1 inhibitor, along with AFM13's good clinical safety profile in patients, strongly advocate for the further investigation of the combination therapy in hematologic malignancies expressing CD30," stated Dr. Holbrook Kohrt.

Natural killer cells or NK-cells are one of the key drivers of innate immunity. The innate immune system is the body's first line of response to aberrant cells such as cancer cells. Hence, every cancer cell that survives in the human body must initially develop protection mechanisms in order to avoid its destruction by the innate immune cells. Affimed's approach to redirect NK-cells to the tumor through its CD16A bispecific TandAb-platform is aiming to reestablish the killing mechanism by the innate immune system. As the innate immune system is also the gate keeper of the adaptive immune system, namely the T- and B-cells, this redirection and killing of cancer cells may also be crucial to activating the adaptive immune response. Affimed is presently investigating this clinically with AFM13 as a monotherapy and is planning to initiate a clinical study with AFM 13 in combination with a checkpoint inhibitor. AFM13 is the only CD16A NK-cell engager currently in clinical investigations in the world.

About AFM13

AFM13 is a first-in-class bispecific NK-cell TandAb®, which binds NK-cells specifically via CD16A and has a second binding domain for CD30, a cancer-specific target. CD16A is expressed on NK-cells, highly potent cytotoxic effector cells of the innate immune system, enabling AFM13 to selectively bind these effector cells. AFM13 redirects the NK-cells to CD30-expressing cancer cells and binds both targets with high affinity, establishing a bridge whereby the NK-cells are activated and redirected to kill the cancer cells. AFM13 is designed to treat CD30-positive malignancies including Hodgkin lymphoma (HL) and T-cell lymphoma (TCL) and is currently in phase 2 studies in HL patients. Like all TandAbs®, AFM13 is a stable, off-the-shelf, targeted immunotherapeutic which does not require continuous infusion due to a favorable half-life in a patient's bloodstream, yet is tunable by dosing adjustment when required. This highly specific NK-cell antibody and the related bispecific platform are unique to Affimed.

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