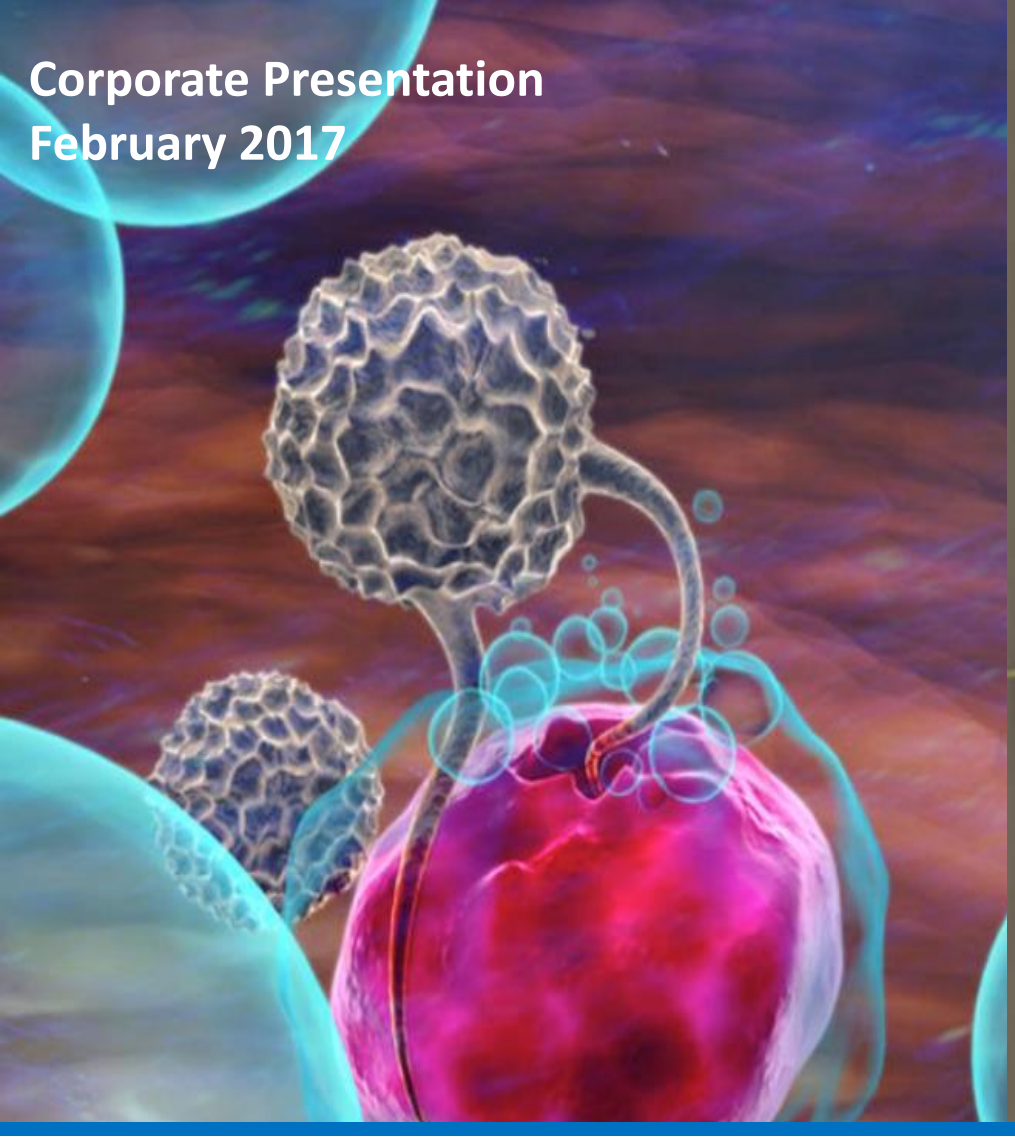
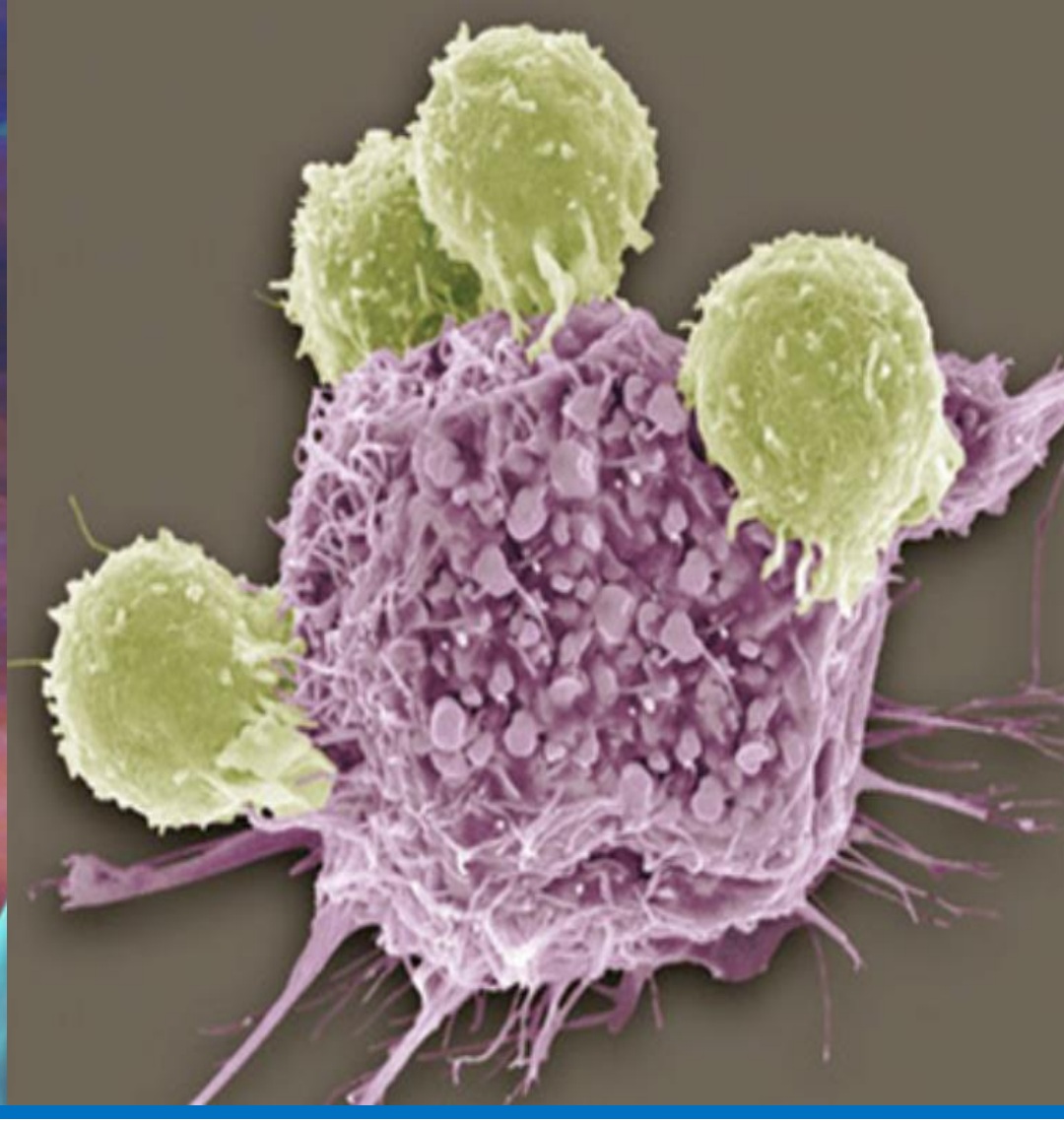
Corporate Presentation
February 2017





affimed

Transforming Immuno-Oncology Using Next-Generation Immune Cell Engagers

Forward-looking statements / safe harbor

This presentation and the accompanying oral commentary contain "forward-looking" statements that involve substantial risks and uncertainties. All statements other than statements of historical facts contained in this presentation and the accompanying oral commentary, including statements regarding our future financial condition, business strategy and plans and objectives of management for future operations, are forward-looking statements. In some cases, you can identify forward-looking statements by terminology such as "believe," "will," "may," "estimate," "continue," "anticipate," "intend," "should," "plan," "might," "approximately," "expect," "predict," "could," "potentially" or the negative of these terms or other similar expressions. Forward-looking statements appear in a number of places throughout this presentation and the accompanying oral commentary and include statements regarding our intentions, beliefs, projections, outlook, analyses and current expectations concerning, among other things, our ongoing and planned preclinical development and clinical trials, our collaborations and development of our products in combination with other therapies, the timing of and our ability to make regulatory filings and obtain and maintain regulatory approvals for our product candidates our intellectual property position, our collaboration activities, our ability to develop commercial functions, expectations regarding clinical trial data, our results of operations, cash needs, financial condition, liquidity, prospects, future transactions, growth and strategies, the industry in which we operate, the trends that may affect the industry or us and the risks uncertainties and other factors described under the heading "Risk Factors" in Affimed's filings with the Securities and Exchange Commission.

Forward-looking statements involve known and unknown risks, uncertainties, assumptions and other factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements.

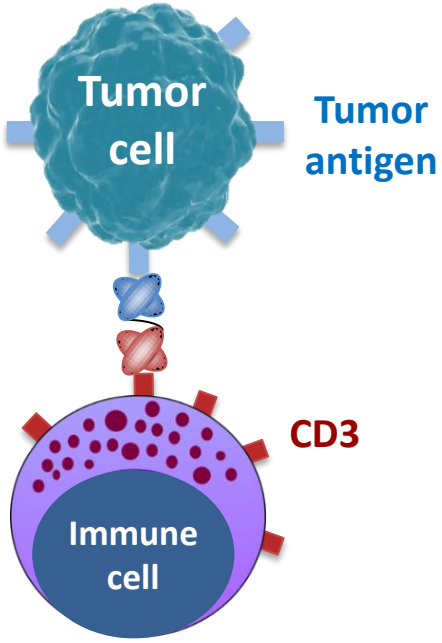
Forward-looking statements represent our management's beliefs and assumptions only as of the date of this presentation. Except as required by law, we assume no obligation to update these forward-looking statements publicly, or to update the reasons why actual results could differ materially from those anticipated in the forward-looking statements, even if new information becomes available in the future.

History of cutting-edge science, innovation, and expertise

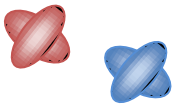
- Clinical and pre-clinical pipeline based on bi- and trispecific antibodies
- Eliminate tumor cells by recruiting NK-cells or T-cells
- Tetravalent bispecific antibody formats
- AFM13, most advanced NK-cell engager in clinical development, with solid Phase 1 data
- Strong preclinical rationale for combination of NK-cell engager with anti-PD-1 antibodies
- Partnerships with industry, academic, and advocacy groups (incl. Merck, MDACC, LLS)

Affimed's tetravalent bispecific antibody formats

Most competitors
Bivalent

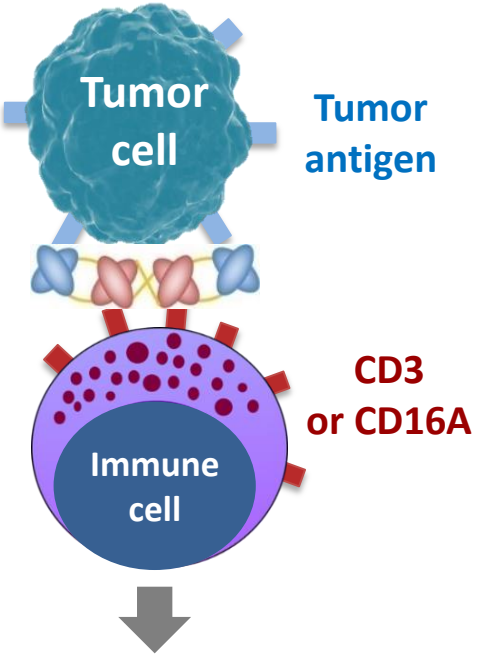


Antibody binding domains



Assembly

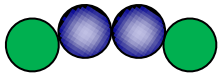
AFMD
Tetravalent

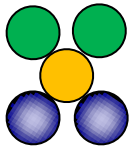


Avidity
High specificity
Trispecific
Tailored PK

AFMD has multiple formats for NK- or T-cell engagement

- **AAF are based on tetravalent and bispecific structures aimed at tailoring dosing regimes (both NK-cell and T-cell platform)**





	TandAb	AAFs
Potency	Low pM	Low pM
Affinity	pM	pM
Stability	High at 37°C	High at 37°C
Expression/Yield	High	Very high
Safety	Excellent	Excellent*
Serum PK	1-2 days	~1 week**

* based on *in vitro* data

** based on comparator data

Current pipeline and programs

	Compound	Disease Target	Immune Cell Target	Indication	Discovery	Preclinical	Phase 1	Phase 2	Collab.	Partners
NK-cell engagers	AFM13	CD30	CD16A	Hodgkin Lymphoma Combination with PD-1					MERCK	
				Hodgkin Lymphoma					someday is today; GHSG	
				Hodgkin Lymphoma Combination with active NK-cells					MD Anderson Cancer Center	
				CD30+ Lymphoma incl. TCL						
	AFM24	EGFRwt	CD16A	Solid Tumors incl. Lung, Head & Neck, and Colon Cancer						
	AFM26	BCMA	CD16A	Multiple Myeloma						
	Trispecific Abs	BCMA/CD200 BCMA/XX	CD16A	Multiple Myeloma						
T-cell engagers	AFM11	CD19	CD3	Non-Hodgkin Lymphoma						
				Acute Lymphocytic Leukemia						
	AMV564	CD33	CD3	Acute Myeloid Leukemia						AMPHIVENA Therapeutics *
	N.N.	MHC-peptide complexes	CD3	Undisclosed						

Completed

Ongoing/in preparation

Partnered program

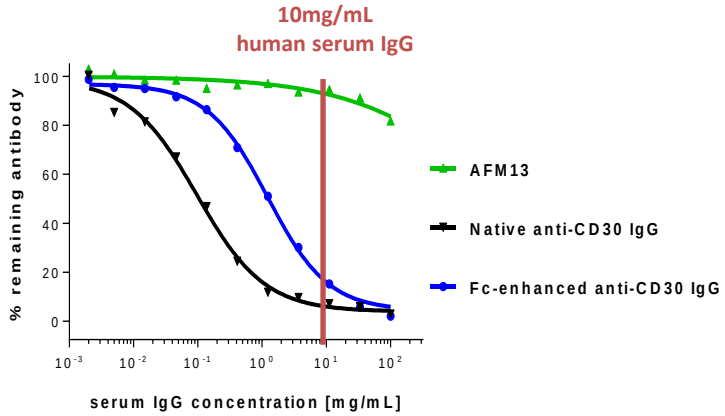
* Affimed with >20% equity ownership

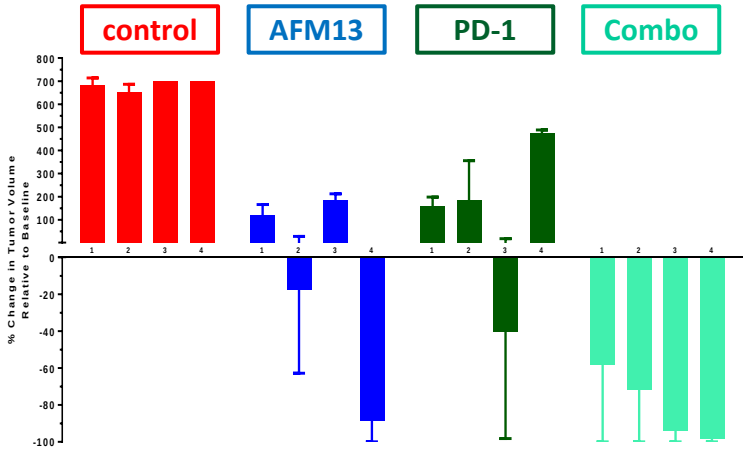
Leader in engaging innate and adaptive immune systems through NK-cells and macrophages

- Achieving more potent and deeper responses
  - CD16A is a dominant activating receptor on innate immune cells
  - Enhancing crosstalk with adaptive immune cells
  - Synergy with checkpoint modulators, such as PD-1
  - Further combination opportunities to enhance efficacy, such as with cytokines
- Safety profile differentiated from T-cell engagement: Lower toxicity
  - Potentially better treatment choice for elderly patients in hem/onc (e.g. MM or AML)
  - Could position NK-cell engagement as leading platform in solid tumor indications
- Strongly differentiated from regular IgG or Fc-enhanced IgG antibodies
  - High affinity binding to CD16A (>1000 fold improvement vs. IgG)
  - Addressing efficacy issues of IgG (competition with circulating IgG, polymorphism)

AFM13: A first-in-class CD16A-targeting NK-cell engager

- Most advanced NK-cell engager in clinical development
- More potent than IgG-based CD30 antibodies - higher affinity to CD16A, limited competition with circulating IgG
- Clinical/PD activity in heavily pretreated HL patients
- Tumor shrinkage in 62% and PRs in 23% patients treated with just 4 weekly doses of at least ≥ 1.5 mg/kg
- Favorable safety profile, offering opportunities for combination with wide range of other drugs
- Synergistic when used in combination with PD-1 suggesting crosstalk between innate/adaptive immunity (PDX model)

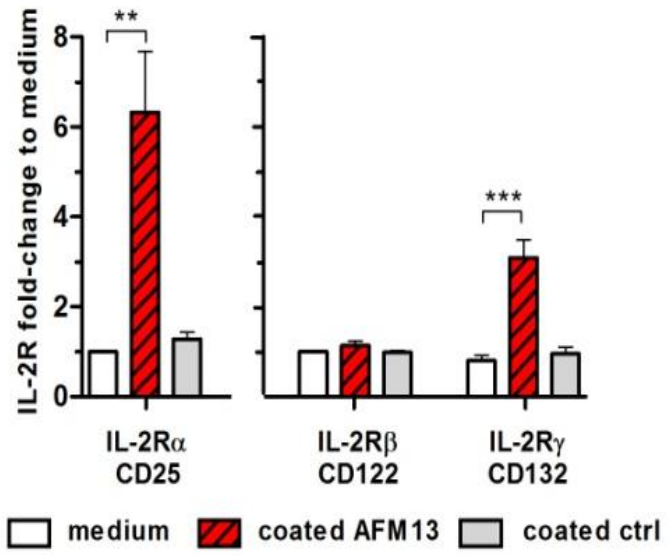




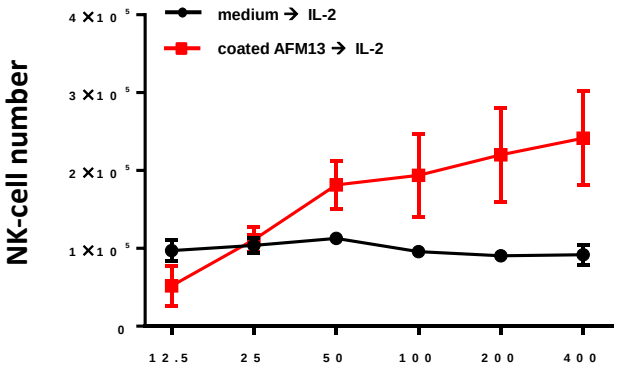
Affimed´s NK-cell antibody technology sensitizes NK-cells to IL2 and IL15 stimulation

- CD16A-mediated activation with AFM13 amplifies IL-2 and IL-15-mediated NK-cell proliferation
- Opportunity for rational combination between Affimed´s NK-cell engager and clinical stage IL-15

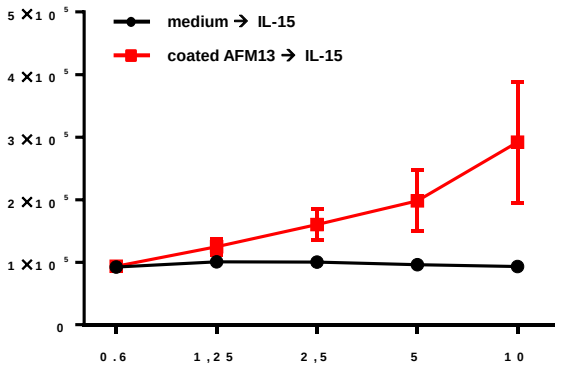
IL-2R expression



IL-2



IL-15



AFM13: Development update

- Phase 1b in r/r HL in combination with Merck's Keytruda® (pembrolizumab)
  - 2nd dose cohort fully recruited; 1st cohort determined safe with efficacy within expectations
  - Update to be provided during next earnings call
- Phase 2a IST in r/r HL led by the German Hodgkin Study Group (GHSG)
  - Study design amended with new inclusion criteria (B.V. and anti-PD-1 r/r HL)
- Additional indications: Expansion into TCL or ALCL
- Additional combinations being investigated:
  - AFM13 in combination with adoptive NK-cell transfer (preclinical and clinical collaboration with MD Anderson Cancer Center)
  - AFM13 in combination with cytokines (synergy determined preclinically with IL-2 and IL-15 resulting in NK-cell expansion through receptor upregulation on the NK-cells)

AFM24: Affimed's first-in-class NK-cell engager targeting solid tumors

- Unmet need in EGFR-positive tumors such as lung, H&N, colon cancers, etc.
- Some tissues (e.g. lung) have prevalent tissue-resident NK-cells
- AFM24 (EGFR/CD16A) is differentiated from cetuximab
  - More potent cytotoxic activity *in vitro* and *in vivo*
  - Tumor cell killing including cells expressing the proto-oncogene *ras*
  - Virtually no competition of NK-cell binding by circulating IgG
- PD-1 / PD-L1 antibodies were recently approved in a variety of cancers including cancers with high EGFR expression (e.g. NSCLC or SCCHN)
- Rationale for combination of AFM24 with PD-1/PD-L1 antibodies in NSCLC or SCCHN
- Development update: IND-enabling toxicology studies ongoing, update planned for H1/2017

AFM26: Affimed's novel candidate for multiple myeloma targeting BCMA

- **Therapeutic rationale**
  - Current treatments fail to achieve MRD negativity in majority of multiple myeloma (MM) patients; most patients eventually relapse
  - MM is characterized by high M-protein serum levels (up to 170mg/mL)
  - Competition by serum IgG is known to strongly impair ADCC activity of mAbs

- **AFM26 (BCMA/CD16A) introduces a novel MoA**
  - NK-cell binding of candidates largely unaffected by circulating IgG, indicating potential for NK-cell activation in the presence of M-protein
  - High affinity to target and NK-cells leads to prolonged cell retention
  - High cytotoxic activity towards BCMA-expressing myeloma cell lines
  - Potential positioning as 1st line in combo with adoptive NK-cell transfer during ASCT or in salvage
  - Likely safer than T-cell-based approaches, allowing for faster development timelines

T-cell engagement: Every indication/target currently presents a different challenge

- NHL and ALL
  - Several CD19-approaches (including CAR-Ts and BiTEs) have shown high efficacy
  - All have similar side effects with different degrees of severity (CAR-T >> BiTE)
  - CD19-Fc-DART/ibrutinib combination with suspended enrollment
  - CD20/CD3-IgG data with moderate efficacy and highly variable inter-patient PK
- AML
  - No solid ORR data yet reported for CD33- or CD123-T-cell targeting CD3 bispecifics
  - Program based on full-length bispecific antibody currently with suspended enrollment
- MM
  - BCMA CAR-T from BLUE with signs of efficacy and reasonable safety, other CAR-T approaches with CNS side effects
  - No data yet available for BCMA- or CD38-targeting bispecific antibodies

T-cell engagement: Differentiation from other therapies

- AFM11 (CD19/CD3) offers a significant opportunity
  - High unmet need remains in DLBCL and MCL despite recent CAR-T data
  - Addresses limitations of other drugs have (e.g. CIV administration or low efficacy);
  - Has shown a 40-fold higher potency compared to a CD19/CD3 BiTE at low T-cell numbers
  - Phase 1 dose escalation of AFM11 in ALL and NHL patients ongoing
  - Next progress update in H1/2017
- AMV564 (CD33/CD3) is derived from Affimed's platform
  - High unmet need in AML, with a very low 5-year survival rate of about 26%
  - Competitive programs at early stage, however, AMV564 is well differentiated (potent and selective cytotoxic activity and robust tumor growth inhibition)
  - IND approval in July 2016, Amphivena plans to initiate a Phase 1 study
- Undisclosed program in MM
- Platform targeting MHC-peptide complexes

Key corporate milestones

	Compound	Disease Target	Immune Cell Target	Indication	Discovery	Preclinical	Phase 1	Phase 2	Collabor./ Partner	Key Corporate Milestones
NK-cell engagers	AFM13	CD30	CD16A	Hodgkin Lymphoma Combination with PD-1					MERCK	Update 1H/17 Update 2H/17
				Hodgkin Lymphoma					someday is today, GHSG	Update 2H/17
				Hodgkin Lymphoma Combination with active NK-cells					MD Anderson Cancer Center	Update 1H/17
				CD30+ Lymphoma incl. TCL						Update 2H/17
	AFM24	EGFRwt	CD16A	Solid Tumors incl. Lung, Head & Neck, and Colon Cancer						Update 1H/17
	AFM26	BCMA	CD16A	Multiple Myeloma						Update 2H/17
	Trispecific Abs	BCMA/CD200 BCMA/XX	CD16A	Multiple Myeloma						
T-cell engagers	AFM11	CD19	CD3	Non-Hodgkin Lymphoma						Update 1H/17
				Acute Lymphocytic Leukemia						Update 1H/17
	AMV564	CD33	CD3	Acute Myeloid Leukemia					AMPHIVENA Therapeutics *	
	N.N.	MHC-peptide complexes	CD3	Undisclosed						

Completed | Ongoing/in preparation | Partnered program

* Affimed with >20% equity ownership

Q3 2016 Cash Flow statement

In thousands of €	For the nine months ended September 30, 2016
Cash and Cash equivalents beginning of period	76,740
FX-related changes to Cash and Cash equivalents	(655)
Net cash used in operating activities	(25.546)
Cash used in investing activities	(13,767)
Cash and Cash equivalents end of period	35,693
Financial assets* end of period	13,440
Cash and cash equivalents and financial assets* end of period	**49,133**

* short-term deposits

- **Raised ~17m€ in follow-on financing in Q1/17**
- **Runway through Q4/2018**

Path forward
Maximize value from pipeline and technologies

- Expand NK-cell engagement leadership
  - Develop AFM13 (CD30/CD16a) in combination with Keytruda and as monotherapy in r/r HL
  - Advance AFM24 (EGFRwt/CD16A) in solid tumors (incl. lung, head and neck, and colon cancer) and AFM26 (BCMA/CD16A) in multiple myeloma
  - Combine NK-cell engagers with adoptive NK-cell therapy (MDACC) or cytokines (e.g. IL-15)
- Focus on DLBCL, MCL and AML in T-cell engagement
  - Generate PoC for T-cell-engaging TandAbs with AFM11 (CD19/CD3)
  - Amphivena plans to initiate Phase 1 for AMV564 (CD33/CD3)
- Expand platforms (AAF, targeting of MHC-peptide complexes)
- Use pipeline and technologies to create value through both next-generation products and partnership opportunities

Backup

Anti-HLA-A2/peptide T- & NK-cell antibodies show highly-specific killing of endogenous tumor cells

- Candidates identified which specifically recognize target MHC-peptide but not control MHC-peptides

- Candidates
  - Specific killing of endogenous tumor cells only
  - Killing by both, T- and NK-cell engager platform molecules
  - Excellent biophysical properties

			T-cell		NK-cell	
	TandAb:		T714	Positive control	T802	Positive control
cell line	HLA-A2	MMP1	Mean EC_{50} [pM]		Mean EC_{50} [pM]	
B-CPAP	+	+	236.6	3.8	1690	4.2
SW-982	+	+	155.9	98.1	2861	7.3
MCF-7	+	-	no	1.6	n.t.	n.t.
KMS-27	+	-	no	0.6	no	17,8
COLO-205	+	-	no	1.8	n.t.	n.t.
JVM-2	+	-	no	1.4	no	16.6
ES-2	-	+	no	47.0	no	5.8
BXPC-3	-	+	no	1.5	no	3.1
RPMI-8226	-	-	no	0.5	n.t.	n.t.
MM.1S	-	-	no	0.2	no	4.1
KARPAS-299	-	-	no	40.8	no	20.7
NCI-H929	-	-	no	10.7	n.t.	n.t.
HeLa	-	-	no	4.8	n.t.	n.t.

n.t.: not tested