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Company Report

December 10, 2015

Affimed Therapeutics N.V. (AFMD - $ 6.87)

Healthcare/Biotechnology

A Pure Play of NK- and T-cell Engaging Bi- and Tri-specific Antibody Developer

Ticker:	**AFMD**
Rating:	**Buy**
Price Target:	**$ 15.00**

We are initiating coverage of Affimed Therapeutics with a Buy rating and a $15 12-month price target. By leveraging its TandAb platform, AFMD has developed several novel NK and T cell engaging bispecific antibodies as potential treatment for both Hodgkin's and Non-Hodgkin's lymphoma.

- **NK- and T cell engaging TandAb generates improved next generation bi- and tri-specific antibodies.** After the approval and continued development of blincyto, effector cells (CTL or NK) engaging bispecific antibodies have emerged as a potentially powerful treatment modality. Supported by substantial improvements, such as enhanced affinity and improved dosing potential, AFMD's TandAb platform could become the engine creating next generation effector cells engaging bispecific antibodies.
- **Positive Phase II study results could potentially validate the NK cell engaging platform as a versatile treatment modality.** AFM13 is a first-in-class NK cell engaging bispecific antibody with potential for treating $CD30^+$ B cell malignancies, by binding CD30 and CD16. Supported by encouraging Phase I study results, AFM13 in HL and $CD30^+$ lymphoma Phase II trial results could be available in 2016/17. An AFM13/CPI combination clinical study could start in 1H16 with results possibly in 2017. Multiple data releases over the next 18 months, we believe, could fully validate AFM13's potential in treating B cell malignancies under various circumstances.
- **AFM11 could be a materially improved CD19 targeted bispecific antibody.** With well recognized improvements over blincyto, AFM11, we believe, could be a more advanced second generation T cell engaging bispecific antibody to treat NHL as well as ALL. AFM11 in ALL and NHL Phase I studies are already underway or about to start, with top-line results expected in 2H16.
- **Additional pipeline and partnerships.** They include a pre-clinical EGFRvIII targeted program (AFM22/21) for solid tumors, and a Johnson & Johnson partnered CD33/CD3 bispecific antibody for AML. Developments are all in progress.
- **Material upsides remain at the current valuation.** With two leading differentiated bispecific antibodies in place, we believe AFMD shares remain undervalued at current levels. Our 12-month $15 price target is based on peer comparable, probability adjusted DCF and sum-of-the-parts analyses.

Trading Data:

Last Price (12/09/2015)	$ 6.87
52–Week High (7/17/2015)	$ 24.20
52–Week Low (12/17/2014)	$ 5.14
Market Cap. (MM)	$ 228
Shares Out. (MM)	33

Earnings Estimates: (€ per share)

(Dec)	1Q	2Q	3Q	4Q	FY	P/E
FY-15E	-0.06A	-0.19A	-0.24A	-0.11	-0.60	N.A.
FY-14A	-1.06	0.03	0.37	0.32	-0.01	N.A.
FY-13A	NA	NA	NA	NA	-1.76	N.A.
FY-12A	NA	NA	NA	NA	-0.97	N.A.

Source: Laidlaw & Company estimates

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Investment Thesis

Our $15 price target is supported by peer comparable, probability adjusted DCF and sum-of-the-parts analyses.

- **We are initiating coverage of Affimed Therapeutics (AFMD) with a Buy rating and a 12-month price target of $15.** Affimed Therapeutics is a mid-clinical stage biotechnology company focusing on the development of NK and T cell engaging bi- and tri-specific antibodies as a potential treatment for hematological and solid tumors. AFMD is a bi- and tri-specific antibody pure play and a leader in developing NK-cell engaging antibodies. Given the successful clinical proof-of-concept demonstrated by the approved blincyto, the potential of engaging NK and T cells in eradicating tumor cells, and the potentially acceptable safety profile; we believe NK and T cells engaging antibodies could likely become part of the emerging immune-oncology (IO) cancer treatment armamentarium.

AFM13 in r/r HL monotherapy Phase IIa trial is underway with interim and top-line results expected in 1H16 and 2H16, respectively. A Phase IIa trial in CD30$^+$ lymphomas is scheduled to start in late 2015. An AFM13/CPI (possibly PD-1) combination therapy Phase I trial in HL is scheduled to start in 1H16.

- **By targeting CD30 via engaging NK cells (through CD16), AFM13 could potentially be a novel treatment for r/r Hodgkin's lymphoma and other CD30 expressing hematological cancers.** AFM13 is AFMD's lead NK cell engaging bi-specific antibody that could have the potential for treating relapsed/refractory Hodgkin's Lymphoma (HL) and other CD30$^+$ lymphomas, such as diffuse large B-cell lymphoma (DLBCL) and anaplastic large cell lymphoma (ALCL). By binding to CD16a specifically but not to CD16b, AFM13 is highly specific in engaging NK cells (without effects from neutrophils) as an effector cell in killing targeted cancer cells. An earlier Phase I study (n=26) that evaluated AFM13 in heavily pretreated r/r HL patients demonstrated promising results as AFM13 is safe and well tolerated and MTD level was not reached. Further, the study exhibited an ORR of 12% and DCR (disease control rate) of 62% for all doses tested. Encouraging results from the highest dose tested (>1.5 mg/kg, n=13) exhibited 23% ORR and 77% DCR. Bi-specific antibody inducing NK cell cytotoxicity alone as an effective cancer treatment modality still needs more clinical validation. Multiple studies have suggested that an activated NK cell under the "right" circumstance could elicit additional cellular immunity, like CTL, to create a concerted anti-tumor action.

We anticipate 2016 and 2017 would be critical for AFMD share value; based on several clinical outcomes from mono- and combination studies to provide greater visibility of how AFM13 could be used in the rapidly moving IO cancer treatment landscape in HL and other CD30$^+$ lymphomas.

After the recently positive pre-clinical results reported at the 2015 ASCO conference, AFMD is scheduled to start an AFM13/checkpoint inhibitor (CPI) combination clinical study to explore its potential in future IO treatment landscape. An AFM13 in r/r HL monotherapy Phase IIa trial is underway with interim and top-line results expected in 1H16 and 2H16, respectively. The primary endpoint is ORR after three months treatment and secondary endpoint is PFS. A Phase IIa trial in CD30$^+$ lymphomas is scheduled to start in late 2015. Further, an AFM13/CPI (possibly PD-1) combination therapy Phase I trial in HL is scheduled to start in 1H16. Together, we anticipate that 2016 and 2017 will be critical for AFMD share value; based on several clinical

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outcomes from mono- and combination studies to provide greater visibility of how AFM13 could be used in the rapidly moving IO cancer treatment landscape in HL and other $CD30^+$ lymphomas.

- **AFM11 could become a materially improved therapy vs. blincyto and potential treatment for other CD19 expressing NHLs.** AFM11 is a second lead product with the potential as a best-in-class cytotoxic T cell engaging bi-specific antibody comparing to the marketed blincyto (blinatumomab) as treatment for Acute Lymphoblastic Leukemia (ALL) and other relevant B-cell malignancies (i.e. NHL). Blincyto is approved in the U.S. and Europe as a treatment for Philadelphia chromosome-negative (Ph-) relapsed or refractory B-cell ALL. CMS in 2Q15 announced an increase of Medicare payment to $178,000 for the standard course of blincyto treatment. It was a reversal of an earlier lower payment decision, and it is a testament to the effectiveness of blincyto treatment.

AFMD initiated an AFM11 in r/r $CD19^+$ NHL and plans to start the ALL Phase I study in 1Q16. The top-line results could be available in in 2H16 with more updates in 2017.

In comparison to blincyto, AFM11 could have the following advantages: 1) potential for less frequent administration; 2) greater potency; and 3) manufacturing flexibility. Mainly due to its larger molecular weight (104kDa vs 55kDa), AFM11 has a substantially longer half-life ($t_{1/2}$) in plasma than blincyto and, therefore, enables the drug to be administrated via a regular periodical intravenous infusion. Compared to blincyto, AFM11 demonstrated greater cytotoxic potency (40x) in eliminating cancer cells, and exhibited greater complete tumor cell lysis (death) at low T-cell counts. AFM11 demonstrated a 100x affinity vs. blincyto (1 nM vs. 100 nM). Since AFM11 could be produced via both prokaryotic and eukaryotic systems with high yields, it could potentially account for modest COGS advantage over blincyto.

AFMD initiated an AFM11 in r/r $CD19^+$ NHL and ALL Phase I study and recently amended the protocol to a less frequent dosing from the prior intensive dosing. The top-line results could be available in 2H16 with more updates in 2017. The primary endpoint is safety and tolerability in a single cycle of treatment; while secondary endpoints include MTD or OBD (optimal biological dose), PK and tumor response. AFMD is also starting the AFM11 in ALL (possibly mainly in adult) trial as a separate Phase I trial with commencement expected in 1Q16.

- **TandAb platform is a novel next generation bi- and tri-specific antibody anti-cancer technology that could target both natural killer (NK) and T cells.** AFMD's proprietary antibody development platform, TandAbs or tandem diabodies, can develop cytotoxic T lymphocytes or natural killer (NK) cells engaging bi- and tri-specific antibodies to destroy cancer cells. The TandAbs is comprised of four variable domain fragment (Fv) regions derived from two separated antibodies that each bind to a specific antigen. AFMD is the leading developer of NK cells engaging bi- and tri-specific antibodies.

AFMD is in the process of evaluating both programs (T and NK cell engaging) to determine which version as the final anti-EGFRvIII candidate (likely in 1H16) before advancing it to pre-clinical IND-enabling study in 2016 and possible Phase I trial in 2017.

- **Additional pipeline products with potential for treating solid tumors and partnerships should validate TandAbs platform.** In addition to the two leading products, AFMD's third un-partnered and pre-clinical stage product target EGFRvIII (Epidermal growth factor receptor variant III) with two versions as a potential treatment of solid

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tumors: NK cells engaging AFM21 via 16A, and CTLs engaging AFM22 via CD3. AFMD is in the process of evaluating both programs to designate one version as the final anti- EGFRvIII candidate (likely in 1H16) before advancing it to the pre-clinical IND-enabling study in 2016 and possible Phase I trial in 2017. EGFRvIIIs (mutant versions of EGFR) are prominently expressed in many solid tumors. AFMD entered a license deal with partially owned Amphivena Therapeutics (~28%) to develop CD33/CD3 bispecific antibody for hematologic malignancies. AFMD is entitled to an interest in Amphivena and certain milestone payments (first €7.5M received in 1Q15). Amphivena also entered into an agreement with Janssen Biotech, part of of Johnson & Johnson. Janssen has an option to buy Amphivena upon IND acceptance by the FDA. This bispecific antibody is currently in preclinical development with CMC and toxicology work is underway. CD33 is a member of the sialic acid-binding immunoglobulin-like lectins (Siglecs) and is expressed in a subset of blasts in nearly all AML patients and in some AML stem cells.

CD33/CD3 bispecific antibody for hematologic malignancies is currently in preclinical development. Janssen has an option to buy Amphivena upon IND acceptance by the FDA.

- **Valuation is favorable.** We believe AFMD shares are undervalued, based on the substantial near term potential of several data-driven positive critical catalysts in 2016 and beyond. The key value proposition is to clinically validate the first-in-class NK bi-specific antibody and potential optimal use of NK and T cell bi-specific antibodies in future cancer treatment. Accordingly, our $15 price target is supported by peer comparable, probability adjusted DCF and sum-of-the-parts analyses. We are recommending AFMD shares to long-term oriented investors with high risk tolerance.

We believe the key value proposition of AFMD share value is to clinically validate the first-in-class NK bi-specific antibody, and potential optimal use of NK and T cell bi-specific antibodies in future cancer treatment.

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Company Description

Affimed Therapeutics is a mid-clinical stage biotech company focused on exploring its proprietary TandAb platform to develop bi- and tri-specific antibodies that could engage NK cells or T cells as an anti-cancer treatment modality. The company currently has two lead products in development: NK cell targeted **AFM13** is intended to treat Hodgkin's lymphoma (initially as salvage treatment for Adcetris r/r patients) and other $CD30^+$ lymphoma. **AFM11** targets CD19 and engages T-cells via CD3 to enable the drug to potentially become a substantially improved therapy vs. recently approved blincyto; as treatment in multiple types of B-cell malignancies, such as NHL and ALL. The company already started an AFM13 monotherapy Phase IIa study in r/r Hodgkin's lymphoma with interim and preliminary results available in 1H16 and 2H16, respectively. A Phase IIa trial in $CD30^+$ lymphomas is scheduled to start in late 2015, and an AFM13 and immune checkpoint inhibitor combined Phase I study is scheduled to start in 1H16 after the promising pre-clinical study results reported at the ASCO and ASH annual meeting in 2015. AFMD has started AFM11 in NHL and ALL dose escalating Phase I study with critical interim results potentially available in 2016.

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Anticipated milestones in 2015 and beyond

Product	Indication	Event	Timing	Importance
AFM13	Hodgkin's lymphoma (r/r)	Commence pre-clinical data of lenalidomide combination study (Mayo Clinic)	**4Q15**	***
		Potentially report Phase IIa study interim results	**1H16**	****
		Potentially start a Phase I CPI combination Phase I study	**1H16**	***
		Potentially report Phase IIa study preliminlary top-line results	**2H16**	****
		Potentially report Phase I CPI combination Phase I study results	**Late 2016/2017**	****
	CD30+ lymphoma	Potentially commence Phase IIa study	**4Q15**	***
AFM11	Non-Hodgkin's lymphoma (NHL)	Potentially start Phase I study	**4Q15**	***
		Potentially report Phase I study interium results	**2H16**	****
	Acute lymphoblastic leukemia (ALL)	Potentially start Phase I study	**1Q16**	***
		Potentially report Phase I study interium results	**2H16**	****
AFM21/22	Solid tumors	Potentially select clinical candidate and IND enabling study	**1H16**	***
		Potentially start Phase I study	**Mid-17**	***

***** / ***** Major catalyst event that could impact share price very significantly while *** event is more informative*

Source: Laidlaw & Company and company presentation.

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Affimed Pipeline

Product	Indication	Preclin	I	II	III	Comments
AFM13	Hodgkin's lymphoma (r/r)					Phase II first part data expected in 1H16
						PD-1 combination Phase I trial expect to start in 1H16
						Combination with Lenalidomide pre-clinical study ongoing
	CD30+ lymphoma					Phase IIa trial to start in 1H16
AFM11	Non-Hodgkin's lymphoma (NHL)					Phase I study updates expected in 2H16
	Acute lymphocytic leukemia (ALL)					Phase I trial to start in 1H16
AFM21	Solid tumors					
CD33/CD3 bispecific antibody	Acute myeloid leukemia (AML)					Janssen might acquire Amphivena following IND acceptance by the FDA possibly in 2016

Source: Laidlaw & Company and company presentation

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TandAb Platform Generates the Next Generation Bi- and Tri-Specific Antibodies

By leveraging their TandAb platform, AFMD develops tetravalent, bi- and tri-specific antibodies to engage cytotoxic effector (NK or T) cells to eliminate cancer cells

TandAbs (tandem diabodies) is Affimed's proprietary antibody development platform that generates effector cells [cytotoxic T lymphocytes or CTLs or natural killer (NK) cells] engaging bi- and tri-specific antibodies to destroy cancer cells.

The major advantage of effector cell engaging bi-specific antibody is it could fully leverage effector cells' cytotoxic capability, while circumventing most of cellular regulatory, particularly the negative, machineries.

By bringing a specific cancer cell directly to a close proximity of effector cells and enabling the latter to destroy the former, the effector cell engaging bispecific antibody has emerged as a treatment modality for cancer, with potential for other indications, such as inflammatory disorders. The major advantage of this treatment modality is it could fully leverage effector cells' cytotoxic capability, while circumventing most of the cellular regulatory, particularly the negative, machineries. Effector cell engaging bispecific antibody is now a clinically validated treatment modality after the recent approval of Amgen's blincyto (blinatumomab). Blincyto is developed via BiTE technology (bispecific T cell engager); and the drug is approved as a treatment of Philadelphia chromosome-negative (Ph-) relapsed or refractory B-cell precursor acute lymphoblastic leukemia (ALL). Supported by robust clinical data, blincyto has been granted a breakthrough therapy designation and received approval only two and half months after BLA filing (via Priority review designation). Amgen is also actively conducting multiple clinical studies evaluating blincyto in several hematological malignancies and in combination with PD-1 inhibitor (Keytruda from Merck) to further explore the drug's potential.

Effector cell engaging bispecific antibody is clinically validated treatment modality after the recent approval of Amgen's blincyto (blinatumomab).

Despite blincyto's impressive clinical performance, we believe there is still room for improvement of the first generation effector cell engaging bispecific antibody platform. Examples include their short plasma half-life resulting in need for administrating the drug by continuous infusion (via a portable pump). Affimed's TandAb platform has the advantages of generating an improved and more versatile method to create both T and NK cell engaging bispecific antibodies.

The TandAbs is comprised of four variable domain fragment (Fv) regions derived from two separated antibodies that each binds to a specific antigen (Figure 1). Fv region consists of the target specific binding site which is

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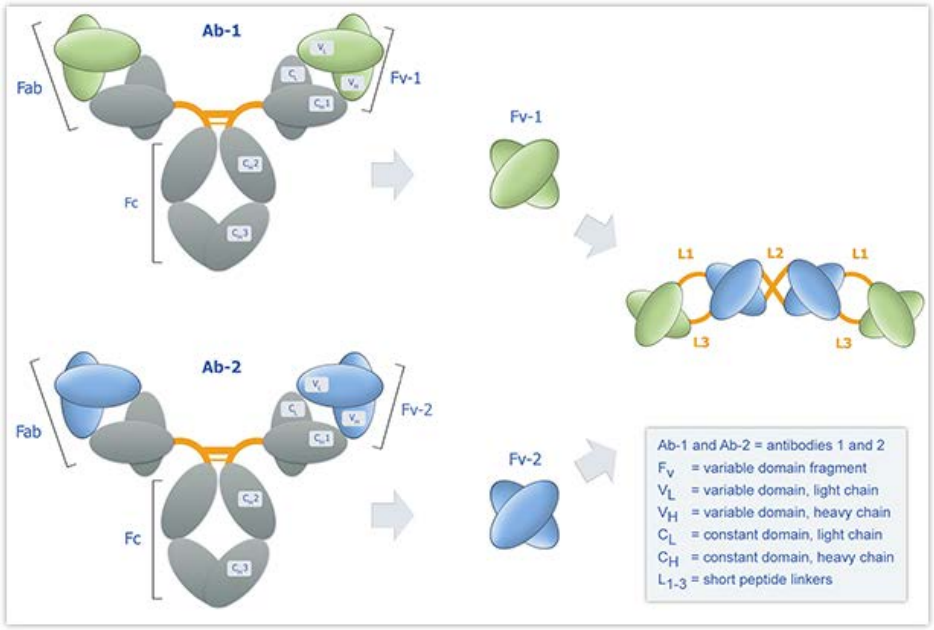
comprised of a heavy and light chain from the initial antibody. The four Fv domain fragments of a TandAb are connected by short peptide linkers. TandAbs are initially expressed from a single gene construct, and two chains are spontaneously assembled as a biologically active homodimer. Each TandAb contains four binding domains (tetravalent) with two for each molecular target.

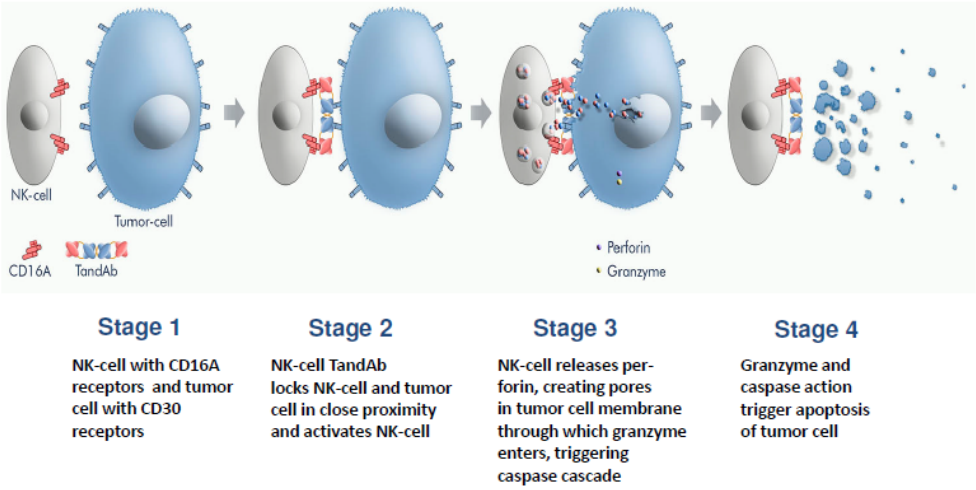
Figure 1:The construction of TandAbs



Source: Company presentation

The process of effector cell engagement for destroying cancer cells is depicted in Figure 2 using NK cells as an example. Basically, a NK cell engaging bispecific antibody is to mediate cancer cell destruction similar to the process of naturally occurring antibody-dependent cell-mediated cytotoxicity (ADCC).

Figure 2: The mechanism of bispeicific antibodies that engage cytotoxic effector cells



Source: Company presentation

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ADCC recap. As part of innate immune response, NK cells, macrophages and neutrophils can carry out ADCC as part of cell-mediated immune defenses against infection and to much less extent, cancer cells. After a cell is infected with a pathogen, certain molecules from the pathogen could re-appear on the cell surface after these molecules are processed by the cell. Afterward, antibodies that recognize such antigens could later bind to these surface antigens or "coat" this cell. Subsequently, effector cells, such as NK cells via its surface CD16 or FcgIIIA (Fc receptors) will bind to the Fc regions of native full-length antibodies which already "coated" the infected cells. NK cells then will release granzyme and caspase cascade to lyse the target cell.

Potential benefits of TandAb platform. One of the major distinctions between TandAb and other similar effector cell engaging bispecific antibody platforms is that it has developed the first-in class NK cell engaging antibody as well as the T cell engaging antibody. In addition, TandAb platform also has the following advantages over its competitors:

TandAb platform also have the following advantages over its competitors: manufacturing advantages, improved binding affinity, and enable regular intravenous delivery.

- **Manufacturing advantages:** Given TandAb production methods are versatile and simpler; either prokaryotic or eukaryotic systems for production can be used. The production costs, therefore, could be materially lower than that of other competitors, and also with high production yield (97%).
- **Improved binding affinity.** By a combined phage and yeast display antibody library and a proprietary algorithm, AFMD has designed TandAb, such as AFM11 with high affinity against CD3 (100X vs. blinatumomab); as well as building two CD3 binding sites. Together, AFM11 exhibited greater cytotoxic potency in eliminating cancer cells. Clinically, it could translate to better efficacy, particularly for patients with lower levels of T cells.
- **Enable regular intravenous delivery.** Bispecific antibodies of smaller molecular weight, such as blincyto (55 kDa), will require continuous infusion due to their short half-life ($t_{1/2}$) in plasma. The molecular weight (104 kDa) of TandAb antibodies are much closer to the size of a regular IgG (160 kDa), and larger than other competitors (Figure 3). The $t_{1/2}$ is substantially longer in plasma and, therefore, enables them to be administrated via a regular periodical intravenous infusion.

Figure 3: The molecular weight of different bispecifc antibodies

Source: Adapted from Spiess, C., et. al.,2015 Mol. Immunology

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Different types of products developed via TandAb platform. The TandAb platform has created several types of bi-specific antibodies that engage either T or NK cells; and a tri-specific antibody that recognizes two different cancer cell antigens in addition to effector cell antigens (Figure 4).

Figure 4: Two currently in development TandAb programs and trispecific Ab

Source: Company presentation

It is noted that AFMD is the only developer of the NK cell engaging bispecific antibody. The potential of developing a bispecific antibody that could engage either T or NK cells affords AFMD the flexibility to design the most appropriate bispecific antibody configuration to best treat a particular indication.

Activated NK cells could have a better safety profile than cytotoxic T cell with less release of cytokines

NK cells vs. T cells as effector cells in a context of bispecific antibody therapy. Compared to cytotoxic T cells, NK cells could potentially have some advantages. According to AFMD management, as a targeted cell for executing cytotoxic activities, NK cells could have a better safety profile than cytotoxic T cells. Specifically, after activation, T cells could proliferate and release various cytokines, such as IFN-γ, TNF-α, and TNF-β – all could potentially have a greater safety impact than activated NK cells. The more extreme scenario caused by massive activated cytotoxic T cells is the presence of cytokine release syndrome (CRS), which is due to rapid and massive release of cytokines (such as IL-6 and interferon γ) into the bloodstream, resulting in dangerously high fevers and precipitous drops in blood pressure which could be life-threatening. CRS is a major issue that needs to be resolved in some chimeric antigen receptor T-cell (CAR-T) developments.

After engaging NK cells by bispecific antibody in destroying cancer cells, the debris released from lysis cells could potentially stimulate growth of additional T cells to attack cancer cells via regulatory adaptive immunity.

In addition, after engaging NK cells with a bispecific antibody in destroying cancer cells, the debris released from lysed cells could potentially stimulate growth of additional T cells (possibly both $CD4^+$ and $CD8^+$) to attack cancer cells via regulatory adaptive immunity. Specifically, the cellular components from the destroyed cancer cells might be processed by Antigen Presenting Cells (APCs) and generate T cell immunity after APCs present the MHC class I complex to CTLs. Such an effect could potentially become more pronounced if in the presence of a checkpoint inhibitor (CPI). T cell activation by a T cell engaging bispecific antibody alone might not have the same synergistic effect.

A trispecific antibody can recognize two different cancer cell antigens besides the effector cell antigen. A potential benefit is to create high affinity and increase selectivity for malignant tissues compared to healthy tissues by binding

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to more than one target on cancer cells (Figure 5). AFMD plans to develop trispecific antibody as a potential multiple myeloma treatment. The trispecific antibody development is currently supported by a €2.4MM ($3MM) research grant from the German government.

Figure 5: Trispecific antibody increases affinity and selectivity

X-Y-CD3 Trispecific Ab	X single positive cell line	Y single positive cell line	X-Y double positive cell line 1	X-Y double positive cell line 2
CYTOTOXICITY/ EC_{50} [pM]	378	247	38	26

Source: Company presentation

Solid intellectual properties support TandAb and each specific product. AFMD has established a broad intellectual property protection for its TandAb platform and each specific product pending patent applications. The family of patents for engineered antibody format, TandAb, which is comprised of the methods of making or using such bispecific, tetravalent domain antibodies will expire in 2019.

For AFM13, an additional patent family for the use of the specific target combination for the treatment of cancer will expire in 2020. A pending patent family that covers AFM13's mode of action for the recruitment of immune effector cells via a specific receptor could expire in 2026 if a patent is issued. For AFM11, several issued patents could expire in 2019, while several pending patients, if issued, could expire in 2030.

Bi-specific antibody competitors. The competitors for an effector cell engaging bispecific antibody remains limited, and here are several selected developers:

- **Amgen** Clinically, it is the most advanced developer with its BiTE-based bispecific antibodies. Blincyto is approved and also is under several late stage clinical studies. Amgen acquired all BiTE antibody assets after it acquired Micromet in 1Q12 for $1.06 billion. Another BiTE antibody in development includes AMG 211 or MEDI 565 (CD3/CEA), which is in Phase I and is co-owned by Amgen and MedImmune of AstraZeneca. The clinical status of two other BiTE antibodies, AMG330 (CD3 / CD33) and solitomab or AMG 110 (CD3/ EpCAM) is not clear.
- **Macrogenics** has two bispecific antibodies, MGD006 (CD3/CD123) and MGD007 (CD3/gpA33), and both are developed via MGNX's DART platform. Both products are in Phase I studies and partnered with Servier. MGD006 is indicated in AML, while MGD007 is in CRC. Judging from their similar molecular weight to that of BiTE antibodies (Figure 3), we believe the DART bispecific antibodies are likely to have a short plasma half-life and might require frequent dosing.

A broadly defined bi-specific antibody could further divided into two different groups: effector cell engaging antibody and dual target antibody.

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- **Zymeworks** is a Vancouver, Canada-based privately owned biotech company that leverages its proprietary Azymetric platform, to develop bispecific antibodies via IgG1 based heterodimeric antibody scaffold. The company's ZW38 is a CD19 and CD3 binding T cell engaging bispecific antibody with potential in treating CLL, refractory ALL and NHL with projected IND fining in 2016.

Bi-specific antibodies are increasingly becoming a potential treatment modality not only for cancer, but also other inflammatory indications, such as psoriasis and rheumatoid arthritis. A broadly defined bi-specific antibody could be further divided into two different groups: effector cell engaging antibody and dual target antibody. Figure 6 illustrates a more comprehensive list of bi-specific antibodies currently under clinical development.

Figure 6: Selected bi-specific antibodies in clinical development

Product	Developer	Targets	Status	Indications
Effector cell engaging antibody				
Blinaumomab (blincyto)	Amgen	CD19/CD3	Approved	ALL
IMCgp100	Immunocore	gp100/CD3	Phase II	Melanoma
AFM11	Affimed	CD19/CD3	Phase I/ II	NHL, ALL
AFM13	Affimed	CD16/CD30	Phase I/ II	HL
Ertumaxomab	Trion	Her2/CD3 and FcR	Phase I	Solid tumor
MEDI-565	MedImmune	CEA/CD3	Phase I	GI adenocarcinoma
MGD006	MacroGenics	CD123/CD3	Phase I	AML
MGD007	MacroGenics	CD33/CD3	Phase I	CRC
Dual target antibody				
XmAb5871	Xencor	CD19/CD32B	Phase I/ II	RA
LY3164530	Eli Lilly	MET/EGFR	Phase I	Advanced cancers
ALX-0761	Ablynx	IL17A/IL-17E	Phase I	Psoriasis
COVA322	Covagen/Johnson	TNF/IL-17A	Phase I/ II	Psoriasis
RG7221 (vanucizumab)	Roche	ANG2/VEGFA	Phase II	CRC
SAR156597	Sanofi	IL-4/IL-3	Phase II	IPF
ABT-981	AbbVie	IL-1a/IL-41b	Phase II	Osteoarthritis
ABT-122	AbbVie	TNF/IL-17	Phase II	RA
MEHD7945A	Genentech	HER3/EGFR	Phase II	CRC

Source: Laidlaw and Co. Equity research and company presentation

TandAb antibodies are well differentiated from other immune-oncology (IO) platforms. Given their mechanism of action is engaging immune-machinery to eradicate cancer cells, bi-specific antibodies can be categorized as part of immuno-oncology (IO) therapies. The IO approach could refer to anti-cancer therapy that generally boosts the active (in few occasions, the passive) immunity to eliminate cancer cells. In practice, some of the approaches are to create novel platforms that circumvent the regulatory mechanism; while others are by removing/boosting the negative/positive regulatory factors to afford the body's regular immune machinery to eliminate cancer cells. IO could be further divided into six groups: checkpoint inhibitors; adoptive cell therapies (ACT), which include chimeric antigen receptor T-cell (CAR-T); effector cell engaging

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antibodies; immune cytokine booster, therapeutic vaccines and miscellaneous therapies:

- ***Checkpoint inhibitors.*** Checkpoint inhibitor refers to molecules (mainly antibodies) that inhibit "immunological brakes" (checkpoints) of immune cells, such as T and NK cells. Checkpoint activation could avoid the immune system overreacting against healthy cells. Many tumors have taken advantage of boosting checkpoint activities, resulting in diminishing the effectiveness of the immune system, such as T cells, in attacking cancer cells. Checkpoint inhibitors against CTLA-4 (Yervoy – melanoma) and PD-1 (Keytruda and Opdivo, both for melanoma/non-small cell lung cancer) are two approved drugs for cancer therapy. We also anticipate more in-development checkpoint inhibitors to gain approval and cover more cancers going forward. In addition to T cells, there are also checkpoint inhibitors against NK cells in development. In addition to negative regulators, positive regulators are also under development as potential therapeutics for cancer treatment.
- ***Adoptive cell therapies (ACT).*** ACT refers to cancer therapies that use enriched or modified immune cells equipped with anticancer activity as treatment, and CAR-T is the most recognized modality. Chimeric antigen receptor (CARs) is a construct comprised of an antigen recognition fragment that targets tumor associated antigens and an intracellular signaling domain (such as CD28, CD3ζ) in one molecule. CAR-T is built by introducing CARs into the patient's own T cells using standard gene therapy techniques. Following a myeloablation, patients subsequently undergo transfusion for the recipient CAR-T graft. CAR-T binds to the surface tumor antigen directly on the cancer cell and activates the cell lysis cascade via the directly attached CD3ζ chain. For normal cytotoxic T cells, the initiation of the cell killing process occurs when its TCR interacts with the MHC and bound peptide of the target cell. With highly active non-physiologic T cell activation, CAR-T is a rather potent cancer treatment. The most pronounced CAR-T treatment side effect is cytokine release syndrome (CRS), which is due to elevations in cytokines, such as interferon-γ, interleukin (IL)-10 and IL-6. Main symptoms of CRS are high fevers and precipitous drops in blood pressure. Corticosteroids are commonly used for reducing the impact of CRS. In addition to T cells, a similar CAR therapy is also being developed for NK cells. Other ACTs also include tumor infiltrating lymphocytes (TILs) and T cells with an engineered T cell receptor (TCR).
- ***Effector cell engaging antibodies.*** This modality mainly refers to bi- or tri-specific antibodies that circumvent the regulatory machinery and bring cancer cells (via binding to their surface tumor antigen) in direct connection with effector cells (either cytotoxic T cells or natural killer cells) to be eradicated. Blincyto, initially developed by Micromet and later acquired by Amgen, is the first approved (in 4Q14) bispecific

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antibody that targets T cell via binding to CD3 and CD19 expressed in B-cell malignancies. Blincyto was approved for treating Philadelphia chromosome-negative relapsed or refractory B-cell precursor acute lymphoblastic leukemia (ALL). Among biotech companies that develop bi- and tri-specific antibodies, AFMD is considered as a pure play in this space.

- ***Immune cytokine booster.*** In the earlier days, several recombinant cytokines, such as IL-2 were considered as immune system stimulators that could boost the anti-tumor activities by recruiting and enhancing immune cells to fight cancer. More current developments in this category are to deliver cytokines or other immune stimulators via a variation of gene therapy approaches to increase the local, and to some extent, systemic immunity to fight cancer. Examples include several developments that deliver IL-12 directly to cancer cells. In addition, recently approved Imlygic or talimogene laherparepvec (in Oct. 2015) by Amgen also belongs to this category. As a first-in-class oncolytic viral therapy, Imlygic delivers granulocyte-macrophage colony-stimulating factor (GM-CSF) via genetically modified herpes simplex virus type 1. Imlygic is approved as a treatment for the local treatment of unresectable cutaneous, subcutaneous and nodal lesions in patients with melanoma recurrent after initial surgery.
- ***Therapeutic vaccines.*** It has been more than two decades since the initial concept and continued development of cancer vaccines as a potential cancer treatment modality. Almost all of the cancer vaccines have failed clinically and only one (Provenge for prostate cancer) gained approval but with a rather dim commercial outlook. Although we believe there is potential for cancer vaccines, development remains challenging due to the complicities and multiple layers of positive and negative regulation of the immune system. We believe the outlook could improve with the addition of immune boosting regimens at the post-checkpoint inhibitor environment.
- ***Miscellaneous therapies.*** There are several IO therapies that employ different mechanism of action to increase immunity against cancer as a treatment. Examples include CSF1R inhibitors and IDO inhibitors. Signaling pathway driven colony-stimulating factor 1 receptor (CSF1R), if overexpressed on the surface of tumor cells, could recruit tumor-associated macrophages (TAMs) surrounding the tumor microenvironment (Strachan et al., 2013). This leads to a poor prognosis in patients with breast, prostate, ovarian, and cervical cancer. CSF1R inhibitor intends to reduce the number of TAMs in the tumor microenvironment. Indoleamine 2,3-dioxygenase (IDO) is an enzyme that catalyzes the oxidative cleavage of tryptophan. Overexpression of IDO (in tumors and antigen presenting cells) could suppress the proliferation and survival of T cells since tryptophan is required. As such, IDO plays a significant role in facilitating tumors to evade attack from the immune system. IDO inhibitors could potentially alleviate

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tryptophan depletion and promote an immune response against the tumor.

Positive AFM13 in NHL Phase IIa Trial Results Could Validate Therapeutic and Commercial Potential of NK Cell Targeted Therapy

By targeting CD30 and CD16, AFM13 is a novel antibody that engages NK cells as a potential treatment of HL and other CD30 expressing hematological cancers.

AFMD started a Phase IIa study to evaluate AFM13 in r/r Hodgkin's Lymphoma (HL) with interim and top-line results expected in 1H16 and 2H16. A clinical plan to use AFM13 to treat various other CD30+ lymphomas is underway.

AFM13 is a first-in-class NK cells (binding to CD16) engaging bispecific antibody that intends to destroy CD30 expressing cancer cells. AFMD started a Phase IIa study to evaluate AFM13 in relapsed/refractory Hodgkin's Lymphoma (HL) with interim and top-line results expected in 1H16 and 2H16, respectively. Given the rapid changing dynamic of the IO treatment landscape, AFMD is also planning an AFM13 / checkpoint inhibitor (likely to be PD1) combination therapy in a HL setting to potentially leverage the cytotoxicity of both T and NK cells. Further, a clinical plan to use AFM13 to treat various other $CD30^+$ lymphomas, such as diffuse large B-cell lymphoma (DLBCL) and anaplastic large cell lymphoma (ALCL) is underway.

Unique attributes for engaging NK cells by AFM13. Fc receptors (which are divided into Fc gamma RI, RII, and RIII) bind to the Fc portion of IgG antibodies to activate NK cells and initiate the antibody-dependent cell-mediated cytotoxicity (ADCC) process. RIII Fc receptor is also called CD16 with two isoforms: CD16a (FcγRIIIa) and CD16b (FcγRIIIb). The structural differences between the two are only a few amino acids. CD16a is expressed in NK cells while CD16b is mainly expressed in neutrophils. One major benefit of AFM13 in engaging NK cells is it has significantly higher binding affinity against CD16a, which is ~1,000x higher than full-length antibodies and >25x higher than best Fc-optimized versions of antibodies.

AFM13 has substantial higher specificity binding to CD16a (and several genetic variants) and does not cross-react with CD16b.

Further, AFM13 has substantially higher specificity binding to CD16a (and several genetic variants) and does not cross-react with CD16b (Figure 7). The advantage of high specificity is that the cross-activities against CD16b could have potentially negative impacts on the antibody's potency due to the presence of high levels of soluble CD16b in the blood plasma. The plasma soluble CD16b is mainly derived from cleaved CD16b from the daily turnover of apoptotic neutrophils.

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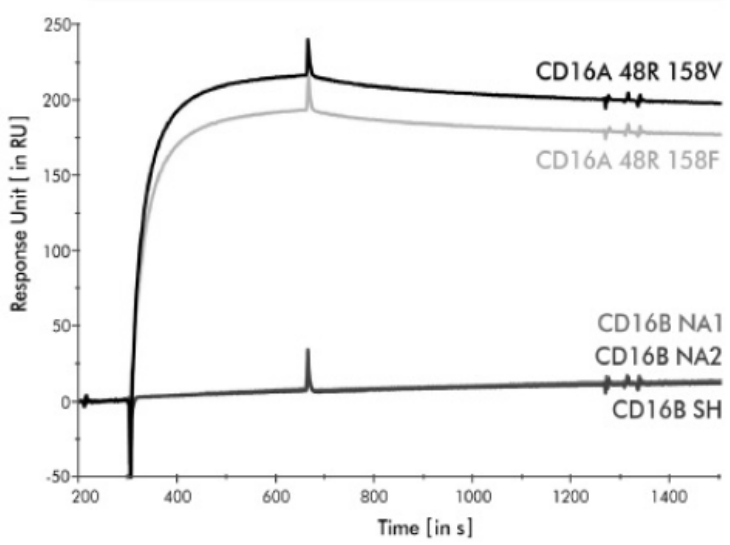
Figure 7: High speific affinity tl but not interaction with neutrophilis



Source: Company presentation

Cytotoxic T lymphocyte (CTL) and NK cells are both immune effector cells that can destroy targeted cells (mainly infected cells) despite their different regulatory mechanisms (Figure 8). The main feature of bispecific antibodies is to engage these cytotoxic effector cells to destroy cancer cells by physically bringing both types of cells in close proximity and circumventing effector cells' regulatory mechanism. Given Amgen's blinatumomab (blincyto) is the only approved (for ALL) bispecific antibody, there are currently more clinical experiences in CTL engaging bispecific antibody. AFMD's AFM13 is the leading bispecific antibody that engages NK cells in cancer treatment.

Figure 8: Major differences between cytotoxic T cells and NK cells

	Cytotoxic T Cells	Natural killer cells
Type of immunity	Adaptive immune	Innate immunity
Antigen specificity	Antigen specific	Antigen non-specific
MHC-restriction	MHC-restricted	MHC non-restricted, less cell killing in MHC class I expressed cells
Receptor type	T cell receptor	Invariant activating and inhibitory receptors
Priming required?	Requires priming (takes days to respond)	Priming not required (rapid response, hours)
Memory	Yes	No
Cytotoxic effectors	Perforins and granzymes	Perforins and granzymes
Repertoire	Thymus	Bone marrow

Source: Laidlaw and Co. Equity research

The initial focus of AFM13 is on Hodgkin's Lymphoma (HL) salvage therapy. Given that in ~50% of relapsed/refractory HL patients are treated by Adcetris, disease progression could occur in ~7 months. There is a substantial unmet need in the salvage treatment setting for these patients. Some of the potential advantages of AFM13 could include:

- Pre-clinical studies demonstrated that AFM13 exhibited a consistently higher cytotoxic potency compared to native and Fc-enhanced anti-CD30 full-length antibodies; and

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- Due to high specific binding to CD16a, but not CD16b; AFM13 is highly selective in binding to NK cells and it does not cross-react with neutrophils.

Promising Phase I results. AFMD from 2Q10 to 2Q13 conducted a dose finding Phase I study evaluating AFM13 in heavily pretreated r/r HL patients. The objective of this study was to determine the safety, PK profile, maximum tolerated dose (MTD), various bio-markers and to assess the activities of single cycles of AFM13 as a monotherapy. The study enrolled 28 patients (14 refractory and four relapsed) in eight dose (0.01, 0.04, 0.15, 0.5, 1.5 4.5 and 7.0 mg/kg) cohorts. Patients had received a median of six (3 to 11) previous lines of therapy, while nine patients had received Adcetris treatment previously. 24 patients received increasing doses of AFM13 (0.01 to 7.0 mg/kg) weekly for four weeks; and four patients were treated with 4.5 mg/kg twice weekly for four weeks.

The study indicated that AFM13 is safe and well tolerated and that the MTD level was not reached. The maximum feasible single dose was 7 mg/kg and without any toxicity concerns. The most common adverse events were fever and chills, and generally were mild to moderate (Figure 9). Severe AEs (SAEs) account for less than 30% of all adverse events.

Figure 9: Most frequent AEs from AFM13 in r/r HL Phase I study

Preferred term	Safety population (n=28)	CTCA grade 1/2	CTCA grade ≥ 3
Pyrexia	15 (53.6%)	14 (50.0%)	1 (3.6%)
Chills	11 (39.3%)	11 (39.3%)	0 (0.0%)
Headache	8 (28.6%)	8 (28.6%)	0 (0.0%)
Nausea	5 (17.9%)	5 (17.9%)	0 (0.0%)
Nasopharyngitis	5 (17.9%)	5 (17.9%)	0 (0.0%)
Vomiting	4 (14.3%)	4 (14.3%)	0 (0.0%)
Pneumonia	4 (14.3%)	0 (0.0%)	4 (14.3%)
Infusion reaction	4 (14.3%)	4 (14.3%)	0 (0.0%)
Rash	4 (14.3%)	4 (14.3%)	0 (0.0%)

Source: Company presentation

From the Phase I study, AFM13 anti-tumor activity was more pronounced at or above 1.5 mg/kg (n=13) with ORR of 23% (3/13) and a DCR (disease control rate) of 77%.

In 26 patient eligible efficacy evaluations, three achieved PR, 13 achieved SD and 10 experienced PD (progressive disease). Anti-tumor activity was more pronounced at or above 1.5 mg/kg (n=13) with 3 who achieved PR and 7 SD with ORR of 23% (3/13) and a DCR (disease control rate) of 77% (Figure 10 and 11).

Figure 10: Clinical results of AFM13 in r/r HL Phase I study

	All doses	≥ 1.5 mg/kg
n (evaluable)	28 (26)	13 (13)
PR	3 (12%)	3 (23%)
SD	13 (50%)	7 (54%)
PD	10 (38%)	3 (23%)
ORR	3 (12%)	3 (23%)
DCR	16 (62%)	10 (77%)

Source: Company presentation and Laidlaw and Co. equity research

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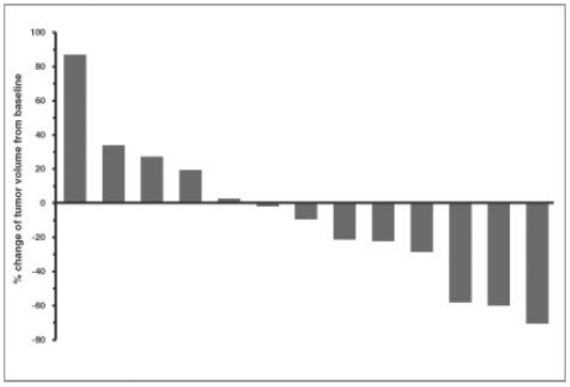
Figure 11: Waterfall plot of ORR of 13 patients received 1.5 mg/kg AFM13



Source: Company presentation

A subset analysis demonstrated that six out of seven AFM13 treated Adcetris refractory patients experienced SD, while one experienced PD (Figure 12).

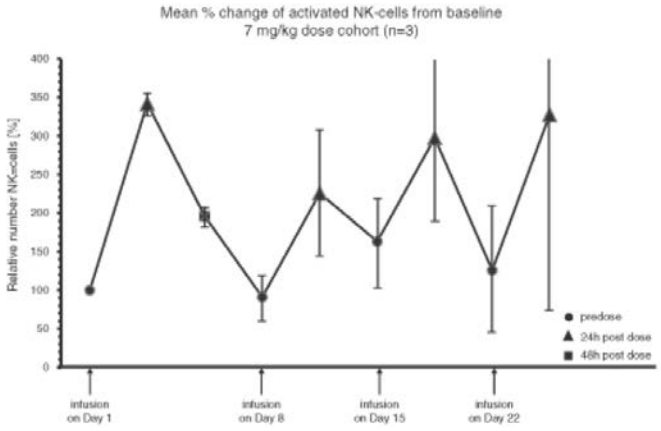
Figure 12: AFM13 treatment respones of patients refractory to Adcertris

PATIENT	AFM13 DOSE (mg/kg)	# PRIOR TREATMENTS	MOST RECENT TREATMENT	TIME LAST ADCETRIS-FIRST AFM13	AFM BEST RESPONSE
001-01	0.01 weekly	6	Adcetris, 5 cycles	1 month	SD
001-02	0.01 weekly	7	Adcetris, 8 cycles	1 month	SD
001-07	0.15 weekly	11	Adcetris, 7 cycles	3 months	SD
001-11	0.5 weekly	7	Adcetris, 5 cycles	3 months	SD
001-12	0.5 weekly	7	Adcetris, 9 cycles	1 month	SD
003-01	0.5 weekly	9	Adcetris, 4 cycles	1.5 months	SD
001-21	4.5 twice	8	Adcetris, 8 cycles	2.5 months	PD

Source: Company presentation

The study illustrated that the plasma mean half-life ($t_{1/2}$) from 1.5 mg/kg cohort was 9 – 19 hours. A measurement of the change of activated CD69 expressing NK cells from three patients who received 7 mg/kg AFM13 correlated with the administration of AFM13 (Figure 13).

Figure 13: The changes of activated NK cells (CD69+) correlate with AFM13 adminstration



Source: Company presentation

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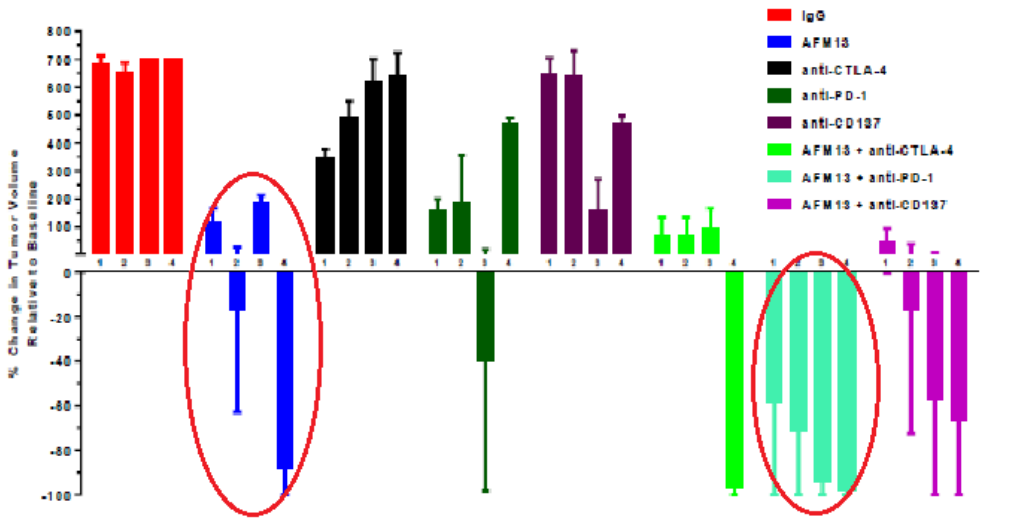
AFM13/CPI combination pre-clinical studies. AFMD has coordinated several physician sponsored pre-clinical studies that evaluate a combination of AFM13 and either checkpoint inhibitors (such as PD-1 or CTLA-4 at Stanford University) or lenalidomide (at Mayo Clinic). At the 2015 ASCO conference, AFMD reported the AFM13 and PD-1 combination afforded enhanced anti-tumor activities, while various combinations could potentially boost immunity from other effector systems judging from the increased tumor infiltrating NK or T cells.

In pre-clinical study, AFM13 and anti-PD-1 or anti-CD137 demonstrated greater anti-tumor activities comparing to AFM13 alone. Anti-CTLA-4, either alone or in combination with AFM13, did not afford much anti-tumor activities.

The pre-clinical analysis indicated that in conjunction with injected autologous human PBMCs in a xenograft tumor mice model, the combination of AFM13 and anti-PD-1 or AFM13 and anti-CD137 demonstrated greater anti-tumor ($CD30^+$ lymphoma, including Hodgkin disease) activities compared to AFM13 alone (Figure 14). Anti-CTLA-4 (another CPI), either alone or in combination with AFM13, did not afford much anti-tumor activities in this model.

Figure 14: AFM13/PD-1 exhibited enhanced anti-tumor activities than AFM13 alone

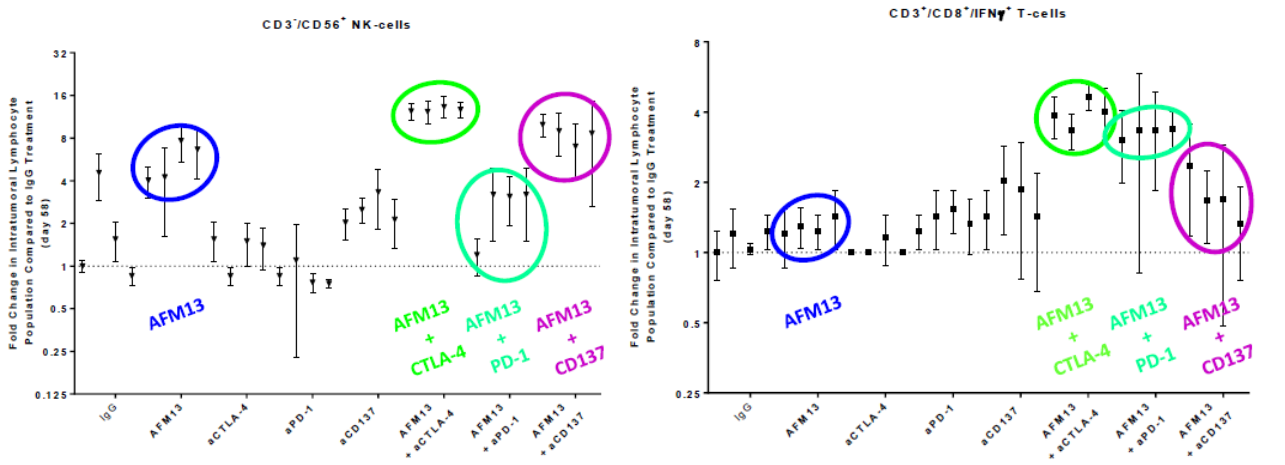


Source: Zhao, X., et. al., 2015 ASCO poster (Abstract#: 3050)

Combinations of AFM13 with either CTLA-4 or CD137 (another potential CPI) have recruited a substantial tumor infiltrating NK cells.

Further, the combinations of AFM13 with either CTLA-4 or CD137 (another potential CPI) have recruited a substantial tumor infiltrating NK cells (Figure 15, left). In addition, the combination with anti-CTLA-4 and anti-PD-1 augmented the number of tumor infiltrating T cells (Figure 15, right), while AFM13 alone did not achieve this goal. Although it might be too early to assess the possible clinical impact, it is, however, possible to speculate that different AFM13/CPI combinations could potentially orchestrate a broader spectrum of immunity to augment anti-tumor potency. The same Stanford researchers also presented a poster at the recent 2015 ASH meeting and re-confirmed that the AFM13 and PD-1 combination not only enhanced anti-tumor activities, but also stimulated T cell infiltration and cytokine release in the tumors. This result is consistent with the theory that an activated innate immunity could trigger the adaptive immune response, resulting in a potentially synergistic therapeutic effect.

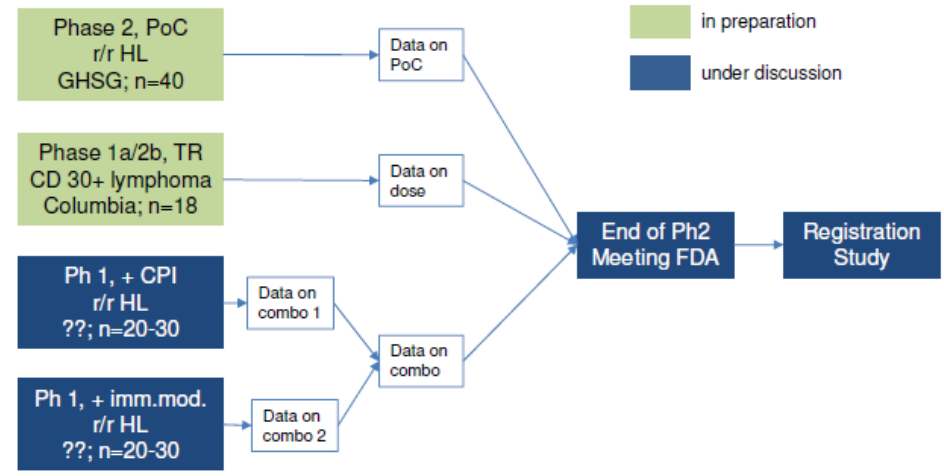
Figure 15: AFM13/CTLA-4 or CD137 and AFM13/CTLA-4 or PD-1 could augment NK and T cells, respectively



Source: Zhao, X., et. al., 2015 ASCO poster (Abstract#: 3050)

AFMD's multiple approaches to explore AFM13. Together, AFMD is exploring multiple approaches to realize the potential value of AFM13 (Figure 16).

Figure 16: AFM13 clinical development strategy



Source: Company presentation

A Phase IIa study that evaluates AFM13 in relapsed/refractory HL patients is underway with top-line results of the first portion of the study expected in 1H16 (n~20) and more data in 2H16. The primary endpoint is ORR after 3 months treatment and secondary endpoint is PFS.

Current and upcoming developments. A Phase IIa study that evaluates AFM13 in relapsed/refractory HL patients is underway (commenced in 3Q15) with top-line results of the first portion of the study expected in 1H16 (n~20) and more data in 2H16. The trial will enroll ~40 Adcetris relapsed/refractory HL patients and will be conducted in Germany. It is an open-label, randomized and multicenter Phase II trial and the study will be divided into two portions. The first part is a dose optimizing trial that examines two regimens with n=10 for each dose. The first regimen is treating with 1.5 mg/kg AFM13, 3x/week for eight weeks, while the second regimen is comprised of two doses of 1.5 mg/kg, 3x/week for two weeks, followed by 7 mg/kg weekly for six weeks. An

optimized regimen will be used to treat patients during the second portion of the study (n=20). A total of eight weeks of therapies are considered as one treatment cycle. Patients are eligible for a second cycle of treatment after four-weeks without treatment and have achieved SD or better. The primary endpoint is ORR after three-months of treatment and the secondary endpoint is PFS (progression free survival). The first portion is completed and the second portion of the study is underway. The Phase IIa study will be co-funded by the Leukemia and Lymphoma Society (LLS) for $4.4MM.

AFMD is scheduled to start a Phase Ib/IIa trial to evaluate AFM13 in CD30+ lymphoma, potentially to start in 4Q15 with top-line results expected in 2017.

Supported by encouraging pre-clinical study results reported at the ASCO and ASH, AFMD is scheduled to start a Phase Ib/IIa trial to evaluate AFM13 in $CD30^+$ lymphoma, potentially to start in 4Q15 with top-line results expected in 2017. The company has not revealed a study design but indicated it will examine multiple biopsies from subcutaneous manifestation of tumors to gain more insights on recruitment of NK-cells, different cytokines and T cell engagement in the tumor microenvironment.

AFMD is planning to evaluate AFM13 in a Phase Ia/IIb trial in various CD30+ lymphomas and we estimate the study will start in 4Q15.

Besides r/r HL, AFMD is also planning to evaluate AFM13 in a Phase Ia/IIb trial in various $CD30^+$ lymphomas (n=18). We estimate the company will start the study in 4Q15. It is estimated that approximately 30% of lymphoma (combined HL and NHL) are $CD30^+$. Examples of $CD30^+$ lymphomas include a substantial percentage of diffuse large B-cell lymphoma (DLBCL), anaplastic large cell lymphoma (ALCL) and cutaneous T-cell lymphoma (CTCL).

Together, Phase IIa POC monotherapy study data in 2016/2017 could be major catalysts for AFMD shareholders and it is possible that AFM13 could enter pivotal clinical studies afterward if mono- and/or combination study results are positive.

Together, Phase IIa POC monotherapy study data in 2016/2017 could be major catalysts of AFMD shareholders

In our opinion, the potential clinical study updates in 2016 for both AFM13 monotherapy (in Adcetris relapsed/refractory HL) and as combination with PD-1 study will be important in assessing: 1) activities as a single agent; and 2) what might be the most optimal configuration (mono vs. combination) for future development. It is noted that despite the earlier reported robust nivolumab (Opdivo) in r/r HL Phase I trial (n=23) results with 87% ORR (20/23 with 4 in CR and 16 in PR), we believe the CR remains modest (17%) and the patient size is small. We present more details in the ensuing section. Should the upcoming Phase II trial results demonstrate material efficacy (for example, an ORR of 30+% with longer duration), AFM13 could remain a very viable treatment option, especially if the CPI combination data are also robust.

Targeting NK cells is gaining attention. Although there are more T cell modulating cancer treatments (i.e., checkpoint inhibitor and CAR-T) in clinical development, the advancement of regulating NK cells as a novel cancer treatment approach recently has gained greater attention (Figure 17). Except for AFMD's AFM13, which circumvent the NK cell regulatory circuitry, most other developments center around: 1) various positive or negative regulatory mechanisms to enable NK cells becoming more active in eliminating cancer

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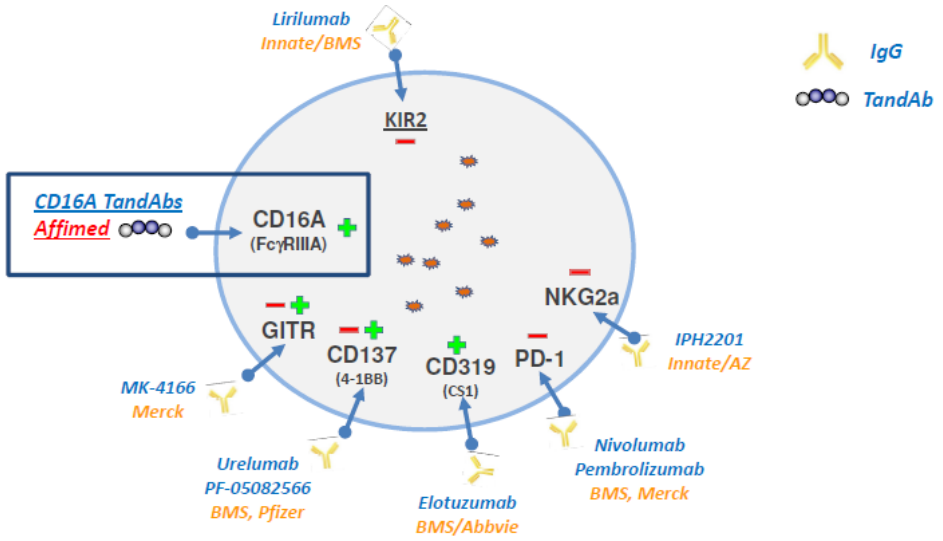
cells; and 2) selected or engineered NK cell lines that possess anti-tumor activities.

Figure 17: Modulating NK cells a recent major approach as cancer treatment



Source: Company presentation

For instance, Innate Pharma / Bristol-Myers Squibb's anti- KIR (or killer-cell immunoglobulin-like receptors) monoclonal antibody, Lirilumab, is currently undergoing a Phase II study (EffiKIR) in acute myeloid leukemia (AML). KIR acts as a checkpoint inhibitor that negatively regulates the immune activation of NK cells. KIR interacts with MHC class I molecules of the targeted cells, resulting in the suppression of NK cell mediated cell killing. In addition, Innate Pharma recently forged collaboration with AstraZeneca to co-develop IPH2201 and the programs are currently in Phase II clinical studies in multiple solid (head and neck) and hematological cancers. IPH2201 targets NKG2A, which is another checkpoint inhibitor that is expressed in NK cells and in 50% of tumor infiltrating T-cells.

NantKwest is developing a NK cell driven cellular immunotherapy platform as a potential cancer treatment. The company's platform has three variations: activated natural killer (aNK) cells, high affinity natural killer (haNK) cells and target activated natural killer (taNK) cells. aNK is an unmodified high potency NK-92 cell line. haNK is NK cells equipped with an engineered high affinity CD16 receptor that enables the cell to bind to antibodies, resulting in generating greater ADCC. taNK is very similar to the concept of CAR-T therapy by incorporating chimeric antigen receptors (CARs) into NK cells and enabling the cell to bind specifically to tumor specific antigens to induce cell death. The early stage clinical data is impressive (mainly from studies of aNK) and could have substantial potential. However, the results remain preliminary and require more studies for further validation.

In addition, modulating macrophages could also potentially be a modality for cancer treatment since anti-tumor T cells could be inhibited by PD-L1 expressed

by intra-tumoral macrophages. T cell's anti-tumor responses against cancer could potentially improve by reducing intra-tumoral macrophages via negative regulator, such as colony stimulating factor-1 receptor (CSF1R). Five Prime Therapeutics is exploring FPA008 (anti-CSF1R) in combination with checkpoint inhibitors (Opdivo) as a potential treatment in solid tumors.

There are several in-development immunologic checkpoint inhibitors that potentially could also modulate both the CTL and NK cells immunity. Examples include Merck's MK-4166, which is a GITR (glucocorticoid-induced TNF receptor-related gene) -targeted monoclonal antibody currently in a Phase I study in advanced solid tumors.

CD30 is a well validated cancer treatment target. CD30 or TNFRSF8 is a member of the tumor necrosis factor (TNF) superfamily and plays a pleiotropic effect on cell growth and survival. In healthy tissues, CD30 is expressed in activated, but not resting T and B cells. CD30 expression is elevated in many hematological malignancies and a few solid tumors (i.e. testicular cancer). Cancer therapy that targets CD30 is well established with Adcetris (brentuximab vedotin) from Seattle Genetics as the first approved anti-CD30 antibody-drug conjugate treatment indicated for: 1) Hodgkin lymphoma (HL) after failure of autologous stem cell transplant (ASCT) or in relapsed/refractory patients; and 2) systemic anaplastic large cell lymphoma (ALCL) in relapsed/refractory patients. CD30 is expressed in > 95% of cases of classical HL, and in a majority of ALCL. In addition to HL and ALCL, CD30 expression is found in ~30% of diffuse large B-cell lymphoma (DLBCL) patients, and in ~10% of cutaneous T-cell lymphomas (CTCL) subtype called primary cutaneous CD30-positive large cell lymphoma ($CD30^+$ PCLCL).

Adcetris' mechanism of eliminating $CD30^+$ HL or ALCL cells is by releasing a microtubule disrupting agent, monomethyl auristatin E (MMAE) from the antibody once inside the cancer cells after the antibody binds to the surface of the cancer cell. The Adcetris pivotal studies in relapsed/refractory HL demonstrated a 73% ORR (CR: 32% and PR: 40%) and an overall 6.7 month median duration of response or DOR (3.5 months in PR patients and 20.5 months in CR patients). For relapsed/refractory ALCL, the ORR was 86% (CR: 57% and PR: 29%) and overall median DOR was 12.6 month (2.1 months in PR patients and 13.2 months in CR patients).

Seattle Genetics is conducting more than 30 corporate and investigator-sponsored clinical trials evaluating Adcetris in various indications. Some of the advanced studies include four Phase III studies: AETHERA (in post-transplant HL relapse prevention), ALCANZA (in relapsed $CD30^+$ cutaneous T-cell lymphoma), ECHELON-1 (plus chemotherapy as frontline HL), and ECHELON-2 (plus chemotherapy as frontline $CD30^+$ mature T-cell lymphoma). Three major Phase II studies are looking at 1) a frontline treatment in HL patients 60 years or older ($\pm$ other agents), 2) a frontline treatment in $CD30^+$ DLBCL (+ RCHP), and 3) a treatment in relapsed $CD30^+$ DLBCL (Rituxan + bendamustine $\pm$ BV).

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We view nivolumab (Opdivo) in r/r HL development from BMY could potentially be an important competitor.

Major competitors in HL. There are approximately a dozen of Phase II and beyond HL treatments in development. Among them, we view nivolumab (Opdivo) from BMY as potentially being an important competitor. BMY reported robust nivolumab in r/r HL Phase I trial (n=23) results at the 2014 ASH meeting illustrated an 87% ORR (20/23 with 4 in CR and 16 in PR) and an 86% PFS rate at 24 weeks. The FDA has granted nivolumab a breakthrough designation in relapsed classical HL. A larger (n=120) registration trial (CheckMate 205) is underway that evaluates nivolumab with top-line data possibly available in 2016. CheckMate 205 is a two-cohort, single arm, open-label, Phase II study that enrolls classical HL (cHL) patients after failure of ASCT. Both Adcetris naïve and experienced patients are eligible for the study. The primary endpoint is ORR, while secondary endpoints include DOR (duration of response), complete remission (CR) rate, duration and others.

Hodgkin's Lymphomas (HL). According to the American Cancer Society, the annual incidences for Hodgkin lymphoma (HL) in the U.S. are 9,000. HL is a type of lymphoma with the presence of a large size Reed-Sternberg cell. The cause of HL is unknown. HL can be further classified into classical Hodgkin's lymphoma (cHL), which accounts for ~95% of HL; and multiple rare HL subtypes, such as nodular sclerosing HL, lymphocyte-rich classical HL and mixed cellularity HL. For cHL, > 98% of patients is $CD30^+$; while for the remaining HLs, only <8% are $CD30^+$, like nodular sclerosing HL.

Based on the severity of the disease, HL can be staged according to the Ann Arbor classification as Stage I – IV. Treatments for Stage I and II patients are mostly radiation therapy and chemotherapies, such as MOPP (mechlorethamine, vincristine, procarbazine, prednisone) and ABVD (Adriamycin [doxorubicin], bleomycin, vinblastine, dacarbazine). Treated Stage I and II patients are with high cure rate (~ 90%). Advanced disease (III, IVA, or IVB) patients are treated with combination chemotherapy, such as ABVD (up to 6 cycles), Stanford V or repeated BEACOPP (bleomycin, etoposide, doxorubicin, cyclophosphamide, vincristine, procarbazine, and prednisone). Dependent on prognostic factors, relapse rate after treatment in localized and advanced (stages IIIB or IV) HL are 10% – 15% and 20% – 40%, respectively. Second and third-line chemotherapy combinations generally achieve 30% – 40% CR with aggressive or resistant disease.

Non-Hodgkin's Lymphomas (NHL). NHL is another type of lymphoma and according to the American Cancer Society, the annual incidences in the U.S. are ~70,000. NHL could further be divided into two sub-types, B-cell and T-cell lymphoma, based on the origin of the diseased cell. B-cell lymphoma accounts for 85% of total NHL, while the remaining 15% are T-cell lymphoma. In addition, a substantial portion of the NHL patients, both B and T cell, are also $CD30^+$. In B cell lymphoma, three sub-groups are with significant $CD30^+$ expression: diffuse large B-cell lymphoma (DLBCL), follicular lymphoma (FL) and primary mediastinal large B-cell (PMBL). DLBCL is the most common form of B cell lymphoma and account for ~37%. DLBCL is one of the most

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aggressive lymphomas and represents ~60% of all aggressive cases. Various studies estimate 4 – 26% DLBCL is $CD30^+$. FL accounts for 29% of total B cell lymphomas and is the most common indolent form of NHL. Patients with FL have no obvious symptoms of the disease at diagnosis. Various studies estimate 14 – 50% of FL is $CD30^+$. In T cell lymphoma, multiple subtypes are expressing CD30 of different levels, and in total, approximately 30% of T cell lymphoma are $CD30^+$. Within T cell lymphoma, all anaplastic large cell lymphoma (ALCL) express CD30. ALCL accounts for ~14% of all T cell lymphoma.

We estimate total annual peak sales for AFM13 could reach $600+MM.

AFM13 in HL and NHL revenue model. Our model (Figure 18) assumes that AFM13 could potentially reach the market in 2019 if approved in Adcetris r/r HL. We conservatively assume an annual course of therapy of $135k. We also assume approvals for various $CD30^+$ r/r lymphomas (DLBCL, ALCL and follicular lymphoma) to start in 2021. We estimate total annual peak sales for AFM13 could reach $600+MM.

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Figure 18: AFM13 in HL and CD30+ lymphoma revenue model

AFM13 in HL and CD30+ lymphoma revenue model	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030
Total U.S. Hodgkin Lymphoma incidences	9,716	9,803	9,891	9,980	10,070	10,161	10,252	10,345	10,438	10,532	10,626	10,722
% of R/R HL patients	67%	67%	67%	67%	67%	67%	67%	67%	67%	67%	67%	67%
R/R HL patients	6,509	6,463	6,521	6,580	6,639	6,699	6,759	6,820	6,881	6,943	7,006	7,069
% of Adertris treated r/r HL patients	50%	50%	50%	50%	50%	50%	50%	50%	50%	50%	50%	50%
Adertris treated r/r HL patients	3,255	3,231	3,261	3,290	3,320	3,349	3,380	3,410	3,441	3,472	3,503	3,534
% of patients eligible for AFM13	66%	66%	66%	66%	66%	66%	66%	66%	66%	66%	66%	66%
Patients eligible for AFM13	2,148	2,133	2,152	2,171	2,191	2,211	2,231	2,251	2,271	2,291	2,312	2,333
% of penetration by AFM13	8%	18%	26%	33%	37%	44%	49%	51%	50%	50%	50%	50%
AFM14 treated patients	172	384	560	717	811	973	1,093	1,148	1,135	1,146	1,156	1,166
AFM13 annual treatment costs ($)	135,000	138,294	141,668	145,125	148,666	152,294	156,010	159,816	163,716	167,710	171,802	175,994
U.S. AFM13 in r/r HL salvage sales ($MM)	**23**	**53**	**79**	**104**	**121**	**148**	**171**	**183**	**186**	**192**	**199**	**205**
Total U.S. Non-Hodgkin Lymphoma incidences	73,615	74,278	74,946	75,621	76,301	76,988	77,681	78,380	79,085	79,797	80,515	81,240
% of DLBCL patients	37%	37%	37%	37%	37%	37%	37%	37%	37%	37%	37%	37%
DLBCL patients	27,238	27,483	27,730	27,980	28,231	28,486	28,742	29,001	29,262	29,525	29,791	30,059
% of r/r DLBCL patients	42%	42%	42%	42%	42%	42%	42%	42%	42%	42%	42%	42%
r/r DLBCL patients	11,440	11,543	11,647	11,751	11,857	11,964	12,072	12,180	12,290	12,400	12,512	12,625
% of patients eligible for AFM13 as CD30+	22%	22%	22%	22%	22%	22%	22%	22%	22%	22%	22%	22%
Patients eligible for AFM13	2,517	2,539	2,562	2,585	2,609	2,632	2,656	2,680	2,704	2,728	2,753	2,777
% of penetration by AFM13			8%	18%	26%	33%	38%	44%	47%	48%	48%	48%
AFM14 treated patients			205	465	678	869	1,009	1,179	1,271	1,309	1,321	1,333
AFM13 annual treatment costs ($)			141,668	145,125	148,666	152,294	156,010	159,816	163,716	167,710	171,802	175,994
U.S. AFM13 in r/r DLBCL sales ($MM)			**29**	**68**	**101**	**132**	**157**	**188**	**208**	**220**	**227**	**235**
Total U.S. Non-Hodgkin Lymphoma incidences	73,615	74,278	74,946	75,621	76,301	76,988	77,681	78,380	79,085	79,797	80,515	81,240
% of ALCL patients !00% CD30+	14%	14%	14%	14%	14%	14%	14%	14%	14%	14%	14%	14%
ALCL patients	1,524	1,538	1,551	1,565	1,579	1,594	1,608	1,622	1,637	1,652	1,667	1,682
% of r/r ALCL patients	42%	42%	42%	42%	42%	42%	42%	42%	42%	42%	42%	42%
r/r ALCL patients	640	646	652	657	663	669	675	681	688	694	700	706
% of penetration by AFM13			7%	17%	25%	33%	37%	41%	44%	45%	45%	45%
AFM14 treated patients			46	112	166	221	250	279	303	312	315	318
AFM13 annual treatment costs ($)			141,668	145,125	148,666	152,294	156,010	159,816	163,716	167,710	171,802	175,994
U.S. AFM13 in r/r ALCL sales ($MM)			**6**	**16**	**25**	**34**	**39**	**45**	**50**	**52**	**54**	**56**
Total U.S. Non-Hodgkin Lymphoma incidences	73,615	74,278	74,946	75,621	76,301	76,988	77,681	78,380	79,085	79,797	80,515	81,240
% of follicular lymphoma patients	29%	29%	29%	29%	29%	29%	29%	29%	29%	29%	29%	29%
Follicular lymphoma patients	21,348	21,541	21,734	21,930	22,127	22,326	22,527	22,730	22,935	23,141	23,349	23,560
% of r/r follicular lymphoma patients	42%	42%	42%	42%	42%	42%	42%	42%	42%	42%	42%	42%
r/r follicular lymphoma patients	8,966	9,047	9,128	9,211	9,293	9,377	9,462	9,547	9,633	9,719	9,807	9,895
% of patients eligible for AFM13 as CD30+	25%	25%	25%	25%	25%	25%	25%	25%	25%	25%	25%	25%
Patients eligible for AFM13	2,242	2,262	2,282	2,303	2,323	2,344	2,365	2,387	2,408	2,430	2,452	2,474
% of penetration by AFM13			8%	18%	26%	33%	38%	39%	40%	40%	40%	40%
AFM14 treated patients			183	414	604	774	899	931	963	972	981	990
AFM13 annual treatment costs ($)			141,668	145,125	148,666	152,294	156,010	159,816	163,716	167,710	171,802	175,994
U.S. AFM13 in r/r follicular lymphoma r/r sales ($MM)			**26**	**60**	**90**	**118**	**140**	**149**	**158**	**163**	**168**	**174**
Total U.S. AFM13 sales ($MM)	**23**	**53**	**141**	**248**	**336**	**432**	**507**	**565**	**601**	**627**	**648**	**670**
(€MM)	**22**	**50**	**132**	**233**	**316**	**406**	**477**	**531**	**565**	**589**	**609**	**630**

Source: Laidlaw & Company estimates

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AFM11 Could Have Significant Improvements Over the Approved Blincyto

AFM11 engages T cells to eliminate CD19 expressing B-cell malignancies

AFMD's second leading bispecific antibody in development is AFM11. After binding to CD19 expressing B-cell malignancies (i.e. in NHL, ALL), AFM11 also binds to cytotoxic T cells via CD3, and brings bound cancer cells within a close proximity of the T cell to be destroyed. The mechanism of action for AFM11 is similar to another recently approved T cell engaging bispecific antibody, blincyto (blinatumomab).

Initially developed by Micromet and subsequently acquired by Amgen, blincyto is a bispecific antibody that binds to both CD19 and CD3. Blincyto was approved in 4Q14 in the U.S. (and in Nov. 2015 in Europe) as a treatment of Philadelphia chromosome-negative (Ph-) relapsed or refractory B-cell precursor acute lymphoblastic leukemia (ALL). The registration pivotal study (n=185) for the initial blincyto approval demonstrated the treatment met the primary endpoint of complete remission/complete remission with partial hematological recovery (CR/CRh*) rate of 42% (CR of 32% and CRh* of 9%). MRD (minimal residual disease) response in CR/CRh* was 75% with CR of 80% and CRh* of 59%. AMGN is currently conducting several clinical studies evaluating blincyto with key programs that include: Phase III in adult relapsed/refractory ALL, Phase II in adult relapsed/refractory Philadelphia chromosome-positive (Ph+) and minimal residual disease of ALL, and Phase II in adult DLBCL. AMGN and Merck recently announced they will start an open-label Phase Ib/III trial to evaluate blincyto/Keytruda combination to treat DLBCL.

By comparing to blincyto, AFM11 exhibited several material advantages, which we believe could render AFM11 potentially as a substantially improved T cell engaging bispecific antibody to treat multiple CD-19^{+} B cell malignancies (Figure 19). They are:

We believe AFM11's less frequent dosing attribute could have substantial advantage over blincyto from treatment logistic perspective.

- Due to its molecular weight (104 kDa) is much closer to the size of a regular IgG (160 kDa) than blincyto (55 kDa), AFM11 has a substantially longer half-life ($t_{1/2}$) in plasma than blincyto and, therefore, can be administrated via a regular periodical intravenous infusion. Blincyto is administrated by continuous infusion via a portable pump since the drug was cleared from the kidney at a much faster rate than larger molecular weight proteins. We believe AFM11's less frequent

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dosing attribute could have substantial advantage over blincyto from the treatment logistic perspective.

- AFMD has constructed AFM11 with two binding sites to CD3, while blincyto only has one. Along with designed higher affinity to CD3, AFM11 demonstrated a total of 100x affinity vs. blincyto (1 nM vs. 100 nM).
- Together, from pre-clinical studies, AFM11 demonstrated greater cytotoxic potency in eliminating cancer cells. In the study, researchers examined the cytotoxic potency of AFM11 vs. a reference compound of the same sequence as blincyto at various effector cell (T-cell) to tumor cell ratios. When researchers placed five folds of tumor cells to that of T-cells (or effector cell to tumor cell or E:T = 0.2), AFM11's potency is 40-fold higher than that of blinatumomab (Figure 20, left). Further, AFM11 led to greater complete tumor cell lysis (death) at low T-cell counts when compared to a blinatumomab reference compound (Figure 20, right).

AFM11 demonstrated a total of 100x affinity vs. blincyto (1 nM vs. 100 nM).

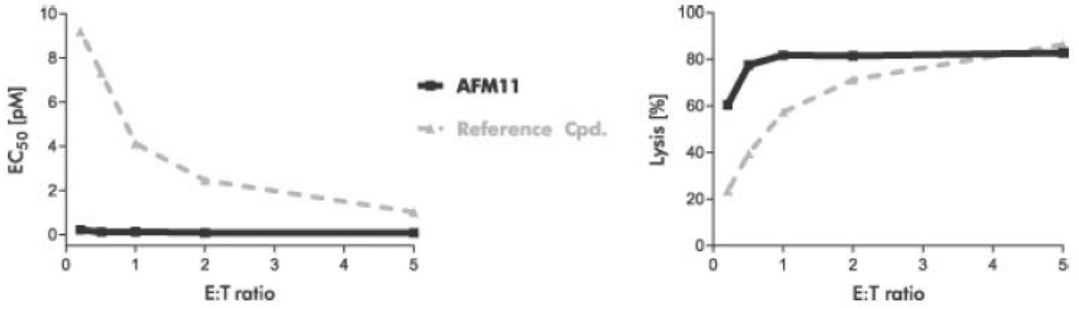
AFM11's potency is 40-fold higher than that of blinatumomab. AFM11 led to greater complete tumor cell lysis (death) at low T-cell counts when compared to a blinatumomab reference compound.

Figure 19: Major advantages of AFM11 vs. Blincyto

	Blinatumomab	AFM11
MW	~55 kDa	~104 kDa
Binding sites	1 for CD3 & CD19	2 for CD3 & CD19
Affinity to $CD3^+$ cells	100 nM	1 nM

Source: Company presentation

Figure 20: Cytotoxic potency of AFM11 vs. a reference compound at various effector cell (T-cell) to tumor cell ratios. Left: cytotoxicity (stronger, if lower EC50); right: % cell lysis at 10 pM antibody concentration.



Source: Company presentation

We believe AFM13's higher cytotoxic potency (needs fewer T cells for destroying cancer cells) could potentially be an advantage clinically. Given that the immune systems of cancer patients are generally weakened and some patients receiving chemotherapy might also suffer from lymphopenia with a significant reduction of T-cells, a more "efficient" therapy, like AFM11 could be more beneficial.

Current developments. AFMD initiated an AFM11 in relapsed/refractory $CD19^+$ NHL and ALL Phase I dose finding clinical trial in 2Q15 to assess safety. The study is expected to enroll ~40 patients and the doses are ranging from 0.0003 to 2.5 μg/kg per infusion. Each treatment cycle is comprised of four weeks. The initial regimen is 5x/week for the first week and 3x/week for the remaining three weeks. The first part of the study focused on treating NHL patients who already have been treated with at least one rituximab-based chemotherapy regimen. The primary endpoint is safety and tolerability in a single cycle of AFM11 monotherapy in NHL patients, while secondary endpoints include MTD (maximum tolerated dose) or OBD (optimal biological dose), PK and tumor response (based on PDG-PET and CT- scans). AFMD recently amended the protocol to a less frequent dosing from the prior intensive dosing. During the 3Q15 conference call, management indicated that top-line results could be available in in 2H16 with more updates in 2017.

AFMD recently amended the protocol to a less frequent dosing from the prior intensive dosing. Top-line results could be available in in 2H16 with more updates in 2017.

AFMD also starts the AFM11 in ALL (possibly mainly in adults) trial as a separate Phase I trial with commencement expected in 1Q16.

AFMD also starts the AFM11 in ALL trial as a separated Phase I trial with commencement expected in 1Q16

The combination of CD19 and CD3 in B-cell malignancies treatment is a clinically well validated therapeutic modality. We view the combination of targeting CD19 and CD3 to engage T cells to treat CD19 expressing B cell malignancies as well validated clinically. It is not only just that blincyto was already approved for r/r ALL; several late stage clinical studies are also ongoing. In addition, several chimeric antigen receptor (CAR)-T cell programs also have targeted CD19 and have demonstrated compelling clinical results, albeit the studies remain in earlier clinical stages. Examples in the CAR-T arena include JCAR014 (Phase I/II in adult ALL and NHL) and JCAR017 (Phase I/II in pediatric ALL) from JUNO Therapeutics, KTE-19 (Phase I/II trial in DLBCL to start in 1H15) from Kite Pharma, and CTL019 (Phase I/II in relapsed /refractory pediatric ALL) from Novartis.

Amgen reported encouraging clinical results in 4Q14 from Phase II studies that evaluated blincyto in minimal residual disease (MRD) positive B-cell precursor ALL (BLAST or ‘203 trial) and adult relapsed/refractory B-cell precursor acute ALL ('211 trial). In the BLAST trial, 78% (69% - 85%) of blincyto treated patients experienced a complete (MRD) after one treatment cycle. 80% achieved a complete MRD response across all cycles. Nearly all complete responses (98%) occurred within the first treatment cycle. MRD is a state of disease in which the microscopic analysis does not show malignant cells, but more sensitive techniques still detect disease at the molecular level. The primary endpoint of the study is rate of complete MRD response, while secondary endpoints include incidence and severity of AE, hematological relapse-free survival rate, overall survival, time to hematologic relapse, and duration of complete MRD response.

In the '211 trial, 40% of blincyto treated patients achieved a complete remission (CR) or complete remission with partial hematologic recovery (CRh) and were

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enabled to proceed to allogeneic hematopoietic stem cell transplant (HSCT). In addition, 82% of CR / CRh patients also had a MRD response.

Although the treatment landscape in various CD19 positive B cell malignancies seemingly is becoming more competitive, we believe AFM11 could have a certain competitive edge if the upcoming clinical activity and safety data are positive. First, from the bispecific antibody perspective, we view the potentially more potent and possible infrequent dosing of AFM11 could afford it therapeutic advantages over blincyto. Further, AFM11 is currently being developed in both NHL and ALL. One key aspect worth investors' attention near term would be the safety profile of AFM11. In addition, we believe the manufacturing flexibility (using both prokaryotic and eukaryotic systems with high yields) of AFM11 could also account for a modest advantage on the COGS over blincyto.

From the competitive perspective of CAR-T, in spite of the very compelling early clinical results in ALL treatment, and to less extend, in the NHL side, the safety concerns due to cytokine release syndrome (CRS) remain a major hurdle that need to be overcome before many CAR-T programs can have a smooth sail through the approval processes. Cytokine release syndrome is potentially life-threatening. It refers to dangerously high fevers and precipitous drops in blood pressure due to rapid and massive release of cytokines (such as IL-6 and interferon γ) into the bloodstream from the presences of high-level immune activation from larger quantities of potent engineered T cells. In addition, given CAR-T programs are more "customized" than "off-the-shelf", it potentially could be much more costly. As such, a cost / benefit analysis could be critical for its commercial viability.

AFMD is currently pursuing AFM11 developments in NHL and ALL. For ALL, we believe AFM11 could be a viable treatment option if clinically successful despite that blincyto is already approved and CAR-T could be more potent.

AFM11 in NHL and ALL revenue model. Our model (Figure 21) assumes that AFM11 could potentially reach market in 2021 if approved in r/r ALL. We conservatively assume an annual course of therapy of $150k (blincyto Medicare payment to $178k for standard course). We also assume approvals for various NHLs (DLBCL, MCL and follicular lymphoma) to start in 2022. We estimate total annual peak sales for AFM11 could reach $900+MM.

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Figure 21: AFM11 in NHL and ALL revenue model

AFM11 in NHL and ALL Revenue Model	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030
Total U.S. Non-Hodgkin Lymphoma incidences	74,946	75,621	76,301	76,988	77,681	78,380	79,085	79,797	80,515	81,240
% of DLBCL patients	30%	30%	30%	30%	30%	30%	30%	30%	30%	30%
DLBCL patients	22,484	22,686	22,890	23,096	23,304	23,514	23,726	23,939	24,155	24,372
% of 3rd-line r/r DLBCL patients	50%	50%	50%	50%	48%	46%	44%	43%	41%	40%
r/r DLBCL patients	11,242	11,343	11,445	11,548	11,186	10,816	10,439	10,174	9,903	9,627
% of penetration by AFM11		8%	17%	23%	27%	29%	30%	31%	31%	31%
AFM11 treated patients	0	907	1,946	2,656	3,020	3,137	3,132	3,154	3,070	2,984
AFM11 annual treatment costs ($)	150,000	153,000	156,060	159,181	162,365	165,612	168,924	172,303	175,749	179,264
U.S. AFM11 in r/r DLBCL salvage sales ($MM)	**0**	**139**	**304**	**423**	**490**	**519**	**529**	**543**	**540**	**535**
Total U.S. Non-Hodgkin Lymphoma incidences	74,946	75,621	76,301	76,988	77,681	78,380	79,085	79,797	80,515	81,240
% of MCL patients	6%	6%	6%	6%	6%	6%	6%	6%	6%	6%
MCL patients	4,497	4,537	4,578	4,619	4,661	4,703	4,745	4,788	4,831	4,874
% of r/r MCL patients	50%	50%	50%	50%	48%	46%	44%	43%	41%	40%
r/r MCL patients	2,248	2,269	2,289	2,310	2,237	2,163	2,088	2,035	1,981	1,925
% of penetration by AFM11		7%	15%	19%	24%	27%	28%	29%	30%	30%
AFM11 treated patients	0	159	343	439	537	584	585	590	594	578
AFM11 annual treatment costs ($)	150,000	153,000	156,060	159,181	162,365	165,612	168,924	172,303	175,749	179,264
U.S. AFM11 in r/r MCL salvage sales ($MM)	**0**	**24**	**54**	**70**	**87**	**97**	**99**	**102**	**104**	**104**
Total U.S. Non-Hodgkin Lymphoma incidences	74,946	75,621	76,301	76,988	77,681	78,380	79,085	79,797	80,515	81,240
% of FL patients	25%	25%	25%	25%	25%	25%	25%	25%	25%	25%
FL patients	18,737	18,905	19,075	19,247	19,420	19,595	19,771	19,949	20,129	20,310
% of r/r FL patients	20%	20%	20%	20%	19%	18%	17%	16%	15%	14%
r/r FL patients	3,747	3,781	3,815	3,849	3,690	3,527	3,361	3,192	3,019	2,843
% of penetration by AFM11		9%	16%	22%	25%	27%	28%	29%	29%	29%
AFM11 treated patients	0	321	610	847	922	952	941	926	876	825
AFM11 annual treatment costs ($)	150,000	153,000	156,060	159,181	162,365	165,612	168,924	172,303	175,749	179,264
U.S. AFM11 in r/r FL salvage sales ($MM)	**0**	**49**	**95**	**135**	**150**	**158**	**159**	**159**	**154**	**148**
Total U.S. acute lymphoblastic leukemia (ALL) incidences	6,655	6,714	6,775	6,836	6,897	6,959	7,022	7,085	7,149	7,213
% of r/r ALL patients	50%	50%	50%	50%	48%	46%	44%	43%	41%	40%
r/r ALL patients	3,327	3,357	3,387	3,418	3,311	3,201	3,090	3,011	2,931	2,849
% of penetration by AFM11	5%	10%	17%	22%	25%	27%	29%	30%	31%	32%
AFM11 treated patients	166	336	576	752	828	864	896	903	909	912
AFM11 annual treatment costs ($)	150,000	153,000	153,000	153,000	153,000	153,000	153,000	153,000	153,000	153,000
U.S. AFM11 in r/r FL salvage sales ($MM)	**25**	**51**	**88**	**115**	**127**	**132**	**137**	**138**	**139**	**140**
Total AFM11 U.S. revenue ($MM)	**25**	**264**	**541**	**743**	**854**	**906**	**924**	**943**	**937**	**926**
(€MM)	**23**	**248**	**508**	**698**	**803**	**852**	**868**	**886**	**881**	**870**

Source: Laidlaw & Company estimates

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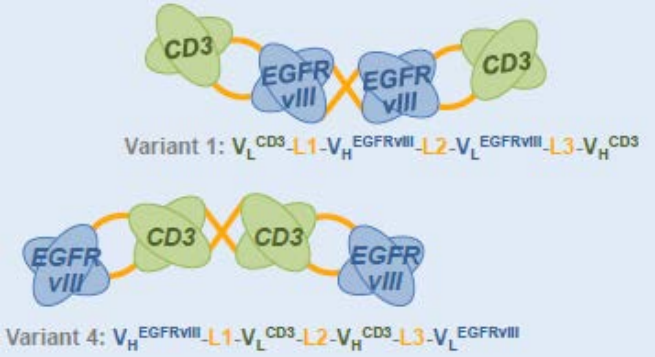
AFM21/22 For Solid Tumors and Multiple Partnerships Amphivena Therapeutics

AFMD's third product targets EGFRvIII (Epidermal growth factor receptor variant III) as a potential treatment of solid tumors by engaging either NK cells (AFM21 via 16A) or CTLs (AFM22 via CD3) (Figure 22). Both programs currently are in pre-clinical development. EGFRvIIIs (mutant versions of EGFR) (Figure 23) are prominently expressed in many solid tumors.

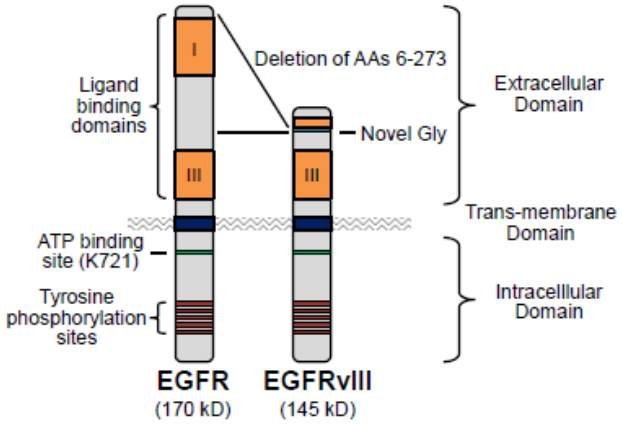
A third product targets EGFRvIII as a potential treatment of solid tumors by engaging either NK cells (AFM21) or CTLs (AFM22). AFMD is in the process in deciding which version will be the final anti-AFM21 TandAb candidate (likely in 1H16) before advancing it for pre-clinical IND-enabling studies with Phase I trial potentially to start in 2017.

Figure 22: AFM22 (via CD3) and AFM21 (via 16A)



Source: Company presentation

Figure 23: EGFRvIII is associated with oncogenic transformation



Source: Company presentation

Possible tumors that could be treated include glioblastoma, hormone refractory prostate cancer and head and neck cancer

AFM21/22 has a high specificity against tumor tissues associated with EGFRvIII but not to wild-type EGFR, which is expressed on healthy tissues. Possible tumors that could be treated include glioblastoma, hormone refractory prostate cancer and head and neck cancer (Figure 24). The company is in the process of deciding which version will be the final anti-AFM21 TandAb

candidate (likely in 1H16) before advancing it for pre-clinical IND-enabling studies with a Phase I trial potentially to start in 2017. Management discussed during the 3Q15 call that a NK engaging bispecific antibody could have some advantages from a safety prospective but a final decision has not yet been reached.

Figure 24: Incidence of EGFRvIII in human cancers

Tumor type	Positive / Total	% Positive	Dection Techique
Glioblastoma	16/31	52%	Immunohistochemistry
	35/62	56%	Western blotting
	9/38	24%	RT-PCR
	8/12	67%	Immunohistochemistry
	7/12	58%	Western blotting
	5/12	42%	RT-PCR
	32/48	67%	cDNA sequencing
Breast	3/11	27%	Immunohistochemistry
	8/10	80%	RT-PCR
	21/27	78%	Western blotting
Ovary	24/32	75%	Western blotting
Non-small cell lung	5/32	16%	Immunohistochemistry
Prostate	38/38	100%	Immunohistochemistry

Source: Company presentation

AFMD reported an AFM21 pre-clinical study at the Society for Immunotherapy of Cancer (SITC) conference in Nov. 2015 and demonstrated that NK cells (AFM21) and T cells (AFM22) engaging TandAbs showed similar cytotoxic and *in vitro* potency. In addition, EGFRvIII-specific human scFv isolated and affinity matured to achieve K_Ds at 100 pico-molar range.

Partnerships. AFMD entered a license deal in 2012 and 2013 with Amphivena Therapeutics, which is funded from several investment funds and AFMD (owned ~28%), to develop a CD33 and CTL (via CD3) engaging bispecific antibody potentially for the treatment of acute myeloid leukemia (AML). AFMD is entitled to interest in Amphivena and certain milestone payments. Amphivena also entered into an agreement with Janssen Biotech, part of Johnson & Johnson. Janssen has an option to buy Amphivena upon IND acceptance by the FDA. AFMD reported to receive a €7.5M payment (paid in three installments) from Amphivena in 1Q15 after achieving the second milestone for selection of a development candidate against an undisclosed target for the treatment of a certain hematologic malignancy.

CD33/CD3 bispecific antibody has potential for acute myeloid leukemia (AML) treatment. Janssen has an option to buy Amphivena upon IND acceptance by the FDA.

The CD33 / CD3 bispecific antibody is currently in preclinical development with CMC and toxicology work underway. AFMD presented three posters at the 2015 ASCO annual meeting highlighting the CD33/CD3 bispecific antibody (AMV-564) based on various *in vitro* analyses.

CD33 is a member of the sialic acid-binding immunoglobulin-like lectins (Siglecs). In addition, as a myeloid differentiation antigen, CD33 is found on at least a subset of blasts in nearly all AML patients. Further, CD33 expression is also found in some AML stem cells. There is an anti-CD33 and CD3 bispecific

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antibody (AMG 330) currently under development by Amgen. AMG 330 was initially developed by Micromet and it is a BiTE antibody. Pre-clinical data presented at the 2014 ASH annual meeting suggested that AMG 330 causes potent cytolysis of primary human AML cells *in vitro* in a dose- and E:T cell ratio-dependent manner across the entire spectrum of cytogenetic/molecular risk and disease stage, even in specimens with very low expression of CD33. Lower activity of AMG 330 was observed in relapsed/refractory AML specimens and intermediate- or adverse-risk disease specimens suggesting the potential presence of yet undefined, CD33-independent, relative resistance mechanisms in defined patient subsets.

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Financial projections and valuation

After its post IPO equity offering (May 12, 2015), AFMD recently reported it ended 3Q15 with ~€60.4MM. Combined with €19.1MM raised in Oct. 2015, we estimate the company has pro forma cash of ~€78MM (~$83MM), which could be sufficient for the company's operation into 2018.

Our probability-adjusted DCF analysis suggested a one-year target value for AFMD of $14.98 based on cash flow until 2025 with an assumed terminal value multiple of three and a probability adjustment of 42%.

Probability-adjusted DCF analysis

Cash driven NPV	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Revenue	7	7	7	29	57	163	488	831	1,111	1,287
R&D	(31)	(35)	(39)	(42)	(46)	(49)	(52)	(56)	(60)	(64)
SG&A	(9)	(10)	(11)	(33)	(38)	(43)	(45)	(47)	(50)	(52)
Operating income	(33)	(39)	(42)	(49)	(34)	48	319	604	836	1,016
Net income	(35)	(41)	(44)	(51)	(36)	29	200	380	526	639
Period	0.1	1.1	2.1	3.1	4.1	5.1	6.1	7.1	8.1	9.1
NPV	(35)	(35)	(34)	(34)	(21)	15	88	146	176	187

Total DCF	488
Terminal value	630
Cash (4Q16)	40
Total valuation (€ '000)	1,158
Probability adjustment	42%
Value per share	**€ 14.08**
	$14.98
Share outstanding (YE16)	35
Discount rate	15%
Terminal value multiple	3

Source: Laidlaw & Company estimates

Our probability-adjusted-PV-driven, sum-of-the-parts analysis illustrates a breakdown of value for each potential value driver, with AFM13, AFM11 and others in the pipeline accounting for 36%, 44% and 12% of the total value, respectively. This analysis suggested a 12-month target price of $15.07.

NPV driven sum-of-the-parts analysis

AFM13	**HL and CD30+ lymphoma**		
	Adjusted NPV =	**€ 178**	
	PV per share =	**€ 5**	**36%**
AFM11	**NHL and ALL**		
	Adjusted NPV =	**€ 215**	
	PV per share =	**€ 6**	**44%**
Other pipeline			
	Adjusted NPV =	**€ 60**	
	PV per share =	**€ 2**	**12%**
Cash			
	Adjusted NVP =	**€ 40**	
	NVP per share =	**€ 1**	**8%**
	Total =	**€ 14.17**	**100%**
		$15.07	

Source: Laidlaw & Company estimates

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For the peer comparable analysis, we have chosen a group of companies mainly developing IO therapies (especially NK-focused and bi-specific Abs) as comparable peers. As such, our peer comparable analysis suggested a 12-month target price for AFMD of $15.06.

Comparable analysis

Company	Ticker	Rating	Target Price ($)	Price ($) (12/03/15)	Shares Outstanding (MM)	Market Cap ($ MM)	Cash ($ MM)	Debt ($ MM)	Tech Value ($ MM)	Most Advanced Trials	Comments
Macrogenics	**MGNX**	NR	NA	33.12	34	1136	366	0	771	Phase II	Bi-specific and other engineered Abs
Xencor	**XNCR**	NR	NA	14.61	40	591	128	0	463	Phase II	Bi-specific and other engineered Abs
NantKwest	**NK**	NR	NA	15.99	81	1299	359	0	940	Phase II	NK-focused
Prima Biomed	**PBMD**	NR	NA	1.26	66	83	5	0	78	Phase II	T cell (LAG-3 immune control)
Trillium Therapeutics	**TRIL**	NR	NA	13.67	7	100	67	0	33	Phase I	CPI
Immune Design	**IMDZ**	NR	NA	21.37	20	430	120	0	310	Phase II	Dendritic cell activation
Calithera Biosciences	**CALA**	NR	NA	7.24	18	131	92	0	40	Phase I	IO metabolic CPI
Innate Pharma S.S.	**PSE-IPH**	NR	NA	14.20	54	764	279	0	485	Phase II	NK-focused
Sorrento Therapeutics	**SRNE**	NR	NA	7.63	38	288	123	0	165	Phase II	Various IO and non-IO programs
Average						**480**	*142*	*2*	**365**		
Affimed Therapeutics	**AFMD**	**Buy**	**15.00**	**6.64**	**30**	**197**	**83**	**0**	**115**	**Phase II**	**NHL, ALL**

AFMD share fair value matching its IO Phase I/II peers = $15.06

Potential upside = 127%

Source: Company reports and Laidlaw & Company estimates

Together, we assigned our blended 12-month target price for AFMD of **$15**.

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Major risks

Clinical study failure could have a major impact on AFMD share value. Despite promising pre-clinical and Phase I trial results of the company's lead products, AFM13 and AFM11, it remains too early to predict the longer term safety and efficacy from the current ongoing clinical studies. Given clinical validation for these programs has not been fully established, it would be critical for some or all of these studies to demonstrate positive outcomes in order to increase the assets and shareholder value. Negative results of either Phase II studies could have a materially negative impact on the asset and shareholder value; especially each study could fail to illustrate proof-of-concept for AFM13 and AFM11 as potential treatment of different disease indications. Further, it remains too early to predict any potential success of clinical trials in the future should these programs further advance into next clinical stage development.

Yet-to-be-validated NK cell platform and rapidly changing dynamic of IO platforms as cancer therapy could create more uncertainty. Although multiple prior pre-clinical and clinical data from many investigators suggest that NK cell based therapy could have significant potential for treating cancer; currently there is no NK-cell based therapy that is approved or in late clinical stage cancer treatment development. As such, clinical risks for NK-cell based cancer therapy are higher than other treatment modalities. In addition, multiple types of immune-oncology (IO) therapy platforms (i.e. CPI and CAR-T) are all in relatively early and active development, it remains too early to predict, especially for the one that has not yet received approval, which platform could be approved and gaining market shares in the future. Bi- and tri-specific antibodies can be categorized into the IO therapy group.

Product may not be approved or reach anticipated sales. Although AFMD's current pipeline products have exhibited the potential to generate positive clinical outcomes from current and future trials; it remains too early to project whether any of these products would be approved by regulatory agencies. Even if the products were to enter the market, sales could be significantly below projections due to the specific product label under approval, physician consensus for prescribing the drug, changes of treatment paradigms, entrance of competitors, and possibly the changes in pricing flexibility and payer reimbursement. A revenue outlook below expectations could also negatively affect AFMD shareholder value.

Additional financings could dilute shareholder value. Although the company currently has ~$83MM (pro forma) cash after recent financing, AFMD could need more financial resources going forward if they want to expand and further develop their pipeline. Should the future operational expenses, especially from R&D, increase significantly, products not receive FDA approval, or product revenue does not reach expectations; the company might need to issue new equity to raise additional cash. Under such a scenario, the share value of existing shareholders could be diluted.

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Limited trading liquidity limits shareholder options. Given that ALDX shares only entered the public market recently; daily trading volume and name recognition are relatively modest. As such, shareholders wanting to increase or reduce their positions more substantially in a volatile stock market may face constraints.

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Management

Adi Hoess, Ph.D. is Affimed Therapeutics CEO since 2011 and has been the company's Chief Commercial Officer since 2010. Prior to joining Affimed, he served as Chief Commercial Officer at Jerini AG and Chief Executive Officer of Jenowis AG. Prior to Jenowis AG, Dr. Hoess began his professional career in 1993 at MorphoSys as scientist and later as Director and VP of BD of Licensing and BD. Dr. Hoess holds a Ph.D. degree in chemistry and biochemistry from the University of Munich and an M.D. from the Technical University of Munich.

Florian Fischer, Ph.D. has served as CFO of Affimed Therapeutics since June 2005 initially on a part-time basis. Prior to joining Affimed Dr. Fischer was founder and Chief Executive Officer of MedVenture Partners. Prior to joining MedVenture Partners, he served as Chief Financial Officer of Activaero GmbH from 2002 until 2011. Prior, he served as the Chief Financial Officer of Vivendy Ltd. from 2008 until 2013 and as a managing director of AbCheck in 2009. Prior to founding MedVenture Partners, Dr. Fischer worked with KPMG from 1996 to 2002. Before joining KPMG, he worked for Deutsche Bank AG. Dr. Fischer holds a Ph.D. degree in public health from the University of Bielefeld.

Jens-Peter Marschner, MD has served as Chief Medical Officer of Affimed Therapeutics since 2013. Prior to joining Affimed from 2003 to 2011, Dr. Marschner served as Vice President of Immunological Programs Oncology and earlier as Vice President of Global Medical Affairs (2003-2009) for Merck KGaA (Merck Serono). Prior, Dr. Marschner started his pharmaceutical career in 1995 at Boehringer Mannheim. Dr. Marschner holds a M.D. degree from Johann-Wolfgang-Goethe-University.

Martin Treder, Ph.D. has served as Chief Scientific Officer of Affimed Therapeutics since 2015. Prior to joining Affimed from 2003 to 2011, Dr. Treder served as co-founder and Chief Scientific Officer for CT Atlantic AG. Prior to CT Atlantic, he served as co-founder and Program Director of U3 Pharma AG. Dr. Treder holds a Ph.D. degree from Max Planck Institute of Biochemistry

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Affimed Therapeutics N.V. – Income Statement

(€'MM)	2014	1Q15	2Q15	3Q15	4Q15E	2015E	1Q16E	2Q16E	3Q16E	4Q16E	2016E	2017E	2018E	2019E	2020E	2021E	2022E
Revenue																	
Product revenue	0.0	-	-	-	-	0.0					0.0	0.0	0.0	21.8	49.9	155.6	480.9
Research revenue	3.4	2.5	2.2	1.2	2.1	8.0	1.5	1.7	1.4	1.9	6.5	6.5	6.5	6.5	6.5	6.5	6.5
Other revenue	0.4	0.2	0.1	0.3	0.2	0.8	0.2	0.2	0.2	0.2	0.8	0.8	0.8	0.8	0.8	0.8	0.8
Total revenue	3.8	2.8	2.3	1.5	2.3	8.8	1.7	1.9	1.6	2.1	7.3	7.3	7.3	29.1	57.2	163	488
Costs of goods														3.3	7.5	23.3	72.1
Gross sales														18.5	42.4	132.3	408.7
Research and development	(9.6)	(2.9)	(5.6)	(6.4)	(6.9)	(21.9)	(7.2)	(7.5)	(8.0)	(8.6)	(31.4)	(35.5)	(38.7)	(42.1)	(45.5)	(48.7)	(52.1)
General and administrative	(2.3)	(1.8)	(1.7)	(2.1)	(2.2)	(7.7)	(2.2)	(2.2)	(2.3)	(2.4)	(9.2)	(10.5)	(11.0)	(11.6)	(12.1)	(12.7)	(13.4)
Marketing and sales														(21.0)	(26.3)	(30.2)	(31.7)
Total Operating Expenses	(11.9)	(4.8)	(7.3)	(8.5)	(9.1)	(29.6)	(9.5)	(9.8)	(10.3)	(11.0)	(40.6)	(46.0)	(49.7)	(74.7)	(83.9)	(91.6)	(97.2)
Operating Incomes (losses)	(8.2)	(2.0)	(5.0)	(7.1)	(6.8)	(20.8)	(7.8)	(7.9)	(8.7)	(8.9)	(33.3)	(38.7)	(42.4)	(48.9)	(34.2)	48.0	318.9
Finance income / (costs) - net	7.8	0.5	(0.2)	(0.2)	3.1	3.2	(0.5)	(0.5)	(0.5)	(0.5)	(2.0)	(2.0)	(2.0)	(2.0)	(2.0)	(2.0)	(2.0)
Loss before tax	(0.4)	(1.5)	(5.2)	(7.3)	(3.7)	(17.6)	(8.3)	(8.4)	(9.2)	(9.4)	(35.3)	(40.7)	(44.4)	(50.9)	(36.2)	46.0	316.9
Tax	0.2	-	-	(0.0)	0.0	0.0	-	-	-	-	0.0	0.0	0.0	0.0	0.0	(17.0)	(117.2)
Net Income (Loss)	(0.3)	(1.5)	(5.2)	(7.3)	(3.6)	(17.6)	(8.3)	(8.4)	(9.2)	(9.4)	(35.3)	(40.7)	(44.4)	(50.9)	(36.2)	29.0	199.6
Net Income (Loss) Applicable to Common Shareholders	(0.3)	(1.5)	(5.2)	(7.3)	(3.6)	(17.6)	(8.3)	(8.4)	(9.2)	(9.4)	(35.3)	(40.7)	(44.4)	(50.9)	(36.2)	29.0	199.6
Net Income (Loss) Applicable to Common Shareholders ($)	(0.3)	(1.6)	(5.6)	(7.9)	(3.8)	(19.0)	(8.8)	(8.9)	(9.8)	(10.0)	(37.5)	(43.2)	(47.2)	(54.1)	(38.5)	30.8	212.4
Net Earnings (Losses) Per Share—Basic	(€ 0.01)	(€ 0.06)	(€ 0.19)	(€ 0.24)	(€ 0.11)	(€ 0.60)	(€ 0.24)	(€ 0.24)	(€ 0.27)	(€ 0.27)	(€ 1.02)	(€ 1.14)	(€ 1.18)	(€ 1.07)	(€ 0.72)	€ 0.55	€ 3.73
Net Earnings (Losses) Per Share—Diluted	(€ 0.01)	(€ 0.06)	(€ 0.19)	(€ 0.24)	(€ 0.11)	(€ 0.60)	(€ 0.24)	(€ 0.24)	(€ 0.27)	(€ 0.27)	(€ 1.02)	(€ 1.14)	(€ 1.18)	(€ 1.07)	(€ 0.72)	€ 0.55	€ 3.73
Net Earnings (Losses) Per Share—Basic/diluted ($)	($0.01)	($0.07)	($0.20)	($0.26)	($0.12)	($0.64)	($0.26)	($0.26)	($0.28)	($0.29)	($1.09)	($1.22)	($1.26)	($1.14)	($0.76)	$0.59	$3.96
Shares outstanding—basic	22.0	24.0	27.8	30.8	34.1	29.1	34.3	34.5	34.7	34.9	34.6	35.6	37.6	47.6	50.6	52.6	53.6
Shares outstanding—diluted	22.0	24.0	27.8	30.8	34.1	29.1	34.3	34.5	34.7	34.9	34.6	35.6	37.6	47.6	50.6	52.6	53.6
Margin Analysis (% of Sales/Revenue)																	
Costs of goods														15%	15%	15%	15%
R&D	-255%	-106%	-242%	-444%	-300%	-248%	-426%	-397%	-499%	-411%	-430%	-486%	-530%	-145%	-80%	-30%	-11%
SG&A	-62%	-67%	-72%	-142%	-94%	-88%	-130%	-118%	-145%	-115%	-126%	-144%	-151%	-40%	-21%	-8%	-3%
Operating Income (loss)	-217%	-72%	-215%	-486%	-293%	-235%	-456%	-414%	-545%	-426%	-456%	-530%	-580%	-168%	-60%	29%	65%
Pretax	-11%	-54%	-224%	-499%	-159%	-199%	-486%	-441%	-576%	-450%	-483%	-557%	-608%	-175%	-63%	28%	65%
Tax Rate												0%	0%	0%	0%	37%	37%
Net Income	-7%	-54%	-224%	-502%	-157%	-199%	-486%	-441%	-576%	-450%	-483%	-557%	-608%	-175%	-63%	18%	41%
Financial Indicator Growth Analysis (YoY%)																	
Total Revenue	-34%	261%	206%	-27%	862%	135%	-39%	-18%	10%	-9%	-17%	0%	0%	299%	97%	185%	200%
R&D	-33%	-45%	172%	196%	76560%	128%	148%	34%	24%	25%	44%	13%	9%	9%	8%	7%	7%
SG&A	-67%	-61%	-62%	731%	-131%	230%	20%	33%	13%	13%	19%	14%	5%	5%	5%	5%	5%
Marketing and sales															25%	15%	5%
Operating Income (Losses)	-48%	-79%	-13%	1550%	-193%	154%	288%	58%	23%	33%	60%	16%	10%	15%	-30%	-240%	565%
Pretax Income	-98%	-91%	-1118%	-199%	-149%	4035%	457%	61%	27%	159%	101%	15%	9%	15%	-29%	-227%	589%
Net Income	-99%	-91%	-1208%	-199%	-148%	6684%	457%	61%	26%	162%	101%	15%	9%	15%	-29%	-180%	589%
EPS	-99%	-94%	-691%	-164%	-133%	5021%	290%	30%	12%	156%	69%	12%	3%	-9%	-33%	-177%	576%

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Source: Bloomberg LP; Company reports; Laidlaw & Company estimates

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Affimed Therapeutics N.V. – Balance Sheet

(€'000)	2012	2013	2014	1Q15	2Q15	3Q15	4Q15E	2015E	1Q16E	2Q16E	3Q16E	4Q16E	2016E	2017E
Assets														
Cash and cash equivalents	4,902	4,151	39,725	37,033	66,319	60,425	75,783	75,783	66,894	59,177	50,052	40,571	40,571	1,862
Short term investments	0	0	0					0					0	
Liqid assets	***4,902***	***4,151***	***39,725***	***37,033***	***66,319***	***60,425***	***75,783***	***75,783***	***66,894***	***59,177***	***50,052***	***40,571***	***40,571***	***1,862***
Preferred stock issuance receivable – related party														
Inventories	121	140	199	201	222	239	256	256	249	257	285	270	270	285
Trade and other receivables	668	1,001	939	1,057	2,320	1,447	1,343	1,343	1,359	1,397	1,388	1,329	1,329	1,365
Total Current Assets	5,691	5,292	40,863	38,291	68,861	62,111	77,382	77,382	68,501	60,832	51,724	42,170	42,170	3,511
Deferred offering cost	0	0	0	-	-	-	-	0					0	
Deferred tax assets	15	16		-	-	-	-	0						
Leasehold improvements and equipment	1,225	1,034	974	929	899	959	903	903	900	925	890	885	885	880
Intangible assets	260	158	72	70	64	61	60	60	60	62	64	66	66	67
Total Assets	**7,191**	**6,500**	**41,909**	**39,290**	**69,824**	**63,131**	**78,345**	**78,345**	**69,461**	**61,819**	**52,678**	**43,121**	**43,121**	**4,458**
Liabilities and Stockholders' Equity														
Income tax payable						35								
Trade and other payables	1,990	3,862	3,759	3,113	4,563	4,879	4,916	4,916	4,906	4,857	4,979	4,911	4,911	5,020
Convertible notes payable - related parties	0	6,196	0	-	-	-	-	0					0	
Borrowings	0	4,800	0	-	-	932	1,054	1,054	1,000	950	900	850	850	960
Accrued expenses	74	82	2,460	1,016	572	122	163	163	172	177	178	183	183	190
Total current liabilities	2,064	14,940	6,219	4,129	5,135	5,968	6,133	6,133	6,077	5,984	6,056	5,944	5,944	6,170
Borrowings	0	0	3,895	4,508	4,337	3,441	2,996	2,996	2,427	3,250	3,250	3,250	3,250	3,015
Preferred Shares	73,467	77,945	0	-	-	-	-	0	-	-	-	-	0	0
Cash settled share based payments	4,784	12,838	0	-	-	-	-	0	-	-	-	-	0	0
Total Liabilities	80,315	105,723	10,114	8,637	9,472	9,409	9,129	9,129	8,505	9,234	9,306	9,194	9,194	9,185
Series A and B preferred stock	0	0	0	-	-	-	-	-					-	-
Preferred stock	0	0	0	-	-	-	-	-					-	-
Common stock	63	63	240	240	299	299	303	303	303	303	303	303	303	304
Additional paid-in capital	469	469	131,544	131,886	166,710	167,372	186,472	186,472	186,472	186,472	186,472	186,472	186,472	188,472
Deficit accumulated during the development stage	(73,631)	(99,730)	(99,989)	(101,473)	(106,657)	(113,949)	(117,559)	(117,559)	(125,819)	(134,190)	(143,403)	(152,848)	(152,848)	(193,502)
Own shares	(25)	(25)												
Total Stockholders' Equity	(73,124)	(99,223)	31,795	30,653	60,352	53,722	69,216	69,216	60,956	52,585	43,372	33,927	33,927	(4,726)
Total Liabilities and Stockholders' Equity	**7,191**	**6,500**	**41,909**	**39,290**	**69,824**	**63,131**	**78,345**	**78,345**	**69,461**	**61,819**	**52,678**	**43,121**	**43,121**	**4,458**

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Source: Bloomberg LP; Company reports; Laidlaw & Company estimates

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Affimed Therapeutics N.V. – Cash Flow Statement

(€'000) Cash Flows From Operating Activities:	2012	2013	2014E	1Q15	2Q15	3Q15	4Q15E	2015E	1Q16E	2Q16E	3Q16E	4Q16E	2016E	2017E
Net income (loss)	(14,314)	(26,099)	(259)	(1,484)	(5,184)	(7,292)	(3,610)	(17,570)	(8,260)	(8,371)	(9,213)	(9,445)	(35,289)	(40,654)
Adjustments to reconcile net loss to net cash used in operating activities:														
Income taxes	(9)	(1)	(166)	0	0	36	20	56	20	20	20	20	80	80
Depreciation and amortization	408	427	441	84	87	69	80	320	80	80	80	80	320	320
Loss from disposal of leasehold improvements	0	24	3	0	781	(781)	0	0	0	0	0	0	0	0
Share based payments	1,918	8,054	(4,891)	342	(643)	1,754	(309)	1,144	(300)	199	(300)	(289)	(690)	(690)
Finance income / costs - net	3,926	10,397	(7,753)	(518)	518	(108)	280	172	(100)	600	370	300	1,170	1,170
Changes in operating assets and liabilities:														
Trade and other receivables	267	(333)	62	(118)	(321)	(69)	104	(404)	(16)	(38)	10	59	14	(37)
Inventories	(44)	(20)	(59)	(2)	(21)	(17)	(19)	(59)	8	(9)	(28)	15	(14)	(15)
Trade and other payables	(798)	1,880	2,275	(2,090)	1,006	(134)	37	(1,181)	(10)	(49)	121	(67)	(5)	108
Interest received	7	9	2	2	0	3	4	9	4	4	4	4	16	16
Paid interest	(6)	(16)	(202)	(140)	(47)	(239)	(196)	(622)	(196)	(196)	(170)	(170)	(732)	(732)
Net Cash from Operating Activities	**(8,645)**	**(5,678)**	**(10,547)**	**(3,924)**	**(3,924)**	**(6,678)**	**(3,609)**	**(18,135)**	**(8,771)**	**(7,760)**	**(9,106)**	**(9,493)**	**(35,130)**	**(40,433)**
Cash flows from investing activities:														
Purchase of intangible assets	(6)	(23)	(45)	(5)	(1)	(4)	(3)	(13)	(7)	(5)	(4)	(5)	(21)	(22)
Purchase of leasehold improvements and equipment	(29)	(139)	(260)	(32)	(50)	(122)	(75)	(279)	(72)	(51)	(51)	(37)	(211)	(240)
Proceeds from sale of equipment	0	5	7	0	0	0	0	0	0	0	0	0	0	0
Net Cash from Investing Activities	**(35)**	**(157)**	**(298)**	**(37)**	**(51)**	**(126)**	**(78)**	**(292)**	**(79)**	**(56)**	**(55)**	**(42)**	**(232)**	**(262)**
Cash Flows From Financing Activities:														
Proceeds from issue of common shares	0	0	43,213	0	33,502	4,022	19,100	56,624	0	0	0	0	0	2,000
Transactions costs related to issue of common shares	0	0	(5,343)	0	0	(3,090)	0	(3,090)	0	0	0	0	0	0
Proceeds from issue of preferred shares	5,417	0	2,999	0	0	0	0	0	0	0	0	0	0	0
Proceeds from convertible debt	4,450	5,100	0	0	0	0	0	0	0	0	0	0	0	0
Transactions costs related to preferred shares and convertible debt	(31)	(16)	0	0	0	0	0	0	0	0	0	0	0	0
Proceeds from borrowings	0	0	4,020	0	0	0	0	0	0	0	0	0	0	0
Proceeds from other borrowings		0	0	0	0	0	0	0	0	0	0	0	0	0
Net Cash Provided by Financing Activities	**9,836**	**5,084**	**44,889**	**0**	**33,502**	**932**	**19,100**	**53,534**	**0**	**0**	**0**	**0**	**0**	**2,000**
Net increase (decrease) in cash	**1,156**	**(751)**	**34,044**	**(3,961)**	**29,527**	**(5,872)**	**15,413**	**35,107**	**(8,850)**	**(7,816)**	**(9,161)**	**(9,535)**	**(35,362)**	**(38,695)**
Cash at beginning of period	3,746	4,902	4,151	39,725	37,033	66,319	60,425	39,725	75,783	66,894	59,177	50,052	75,783	40,571
Exchange-rate related changes of cash and cash equivalents	0	0	1,530	1,269	(241)	(22)	(55)	951	(40)	100	35	55	150	(15)
Cash at end of period	**4,902**	**4,151**	**39,725**	**37,033**	**66,319**	**60,425**	**75,783**	**75,783**	**66,894**	**59,177**	**50,052**	**40,571**	**40,571**	**1,862**

Yale Jen, Ph.D. 212-953-4978

Source: Bloomberg LP; Company reports; Laidlaw & Company estimate

DISCLOSURES:

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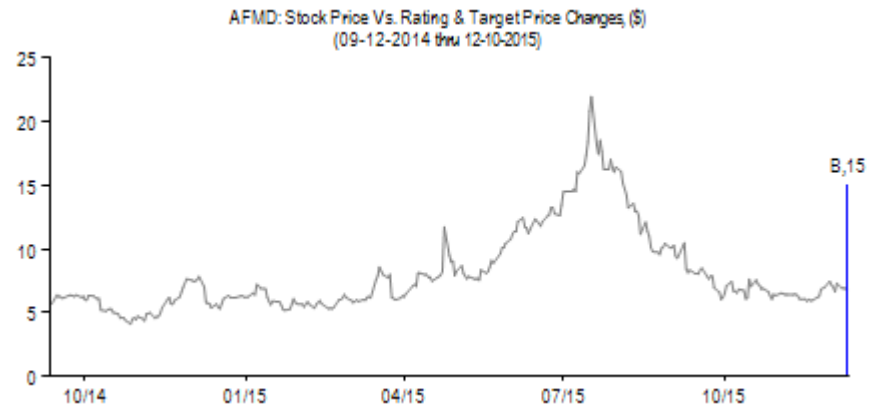
Additional information available upon request.

‡ Laidlaw & Company has received compensation from the subject company for investment banking services in the past 12 months and expects to receive or intends to seek compensation for investment banking services from the company in the next three months.

RATINGS INFORMATION

Rating and Price Target Change History



3 Year Rating Change History

Date	Rating	Closing Price ($)
12/10/2015	Buy (B)	6.87*

3 Year Price Change History

Date	Target Price ($)	Closing Price, ($)
12/10/2015	15.00	6.87*

** Previous Close12/9/2015*

Source: Laidlaw & Company **Created by: Blue-Compass.net**

Laidlaw & Company Rating System*		% of Companies Under Coverage With This Rating	% of Companies for which Laidlaw & Company has performed services for in the last 12 months	
			Investment Banking	Brokerage
Strong Buy (SB)	Expected to significantly outperform the sector over 12 months.	0.00%	0.00%	0.00%
Buy (B)	Expected to outperform the sector average over 12 months.	64.71%	26.47%	2.94%
Hold (H)	Expected returns to be in line with the sector average over 12 months.	0.00%	0.00%	0.00%
Sell (S)	Returns expected to significantly underperform the sector average over 12 months.	0.00%	0.00%	0.00%

ADDITIONAL COMPANIES MENTIONED

Johnson & Johnson (JNJ – Not Rated)
Amgen Inc. (AMGN – Not Rated)
Merck (MRK – Not Rated)
Macrogenics (MGNX – Not Rated)
AstraZeneca (AZN – Not Rated)
Bristol-Myers Squibb (BMY – Not Rated)
NantKwest (NK – Not Rated)
Five Prime Therapeutics (FPRX – Not Rated)
Seattle Genetics (SGEN – Not Rated)
JUNO Therapeutics (JUNO – Not Rated)

Kite Pharma (KITE – Not Rated)
Novartis (NVS – Not Rated)

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