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EQUITY RESEARCH
Company Report

March 3, 2017

Affimed Therapeutics (AFMD - $2.10)

Healthcare/Biotechnology

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

Presentations of *In Vivo* Activities of Several Novel NK- and T-Engaging Bi-Specific Antibodies at the ACCR

AFMD announced the abstracts for the posters to be presented at this year's American Association of Cancer Research (AACR) annual meeting (April 1-5, 2017) which include several novel NK- and T-cell engaging bi-specific antibodies.

- **Details.** AFMD is scheduled to present three posters and one oral presentation at a mini-symposium, at this year's AACR meeting. The subjects of the presentation include AFM13, AFM24, AFM26 and a novel antibody against disease-specific MHC/peptide complexes (MMP1-003). The abstracts of the four posters/oral presentations are included starting on page two of this note.
- **Implications.** Given our overall bullish view on bi-specific antibody with its potential as an effective therapeutic modality, in oncology and inflammatory conditions, we view the AACR presentation a valuable venue to showcase the novel products of this class. AFMD has a strong and dominant position in the development of the novel NK-cell engaging bi-specific antibodies. In addition to the oral presentation at the AACR, which highlights the clinical progress of AFM13, the information gained from AFM24 and AFM26 could be very interesting. These two assets might interest prospective partners, especially BCMA, which is a highly-pursued target for potential multiple myeloma treatment, and most of the competing programs remain in an earlier stage of development. Targeting the tumor-associated MHC/peptide complex (HLA-A*02-binding peptide MMP1-003) certainly is a very novel approach. If this program can advance into clinical studies, it would be a very interesting program to monitor.
- **Action.** We are reiterating our Buy rating and our $15 target price based on peer comparable, probability adjusted m and sum-of-the-parts analyses to reflect the continued execution of pipeline advancements.

Trading Data:

Last Price (3/2/2017)	$2.10
52–Week High (4/25/2016)	$5.00
52–Week Low (12/23/2016)	$1.65
Market Cap. (MM)	$93
Shares Out. (MM)	28.477

Earnings Estimates: (€ per share)

(Dec)	1Q	2Q	3Q	4Q	FY	P/E
FY-17E	-0.33	-0.34	-0.33	-0.35	-1.35	N.A.
FY-16E	-0.25A	-0.24A	-0.31A	-0.31	-1.11	N.A
FY-15A	-0.06	-0.19	-0.24	-0.19	-0.71	N.A.
FY-14A	-1.06	0.03	0.37	0.32	-0.01	N.A

Source: Laidlaw & Company estimates

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- **(3641/14) EGFR/CD16A TandAbs are efficacious NK-cell engagers with favorable biological properties which potently kill EGFR+ tumors with and without Ras mutation**
  Tuesday, Apr 4, 2017, 8:00 AM -12:00 PM, Poster Section 26

  Michael Kluge, Kristina Ellwanger, Uwe Reusch, Ivica Fucek, Michael Weichel, Torsten Haneke, Stefan Knackmus, Joachim Koch, Martin Treder. Affimed GmbH, Heidelberg, Germany

  Constitutive EGFR activation plays an important role in the pathophysiology of various solid cancers, such as colorectal cancer, non-small cell lung cancer or squamous cell carcinomas of the head and neck. Tyrosine kinase inhibitors (TKI) and monoclonal antibodies (mAbs), which interfere with signal transduction and activation of EGFR, are approved for treatment of such cancers. However, intrinsic or acquired resistance to these treatments has been described for many patients. Natural killer cells (NK-cells) are important effectors of innate immunity and NK-cell engagers have shown evidence of improved safety in patients compared to T-cell engagers. To specifically utilize the cytotoxic potential of NK-cells to eliminate EGFR-expressing tumors, we developed tetravalent bispecific EGFR/CD16A TandAbs comprising fully human Fv domains recognizing human and cynomolgus EGFR and CD16A. TandAbs recognizing epitopes in the extracellular domain of EGFR differing from epitopes targeted by other mAbs were characterized. Lead candidate AFM24 shows superior cytotoxicity in terms of ADCC (main mode of action) and reduced inhibition of EGFR-mediated phosphorylation compared to cetuximab. Importantly, inhibition of EGFR-signaling is believed to contribute to skin toxicity caused by therapeutic mAbs and TKI's. AFM24's cytotoxic activity was tested against EGFR+ tumor cell lines including some carrying a Ras mutation, which is a negative prognostic biomarker and renders cells less susceptible to cetuximab or panitumumab. The cetuximab-resistant CRC cell line HCT-116 or the NSCLC cell line A549 (both with Ras mutations) were efficiently killed with EGFR/CD16A TandAbs in vitro. In vivo data in the HCT-116 model indicate anti-tumor efficacy of AFM24, while no efficacy of cetuximab was seen. Importantly, AFM24 does not activate NK-cells without target cell binding and does not bind to any other members of the EGFR family. While binding and cytotoxic efficacy of many therapeutic mAbs are impaired by serum IgG, no substantial change in AFM24's binding affinity to NK-cells was observed in the presence of high concentrations of human IgG. In calcein-release cytotoxicity assays with NK-cells as effectors, we showed that the presence of IgG had only little inhibitory effect on AFM24 efficacy compared to cetuximab. In addition, competition of an anti-CD16 mAb with AFM24 in cytotoxicity assays was substantially lower than with cetuximab. Taken together our data demonstrate that AFM24 is a highly potent human antibody displaying favorable biological properties over existing mAbs. This human/cynomolgus cross-reactive agent is currently in preclinical development to treat EGFR+ malignancies and has the potential to exhibit a favorable side effect profile and reduced toxicity and to overcome resistance to other targeted anti-EGFR therapeutic agents.

- **(3753/9) Identification of antibodies against a novel tumor-associated MHC/peptide-target and generation of highly specific and potent HLA-A*$^{02MMP1-003}$/CD3 TandAbs**
  Tuesday, Apr 4, 2017, 8:00 AM -12:00 PM, Poster Section 30

  Toni Weinschenk[1], Erich Rajkovic[2], Uwe Reusch[2], Michael Weichel[2], Kristina Ellwanger[2], Ivica Fucek[2], Michael Tesar[2], Dominik Hinz[3], Vera Molkenthin[3], Sebastian Bunk[1], Norbert Hilf[1], Oliver Schoor[1], Dominik Maurer[1], Kerstin Mock[1], Carsten Reinhardt[1], Martin Treder[2]. [1]Immatics Biotechnologies GmbH, Tübingen, Germany; [2]Affimed GmbH, Heidelberg, Germany; [3]Abcheck s.r.o., Plzen, Czech Republic

  Tumor-associated antigens for effective and safe T-cell engagement are very limited, leaving a need to open up the therapeutic target space. Targeting disease-specific MHC/peptide complexes with bispecific T-cell-recruiting antibodies is a highly attractive strategy to address this need, but so far, generation of antibodies against these peptides has been reported to be challenging. Immatics' unique target discovery engine XPRESIDENT® holds the promise of identifying novel tumor-associated MHC/peptide complexes by providing direct and quantitative

evidence for their presence on a large collection of primary human tumor and normal tissue specimens. By this approach, MMP1-003, an HLA-A*02-binding peptide originating from matrix metallopeptidase 1 (MMP1), was identified as a promising therapeutic target presented by several tumor types, including colorectal and lung cancer, but absent on normal tissues. These findings are underlined by RNAseq analysis of the source antigen which also points to MMP1 being a highly attractive tumor-associated target. Consequently, a fully human antibody phage display library was screened to identify highly specific single chain antibodies, which were shown to recognize the purified HLA-A*02/MMP1-003-complex in ELISA assays as well as on peptide-pulsed HLA-A*02+ T2 cells. The best candidates were reformatted into bispecific tetravalent TandAbs® through Affimed´s proprietary platform using a human/cyno-cross-reactive CD3-binding domain for T-cell engagement. Specific target recognition was confirmed for the TandAbs in binding and cytotoxicity assays on peptide-pulsed T2 cells. HLA-A*02/peptide-complexes selected from the broad normal tissue immunopeptidome with a high degree of sequence similarity to the HLA-A*02/MMP1-003-complex served as controls to confirm the specificity and hence the low risk of off-target binding. The most promising candidates were tested on a panel of endogenously target-expressing cancer cell lines covering MMP1 +/- and HLA-A*02 +/- expression profiles, as well as the source proteins for the most closely related control peptides. The lead TandAb showed excellent target specificity across a wide range of peptide-pulsed and endogenously expressing cell lines as well as potent cytotoxicity with picomolar EC50. In summary, we have identified a tumor-associated MMP1-derived peptide in an HLA-A*02 context by exploiting the knowledge of tumor and healthy tissue immunopeptidomes using XPRESIDENT®. Overcoming the existing barrier of developing antibodies targeting specific MHC/peptide complexes, we generated and characterized highly specific and potent T-cell-recruiting TandAbs. These hold the potential to open up the therapeutic target space for T-cell engagement by providing access to intracellular proteins that are presented in a disease-specific manner as MHC/peptide complexes.

- **(5671/25) AFM26 - A novel CD16A-directed bispecific TandAb targeting BCMA for multiple myeloma**
  Wednesday, Apr 5, 2017, 8:00 AM -12:00 PM, Poster Section 28

  Thorsten Gantke, Uwe Reusch, Kristina Ellwanger, Ivica Fucek, Michael Weichel, Martin Treder. Affimed GmbH, Heidelberg, Germany

  Multiple myeloma (MM) is the second most common haematological cancer and is characterized by the accumulation of neoplastic plasma cells in the bone marrow and production of high levels of monoclonal immunoglobulin (M-protein). While historically considered incurable, recent approvals and ongoing clinical trials with monoclonal antibodies (mAbs) targeting surface antigens promise greatly improved outcomes and have heralded a new era of MM treatment in which immunotherapies are expected to take center stage. However, an unmet need remains as patients eventually relapse and/or become refractory to currently available treatments. Consequently, novel immunotherapeutic approaches are needed to provide improved treatment options to MM patients. Among the currently explored targets, B-cell maturation antigen (BCMA, CD269) is considered to be particularly attractive due to its limited expression on healthy tissues and almost universal expression on myeloma cells in the majority of patients.

  Natural killer (NK) cells are cytotoxic effectors of the innate immune system capable of rapidly eradicating infected and transformed cells. The cytolytic activity of NK-cells can be used therapeutically to induce tumor cell lysis by direct engagement of the activating receptor CD16A (FcγRIIIa) using mAbs (ADCC). Despite similar mechanisms of target cell lysis, activation of NK-cells is not associated with the systemic symptoms of high level cytokine release as seen with direct T-cell engagement. Hence, it is considered a potent immunotherapeutic approach with reduced toxicity and a well-manageable safety profile. NK-cells readily infiltrate bone marrow and are thought to contribute to the efficacy of current myeloma treatments. Therefore, redirecting NK-cell cytotoxicity to malignant plasma cells appears to be a suitable therapeutic approach for MM.

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Here we describe the characterization of AFM26, a novel tetravalent bispecific tandem diabody (TandAb) that specifically targets BCMA and CD16A with high affinity and induces potent and efficacious myeloma cell lysis. AFM26 incorporates an affinity-improved anti-CD16A domain and interacts bivalently with NK-cells, resulting in high avidity and prolonged cell surface retention that is largely unaffected by the presence of polyclonal IgG. Notably, AFM26 potently induces NK-cell-mediated in vitro lysis of target cells expressing low levels of BCMA at low effector:target ratios, even in presence of polyclonal IgG. This may suggest that AFM26, in contrast to classical mAbs, retains ADCC activity at low antibody concentrations in presence of serum IgG and despite high levels of IgG M-protein occurring in about half of MM patients. AFM26 exhibits high protein stability, full cross-reactivity with cynomolgus antigens (BCMA and CD16A) and does not bind APRIL and TACI, two functionally related receptors. These data suggest that TandAb AFM26 is a promising and highly potent drug candidate for MM treatment.

- **(2997) The tetravalent bispecific antibody AFM13 engages and primes innate immune cells for anti-cancer immunity**
  Monday, Apr 3, 2017, 4:05 - 4:20 PM, Room 152, Level 1

  Jens Pahl[1], Joachim Koch[2], Uwe Reusch[2], Thorsten Gantke[2], Adelheid Cerwenka[1], Martin Treder[2]. [1]German Cancer Research Center, Heidelberg, Germany; [2]Affimed GmbH, Heidelberg, Germany

  AFM13 is a tetravalent bispecific antibody with bivalent binding to both CD30 and CD16A. It has been shown to engage NK-cells through CD16A with high affinity and specificity, resulting in strong NK-cell cytotoxicity, and is currently being tested in Phase 2 monotherapy and in combination with pembrolizumab in Phase 1b clinical trials. We have previously shown that AFM13-dependent activation of NK-cell cytotoxicity towards CD30+ tumor cells is more pronounced than that of anti-CD30 mAbs. In addition, AFM13 enhances NK-cell sensitivity to low doses of IL-2 and IL-15, leading to an increased NK-cell proliferative potential. Here, we have extended the panel of phenotypic markers on NK-cells that are modulated after exposure to CD30+ tumor cells in the presence of AFM13. Targeting some of these markers may enable the development of novel combination therapies. Moreover, we have analyzed the kinetics of NK-cell responses to AFM13 exposure. Even though short-term exposure to AFM13 significantly enhanced NK-cell cytotoxicity, long-term exposure led to a partial, transient functionally exhausted phenotype in vitro, which could be fully restored by cytokine stimulation for several days in the absence of AFM13. Importantly, these recovered cells displayed high cytotoxicity towards CD30+ target cells in the presence of AFM13. Interestingly, the transient NK-cell exhaustion was not related to the expression of typical exhaustion markers or insufficient levels of perforin and granzyme. These data may warrant the development of novel metronomic application regimens of AFM13. Further studies imply that immune cells other than NK-cells are able to inhibit growth of CD30+ tumor cells in an AFM13-dependent manner. This appears to be strictly dependent on CD16A and a specific cytokine milieu. Taken together, AFM13 specifically enhances the cytotoxic, proliferative and cytokine-producing potential of NK-cells, parameters that can be utilized to monitor NK-cell responses during AFM13 therapy. Moreover, based on our data, engagement of CD16A+ cells to the tumor site might enable several innate immune effector functions within the tumor microenvironment for synergistic anti-tumor activity.

  Source: 2017 AACR website

Anticipated milestones in 2017 and beyond

Product	Indication	Event	Timing	Importance
AFM13	Hodgkin's lymphoma (r/r)	Potentially report Phase I CPI combination safety Phase I study results	**4Q16/1Q17**	***
		Potentially report Phase II CPI combination Phase II study results	**2017/2018**	****
	$CD30^+$ lymphoma	Potentially to start Phase IIa study (or in T cell lymphoma)	**2017**	***
AFM11	Non-Hodgkin's lymphoma (NHL)	Potentially report Phase I study timeline	**1H17**	****
	Acute lymphoblastic leukemia (ALL)	Potentially to report Phase I study results	**4Q17**	***
AFM24	Solid tumors	Potentially update progress	**1H17**	***
AMV564	Acute myeloid leukemia (AML)	Potentially start Phase I study	**2017**	***

***** / ***** 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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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 ~€67MM cash, 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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Figure 1: Income Statement

Affimed N.V. – Income Statement

(€'MM)	2014	2015	1Q16	2Q16	3Q16	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	7.6	1.9	2.1	0.9	1.0	5.9	3.2	3.2	3.2	3.2	3.2	3.2
Other revenue	0.4	0.7	0.1	0.0	0.0	0.1	0.2	0.2	0.2	0.2	0.2	0.2	0.2
Total revenue	3.8	8.2	2.0	2.1	1.0	1.1	6.2	3.4	3.4	25.2	53.3	159	484
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)	(22.0)	(7.1)	(8.6)	(8.8)	(9.2)	(33.7)	(39.0)	(42.5)	(46.3)	(50.0)	(53.5)	(57.2)
General and administrative	(2.3)	(7.5)	(2.1)	(2.0)	(2.2)	(2.3)	(8.5)	(9.4)	(9.9)	(10.4)	(10.9)	(11.5)	(12.0)
Marketing and sales										(21.0)	(26.3)	(30.2)	(31.7)
Total Operating Expenses	(11.9)	(29.6)	(9.2)	(10.6)	(10.9)	(11.5)	(42.2)	(48.4)	(52.4)	(77.7)	(87.2)	(95.1)	(101.0)
Operating Incomes (losses)	(8.2)	(21.3)	(7.1)	(8.5)	(10.0)	(10.4)	(36.0)	(45.0)	(49.0)	(55.8)	(41.3)	40.6	311.2
Finance income / (costs) - net	7.8	1.1	(1.3)	0.5	(0.3)	(0.3)	(1.5)	(1.4)	(1.4)	(1.4)	(1.4)	(1.4)	(1.4)
Loss before tax	(0.4)	(20.2)	(8.5)	(8.0)	(10.3)	(10.7)	(37.5)	(46.4)	(50.4)	(57.2)	(42.8)	39.1	309.7
Tax	0.2	0.0	(0.0)	(0.0)	-	-	0.0	0.0	0.0	0.0	0.0	(14.5)	(114.6)
Net Income (Loss)	(0.3)	(20.2)	(8.5)	(8.0)	(10.3)	(10.7)	(37.5)	(46.4)	(50.4)	(57.2)	(42.8)	24.7	195.1
Net Income (Loss) Applicable to Common Shareholders	(0.3)	(20.2)	(8.5)	(8.0)	(10.3)	(10.7)	(37.5)	(46.4)	(50.4)	(57.2)	(42.8)	24.7	195.1
Net Income (Loss) Applicable to Common Shareholders ($)	(0.3)	(21.8)	(9.0)	(8.9)	(11.0)	(11.3)	(39.8)	(49.4)	(53.6)	(60.8)	(45.5)	26.2	207.6
Net Earnings (Losses) Per Share—Basic	(€ 0.01)	(€ 0.71)	(€ 0.25)	(€ 0.24)	(€ 0.31)	(€ 0.31)	(€ 1.11)	(€ 1.35)	(€ 1.38)	(€ 1.23)	(€ 0.87)	€ 0.48	€ 3.72
Net Earnings (Losses) Per Share—Diluted	(€ 0.01)	(€ 0.71)	(€ 0.25)	(€ 0.24)	(€ 0.31)	(€ 0.31)	(€ 1.11)	(€ 1.35)	(€ 1.38)	(€ 1.23)	(€ 0.87)	€ 0.48	€ 3.72
Net Earnings (Losses) Per Share—Basic/diluted ($)	($0.01)	($0.76)	($0.26)	($0.27)	($0.32)	($0.33)	($1.18)	($1.44)	($1.47)	($1.31)	($0.92)	$0.51	$3.96
Shares outstanding—basic	22.0	29.1	34.3	33.5	33.7	33.9	33.8	34.4	36.4	46.4	49.4	51.4	52.4
Shares outstanding—diluted	22.0	29.1	34.3	33.5	33.7	33.9	33.8	34.4	36.4	46.4	49.4	51.4	52.4
Margin Analysis (% of Sales/Revenue)													
Costs of goods										15%	15%	15%	15%
R&D	-255%	-268%	-350%	-409%	-915%	-836%	-544%	-1146%	-1249%	-184%	-94%	-34%	-12%
SG&A	-62%	-92%	-104%	-93%	-228%	-206%	-138%	-278%	-292%	-41%	-21%	-7%	-2%
Operating Income (loss)	-217%	-260%	-353%	-403%	-1043%	-942%	-582%	-1323%	-1440%	-221%	-78%	25%	64%
Pretax	-11%	-246%	-418%	-381%	-1076%	-970%	-606%	-1365%	-1482%	-227%	-80%	25%	64%
Tax Rate									0%	0%	0%	37%	37%
Net Income	-7%	-246%	-418%	-381%	-1076%	-970%	-605%	-1365%	-1482%	-227%	-80%	16%	40%
Financial Indicator Growth Analysis (YoY%)													
Total Revenue	-34%	118%	-27%	-9%	-34%	-34%	-25%	-45%	0%	641%	111%	198%	204%
R&D	-33%	129%	142%	54%	36%	31%	53%	16%	9%	9%	8%	7%	7%
SG&A	-67%	222%	13%	17%	5%	16%	13%	11%	5%	5%	5%	5%	5%
Marketing and sales											25%	15%	5%
Operating Income (Losses)	-48%	161%	257%	71%	41%	42%	69%	25%	9%	14%	-26%	-198%	667%
Pretax Income	-98%	4662%	470%	55%	42%	69%	85%	24%	9%	13%	-25%	-192%	692%
Net Income	-99%	7713%	470%	55%	41%	70%	85%	24%	9%	13%	-25%	-158%	692%
EPS	-99%	5931%	299%	28%	29%	66%	56%	22%	3%	-11%	-30%	-155%	676%

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

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DISCLOSURES:

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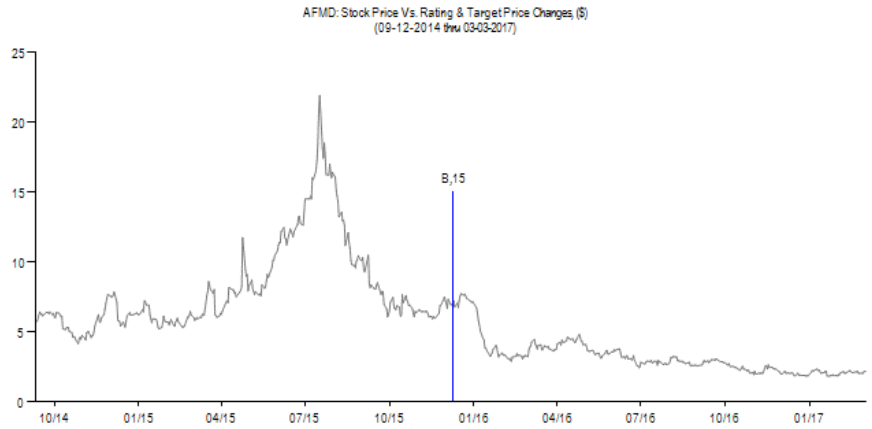
Additional information available upon request.

Laidlaw & Co (UK) Ltd. has not provided any investment banking services for the company (ies) mentioned in this report over the last 12 months.

RATINGS INFORMATION

Rating and Price Target Change History



3 Year Rating Change History

Date	Rating	Closing Price ($)
12/10/2...	Buy (B)	7.19

3 Year Price Change History

Date	Target Price ($)	Closing Price, ($)
12/10/2...	15.00	7.19

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.	2.44%	2.44%	0.00%
Buy (B)	Expected to outperform the sector average over 12 months.	60.98%	26.83%	2.44%
Hold (H)	Expected returns to be in line with the sector average over 12 months.	2.44%	0.00%	0.00%
Sell (S)	Returns expected to significantly underperform the sector average over 12 months.	4.88%	0.00%	0.00%

ADDITIONAL COMPANIES MENTIONED

ADDITIONAL DISCLOSURES

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