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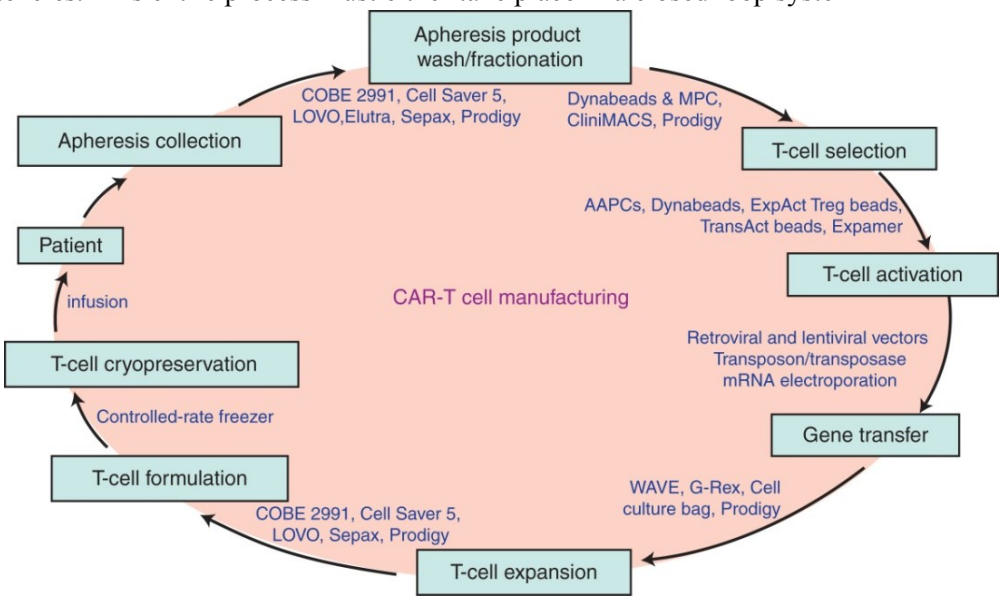
AFM11- Threading the Needle Between CAR-T and Blincyto

Report Amended 8/29/18 to reflect Affimed's announced deal with Roche/Genetech (See last paragraph of this report).

The most recent generation of immune therapies which have utilized T-cells have generated some remarkable responses in patients, which just a decade ago, were left with no options. These therapies demonstrate how redirection of T-cells can be just as, if not more so potent than the blunt tools of chemotherapy. All of these advances would not be possible without the advent of single-chain variable fragment (scFv) domains. These are isolated from parent antibodies, and consist purely of the recognition portion. Accordingly, they are much smaller than their parents which enables them to penetrate into tumor masses that would typically be inaccessible to full-length constructs.

A Chimeric Antigen Receptor (CAR) enabled T-cell consists of a scFv recognition domain fused to the CD3ζ chain for signaling inside the T-cell. Intracellularly, activation of the recognition domain is often designed to trigger a metabolic response through CD28 or CD137 (4-1BB) as is the case in the second generation designs [1]. This 2nd generation of CAR-T cells have garnered recent approvals by the FDA, including Gilead's KTE-C19 (Axicabtagene Ciloleucel, marketed as Yescarta)[2] and Novartis's CTL019 (Tisagenlecleucel, marketed as Kymriah) [3]. The administration difficulties and costs of these treatments have however proven burdensome to care providers. Kymriah for example is marketed as a treatment that costs 475,000 which is difficult enough [4]. But when taking into account the totality of the process and monitoring required as described below, costs can rise towards a million [5].

In the case of autologous therapy (derived from a patient's own cells), the subject undergoes leukaphersis or a donation of white blood cells provided that they have a sufficient population. In certain cases, a CXCR4 antagonist such as Plerixafor or a Granulocyte colony-stimulating factor (G-CSF) can be used to boost white blood cell populations in depleted patients [6]. These cells are shipped out to a manufacturing center where T-cell populations are purified from the sample, a viral vector (retro- or lenti-) containing the code for the CAR is introduced to the T-cells, the population is expanded and the T-cells are purified again in order to avoid inconsistencies. This entire process must either take place in a closed loop system or within an expensive cleanroom to avoid any contaminants that could be detrimental. While some companies are attempting to develop closed loop systems, most manufacturers use cleanrooms [7]. These cells are then shipped back on ice to the treatment center. As

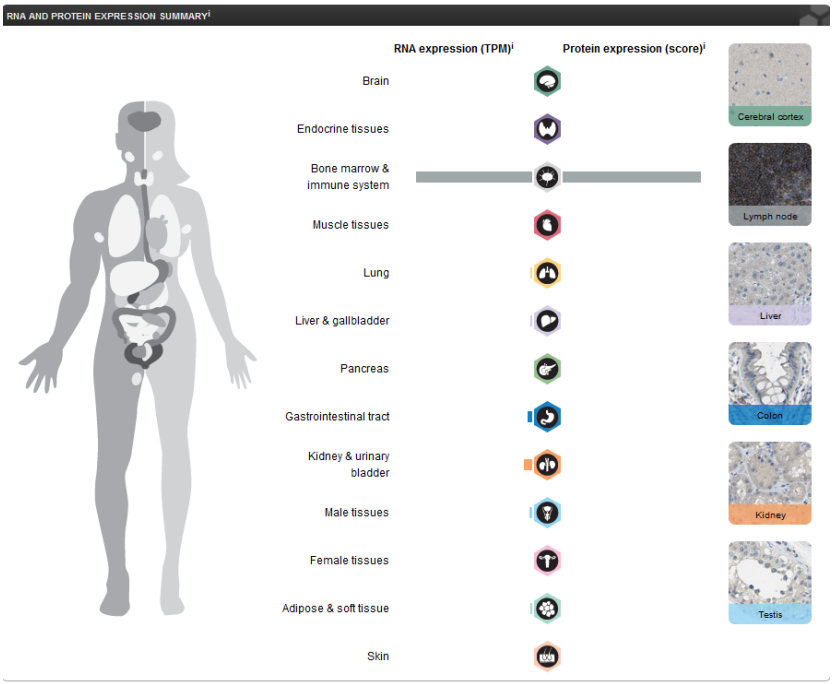


you can imagine this is a complicated logistics chain in itself. However the work for the center has just begun at this point.

In order to prepare a patient for treatment, preconditioning regimens such cytarabine and fludarabine are used to lymphodeplete. This is required in order to prevent rejection of the to-be transfused CAR-T cells, but renders these patients susceptible to infection so significant caution is required in handling of these patients. The modified T-cells are the administered back into the patient where they will need to be under continuous monitoring for at least week [8]. During this time a patient is at serious risk of developing a number of toxicities.

The primary concerns for immunological toxicities are macrophage activation syndrome (MAS) and cytokine release syndrome (CRS) [9]. Both of which are typically hallmarked by excessive levels of the inflammatory markers IL-6 and IFN-gamma. Even in milder immune reactions, the presence of a fever is common place. Neurological toxicities are the second most common adverse events which show up in these patients, notably encephalopathy consisting of speech disorders, aphasia, and tremors [10-12]. Although these neurological toxicities can show up without the presence of CRS, they do seem to require at least a fever at one point during the course of treatment.

At the Memorial Sloan Kettering Cancer Center (MSKCC), researchers sought to understand the mechanisms of this neurological toxicity. They found that aphasia had occurred in 21 of 22 patients with a neurological severe adverse event (sAE). In addition all 33 patients in the study who developed neurological issues had at least a grade 1 CRS accompanied by a fever at some point but not necessarily at the same time as the CRS (8 without CRS, and 5 after fever alone). The median onset of severe neurotoxicity from the beginning of CRS was around 8 days (ranging from 1-11). One rationale for the correlation here is the potential presence of CD19+ cells in the brain [10], despite the fact that initial work didn't find evidence of CD19 mRNA transcripts (see right from the human protein atlas). It was discovered that in some patients, there is the presence of CD19+ cells in the left inferior frontal gyrus (the area of the brain responsible for speech). Therefore toxicities such as aphasia would be consistent with this finding.



It would require some level of disruption of the blood brain barrier (BBB) in order to traffic anti-CD19 CAR-T cells into the brain where they may recognize these CD19+ neurons and set off a secondary, sometimes fatal toxic effect. Consistent with this notion is the well documented activation of the

endothelium which correlates with neural toxicities and high expression of BBB permeable cytokines like IL-6. In response to neural inflammation, a common chemoattractant, CXCL13, is up-regulated on the endothelium of the BBB [13]. This signaling molecule is effective at recruiting cells which express the ligand CXCR5.

Interestingly enough, CXCR5 is expressed both on CD4+ T-cells [14] and B-lineage cancers [15,16]. And it should be further noted that stanard manufacturing for CAR-T is to transfect both CD8+ effector cells as well as CD4+ regulatory cells with the CAR domain. Interestingly, the presence of CAR-T cells and target cells can be seen in the cerebrospinal fluid (CSF). This is accomidated by significant elevations of IL-6 and IFN-gamma in the CSF over serum (190x and 20x higher in one patient) [17,18]. With younger populations, rates of neurotoxic events appeared to be higher than in elderly patients, this is consistent with the CXCR5 hypothesis, given that expression of the ligand on B-cell population drops with age. In summary, it is theorized that the CRS produced by CAR-T therapy, even at low grades can create trafficking to the brain by both CD4+ CAR-T cells and potentially B-ALL cells through binding to the endothelium which expresses "sticky" molecules. Once inside the CSF, the presence of CD19+ cells on some patients (this expression pattern isnt fully understood yet), but not all has led to a heterogeneous toxicity rate where some experience grade 4+ events such as the JCAR015 trial and others more transient.

Management for these toxicities at treatment centers is challenging. Typical response to CRS is via the anti-IL6 antibody tocilizumab, but researchers are finding this approach insufficient given the molecule does not effectively penetrate the BBB. Since the majority of the IL6 expression is in the CSF, tocilizumab alone may not be enough and is often combined with corticosteroids [19]. This of course can dampen the effect of CAR-T therapy itself as well as increase odds of infection like sepsis. This management approach must be used with caution. Further, management of CRS in this way does not seem to alleviate neurologic symptoms per the MKSCC study.

"Six (18%) of 33 patients with neurotoxicity received tocilizumab alone; 14 (42%) received tocilizumab plus corticosteroid; 4 (12%) received corticosteroid alone; and 9 (27%) received neither tocilizumab nor corticosteroid. In the 16 patients with severe neurotoxicity who received tocilizumab, 9 (56.3%) had peak neurotoxicity after the first dose of tocilizumab, whereas 7 (43.7%) had peak neurotoxicity prior to or on the day of tocilizumab administration. Therefore, neurotoxicity did not respond to tocilizumab administration in most patients. The time from first tocilizumab and/or steroid dose to resolution of neurotoxicity (median, 9 days; range, 4–21 days) was longer than the time to resolution of CRS (median, 1 day; range, 0–3 days; $P < 0.001$)"

In all management approaches to the after-effects of CAR-T are incomplete, expensive, and potentially an unviable approach in its current form. Once inside the body there is very little control a physician has over how the T-cells respond and the ensuing CRS storm. Below is a publically available copy of a typical bill one might encounter from a hospital which administers Kymriah. The additional costs for managing the treatment from end to end, especially one which encounters a major CRS event (showing up in around half of b-ALL patients [20] and 26% of NHL [21] for Kymriah). It's easy to see that reimbursements are going to be an extremely difficult process and simplified streamlined approaches are desperately needed.

Children's Mercy
KANSAS CITY
2401 Gillham Road
Kansas City, Missouri 64108
(816) 234-3000

Date: June 22, 2018
Patient: GM
MRN: 1924241
Dx C91.00 ALL not in remission
Service: Kymriah Infusion

Estimated Charges (US$)	#	Hospital Fees	Physician Fees	Total
Consultations				
Follow Up: 99214, G0463	16	$306.00	$228.00	$8,544.00
Prolonged E&M: 99354	1		$253.00	$253.00
Prolonged E&M: each addl 30 min: 99355	1		$246.00	$246.00
Follow Up: 99215, G0463	1	$306.00	$307.00	$613.00
Procedural-related activity				
BMA/LP: 01112, 655000H02, 162180000, 62270, 712872723, 38220, 85097, 85060, anesthesia meds	2	$5,000.00		$10,000.00
INS PICC Age 5 yrs or older: 36569	1	$2,923.00		$2,923.00
Additional LP if needed: 62270	1	$671.00		$671.00
Chemotherapy Drugs				
IT Chemotherapy: 913035000	1	$107.92		$107.92
Chemo into CNS: 96450	1	$713.00		$713.00
Ifosfamide: 913030690	5	$1,300.00		$6,500.00
Etoposide: 905039500	5	$412.00		$2,060.00
LD Chemo Fludarabine: 906039550	4	$1,169.42		$4,677.68
LD Chemo Cyclophosphamide: 903118000	2	$3,091.07		$6,182.14
LD Chemo IV Inf: 96413	4	$686.00		$2,744.00
LD Chemo IV Push: 96411	2	$315.00		$630.00
IV Fluid for LD Chemo: 900500000	2	$80.07		$160.14
Antimetics LD Chemo: 904063000, 912067500, 907025500	4	$507.13		$2,028.52
NS: 919031000 & 9190300000	1	$167.93		$167.93
Premeds: 904064500, 901001555	1	$52.79		$52.79
Diagnostic Puch: 96374BMT	1	$200.00		$200.00
IV Hydration: 96361	1	$258.00		$258.00
Pain medication: 915017500	10	$18.34		$183.40
Blood/Platelet Products				
Type & Cross: 86900H000	6	$33.00		$198.00
86901H000	6	$33.00		$198.00
86920H000	6	$519.00		$3,114.00
86885H001	6	$139.00		$834.00
Transfusion Charge: 36340	6	$1,752.00		$10,512.00
Blood P9040H5BL and P9040H5PS (outpt)	6	$1,839.00		$11,034.00
Platelets P9037 (outpt)	6	$2,421.00		$14,526.00
Transfusion premeds: 904063000, 901005000	12	$4.40		$52.80
Labs/Blood work				
CBC/Diff: 85025	36	$85.00		$3,060.00
Manual Diff: 85007	36	$38.00		$1,368.00
BMP: 80048	36	$93.00		$3,348.00
LFT: 80076	25	$90.00		$2,250.00
GGT: 82977	2	$79.00		$158.00
LDH: 83615	10	$196.00		$1,960.00
PT: 85610	15	$43.00		$645.00
PTT: 85525	15	$130.00		$1,950.00
CRP: 86140	4	$38.00		$152.00
Ferritin: 82728	6	$150.00		$900.00
Fibrinogen: 85384	15	$93.00		$1,395.00
Magnesium: 83735	15	$74.00		$1,110.00
Phosphates: 84100	15	$52.00		$780.00
IGG: 82784	2	$64.00		$128.00

Estimated Charges (US$)	#	Hospital Fees	Physician Fees	Total
Triglycerides: 84478	3	$63.00		$189.00
UA: 81003	5	$25.00		$125.00
Uric Acid: 84550	15	$50.00		$750.00
Blood cultures: 87040	6	$128.00		$768.00
Reticulocycte: 85046	2	$61.00		$122.00
CSF Cell count & Interp: 89050	3	$52.00		$156.00
CSF Cell count & Interp: 88108	3	$199.00		$597.00
Glucose CSF: 82945	3	$43.00		$129.00
Protein CSF: 84157	3	$40.00		$120.00
Flow Cytometry: 88184H000, 88184H002, 88184H009, 88185H015, 88185H006, 88185H022, 88187H001, 88188H001, LIWE88188	2	$3,000.00		$6,000.00
Cytogenetics DNA probe: 88271H007	1	$206.00		$206.00
Cytogenetics 100-300: 88275H000	1	$331.00		$331.00
Cytogenetics FISH ONLY PROCESSING: 88299H003	1	$278.00		$278.00
Tissue Culture Bone Marrow: 88237H000	1	$233.00		$233.00
Inpatient Stays				
Inpatient Treatment 060500100	5	$11,778.00		$58,890.00
Inpatient Treatment (ancillary)	1	$15,400.00		$15,400.00
Inpatient CRS event 041001310	6	$11,126.00		$66,756.00
Inpatient CRS event 005000310	10	$9,874.00		$98,740.00
Inpatient CRS event (ancillary)	1	$113,628.00		$113,628.00
Total for (Hospital and Physicians)				**$473,006.32**
Total for Kymriah Q2040/920500000				$475,000.00
Grant Total				$948,006.32

This estimate does not represent a package price, or the actual costs for diagnostic or medical care. The actual costs of care cannot be determined until services have been provided. It is the responsibility of each family to ensure that all medical services are paid in advance of service.

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Unlike CAR-T, bispecifics like Blincyto do not require genetic modification of T-cells but instead consist of a second scFv domain which binds to the CD3 receptor on T-cells. Therefore, the conditioning and pretreatment steps described above for CAR-T do not apply to Blincyto, making it far more cost effective option for these patients and easier for treatment centers to adopt. This in part explains the continued commercial ramp (+40% yoy per the latest Amgen earnings release [22]) of the therapy in the face of seemingly more efficacious CAR-T approaches that have been launched in the past year. Blincyto reports around a 43% CR rate in b-ALL patients per the package insert when compared to 83% for Kymriah. However unlike Kymriah's nearly 50% CRS rate in the setting, Blincyto has reported grade 4 CRS in two out of 36 patients (6%) and grade 3 CRS in three out of 189 patients (2%) [23,24]. This was in part due to step-wise dosing in combination with dexamethasone making it not exactly ideal (three deaths occured in a 189 patient due to infections).

However the main reason for the discrepancy in CRS events is the difference in mechanism by which these therapeutics work. CAR-T Cells have been shown to expand up to 1000-fold following readministration, consisting of 20% circulating lymphocytes [25], whereas Blincyto increases T-cell populations by 2-4 fold. CAR-T cells generally do not re-engage the targets once activated so rely on

sheer copy numbers (driven by the internal metabolic domain), whereas bispecifics can induce serial killing [26]. This serial killing effect is largely driven by the fact that the CD19 domain on the antibody has a much higher binding affinity than the CD3 domain. Blincyto seems to act like a big red flag on any cells expressing CD19 (which consist mostly of B-cells) for T-cells to attack.

Neurological toxicities of severe grades appear to be similar to CAR-T numbers however. In this instance such toxicity is not driven by the presence of CRS which is far less common with Blincyto, but rather the size of the molecule. Blincyto is a molecule with a molecular weight of 55kDa, smaller than the estimated 70kDa threshold for a molecule to not cross the BBB. Seeing as there is evidence of CD19 expression in the brain combined with significant overlap in the nature of CNS toxicities with CAR-T (eg. aphasia), this provides ample evidence that such toxicity is driven by on-target but off-tumor effects in the language processing center of the brain.

The small size of Blincyto also makes continual infusion a requirement, as the half life of the molecule is estimated as just shy of two hours. This antibody is small enough to not only cross the BBB but also to be filtered out by the kidneys and pass into urine. Attempts to do a non-continual infusion once, twice or thrice weekly in the context of NHL led to premature termination due to neurologic and cytokine toxicities before reaching efficacy [27]. A subsequent study approached treatment as a continual infusion in a stepwise manner from 5-60μg/m^2/day, which did not lead to discontinuations but a 22% grade 3 neural toxicity rate was observed at the target dose. In a follow-on phase 2 in DLBCL, a study where patients were exposed to 9-28-112 μg a day with weekly dose increases in continuous infusion with dexamethasone, we again saw a 22% grade 3 neurological adverse event rate and an overall 2/3 of patients experienced a CNS toxicity of any grade. It appears the more aggressive dosing schedule with steroid use did not improve efficacy rates over the prior study, demonstrating a CR rate of 19% compared to 37% [28].

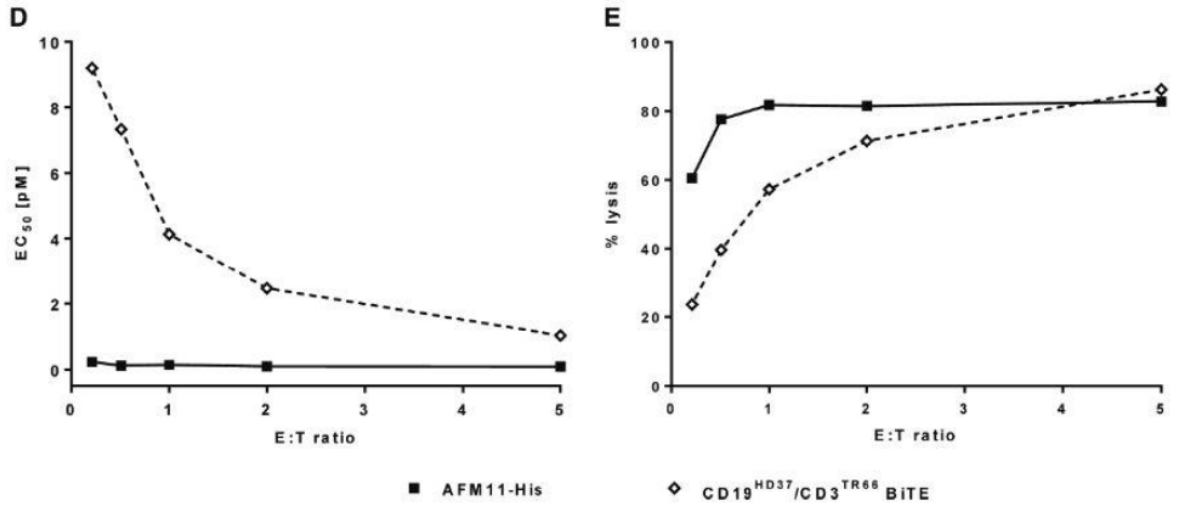
Affimed seeks to build upon the collection of these findings with AFM11. The company has designed the molecule to have 2 binding sites for each CD3 and CD19, increasing binding affinity for both while making the molecule almost twice as large. The binding affinities of AFM11 for CD19 and CD3 are approximately 5-fold and 100-fold higher, respectively, than those of Blincyto. The estimated molecular weight for AFM11 is 105kDa meaning it cannot cross an in-tact BBB nor will it be filtered by the kidneys easily. As such, the estimated half life of the molecule is closer to 19hrs, allowing for easier administration.

Other attempts at improving upon the profile of Blincyto include the Macrogenics/Janssen program for Duvortuxizumab, another CD3/CD19 bispecific molecule based. Like Blincyto it also is a relatively small molecule, but does have a much longer (1 week) half life. The program was terminated early due to the advent of CNS toxicities that were claimed to be similar to other CD19 approaches [30]. Seeing as the efficacy couldn't stand up to CAR-T, yet CNS toxicities were still present, we feel there continues to be an opportunity for the Affimed approach in this space.

Initially a low CD3 binding affinity (a KD of 10^{-7} M and higher) was suggested by Micromet as a requirement to avoid off target T-cell activation [26] and that high affinity bivalent binding (a KD of 10^{-8} M and lower) would lead to non-specific activation. However this has been later disproven, and non-specific triggering of T-cells requires the immobilization of the antibody in question by FcgR+ cells. Antibodies

like AFM11 which lack an Fc domain, therefore wouldn't need to worry about this issue and is reflected in the company's preclinical modelling data [29].

The higher affinity for CD3 on AFM11 over Blincyto gives Affimed the advantage of being able to induce responses with extremely low effector: target ratios, which is more characteristic of the disease states in heavily advanced patients. This may increase depth of response, without giving cells that have low copy numbers of CD19 a chance to repopulate. Despite an increased affinity towards T-cells, the preclinical work suggests that T-cells activated by AFM11, like Blincyto can induce serial killing of target cells. However it will be interesting to see if, unlike Blincyto, the AFM11 construct can actually create a stronger synapse with a give T-cell and this serial killing effect can almost have a CAR-T like effect without induction of CRS side effects.



The key for AFM11 will be to find the correct dosing where efficacy can be achieved, yet no major induction of CRS symptoms are observed. If the BBB is disrupted by large serum increases in cytokines, we may see enough of an opening for the molecule to cross into the CNS. This is an extremely fine needle to thread, and accordingly Affimed re-did the IND application for the drug following a FDA meeting in October of 2015. This new structure delayed the development of AFM11 but created a much more incremental dose escalation, with one patient per dose level in the nano range. Once the minimum efficacious dose is reached, the study converts over to a standard 3+3 dose escalation design until maximum tolerated dose is reached. If the dose window is wide enough, we may be able to see a deeper response than Blincyto, especially in NHL where there is significant room for improvement, with less toxicities (no CNS issues and much more dampened CRS) than we see with CAR-T.

Encouragingly, a different molecule AMV564 which was licensed by Affimed to Amphivena (18% ownership stake by AFMD) has shown efficacy without major toxicity. AMV564 is also based on CD3 engagement but looks to target CD33+ cells, such as those in AML blasts and MDSCs. CD33 is heavily expressed throughout the body and can provide some insight on the level of CRS we may see in the AFM11 studies. Reductions in bone marrow blasts were seen in 6 out of 9 evaluable patients at the low end of the dose range, yet no CRS or treatment related sAEs were observed in the study [31]. "The pharmacokinetics are unprecedented with a gradual rise to steady state drug levels that may help mitigate cytokine release syndrome," according to the lead presenter of the AMV564 data at EHA, Dr.

Westervelt professor of medicine at Washington University in St Louis. “The 0%, 30-day mortality rate in this high-risk population of AML patients is extremely encouraging, and we are seeing evidence of anti-leukemic activity even at very low doses.” [32]

Per clinical trials the NHL trial has a primary completion of August 2018 and a final completion in September. We view any CR rate observed in the NHL trial above 50% in the target dose range as a success so long as this is accompanied by a similarly low CRS rate as we see in Blincyto. Lack of requirement for steroid use would be a significant value-add to the treatment regimen, not only improving efficacy, but also lowering costs and toxicity burden. If this trial proves out, we feel that Amgen would be strongly interested given that AFM11 can expand the breadth of indications for their bispecific program.

In addition there is a direct application of the Affimed platform to be able to bio-better many of the bispecific molecules Amgen has in their pipeline which have failed to make meaningful progress in the clinic due to unfavorable pharmacologic properties. Over the past couple years, Affimed has made major headway in updating and substantiating the IP portfolio, allowing for drop-in replacements of multiple binding domains (now termed the ROCK platform). This broad utility should be appealing to a company like Amgen who already has a major interest in bio-betters/similars. AFM11 may very well be a proof of concept that the drop-in approach to the new matrix will be successful across the Amgen portfolio of bispecifics sourced from Micromet.

It has been hypothesized that the lower T-cell numbers needed by Blincyto for efficacy than CAR-T can allow for a bispecific to move into solid tumors. However, effector: target ratios are still extremely unfavorable for the approach. Perhaps the TandAb matrix may change that, but we do not feel that the current management of Affimed is sufficient to maximize the value from the platform. Therefore, much like Mircromet,

We think if AFM 11 proves successful, in which we think the chances are 80% or so, Affimed is likely to be sold to Amgen at a relatively cheap valuation for the upside (around 800M-1B). We base this opinion on CEO Adi Hoess helping to sell his last company (He was the COO there) for a gross undervaluation – the drug in which his last company was sold for is doing near $700M a year in sales, yet the company sold for $550M – it should have sold for no less than $2B; granted, this was a private company.

On Monday, August 27th, 2018, Affimed announced that they have entered into a collaboration agreement with Roche:

> “Under the terms of the agreement, Affimed will receive $96 million in an initial upfront payment and other near-term committed funding. Affimed may be eligible to receive up to an additional $5.0 billion over time, including payments upon achievement of specified development, regulatory and commercial milestones, and royalties on sales. The agreement is subject to customary closing conditions, including clearance under the Hart-Scott-Rodino Antitrust Improvements Act, and closing is expected to occur in the third quarter of 2018. “This collaboration is based on Affimed’s innate immune cell drug discovery and development expertise and our team’s deep understanding of cancer immunology,” commented James Sabry, M.D., Ph.D., Global Head of Partnering, Roche. “Our partnership with Affimed provides an

opportunity to enhance our existing efforts to understand how the immune system can be activated to help people living with cancer."

The above deal has given the overall market a positive perception of Affimed's science. Since that deal, the stock has surged from the $1.50 a share range, to nearly $7.40 a share on August 29th. This development certainly has been very beneficial for long-time shareholders, especially those of us who averaged down over time.

While the current run-up may have stalled a bit, we do think that the stock will provide for a strong catalyst run-up play, as the company has several important upcoming binary events.

1. Ash data – Both for AFM 13/Keytruda combo, and more importantly, the much awaited first real look at AFM 11.
2. Several biomarker studies which should give us a better idea about the specific pathways Affimed may take moving forward.

We think that if both AFM 11 and 13 data sets prove positive, the stock has the potential to reach into the $20 range. A lot of factors depend on that price. A more reasonable higher-side expectation is a stock price somewhere north of $10 – to $15.

AFM 11 is a mono agent designed to be as such. Basically, it's designed to be a bio better (in practicality) of $AMGN's Blincyto. Therefore, we do think that if proven successful (high chance in our opinion) the drug would render Blincyto useless, and would likely be a 'billion-dollar-drug.'

Even after the advantageous current agreement with Roche, the market *still* seems to be oblivious to this asset and its potential – especially in the near-term.

We do expect Affimed's stock to see a pull back before resuming upwards as we approach Nov.1st – that is the date the ASH abstracts become un-embargoed.

Factoring in probabilities of AFM 11 success. Along with biotech investing mentality, we think the speculation after successful data will be successful, notwithstanding the fact that Affimed is now well-financed. Our price targets assume a fully diluted share count of $75M (now stands around 62M) – factoring in occasional secondary raises;

1 year price target that assumes success with AFM 13M AFM 11 initial data without MTD BEING REACHED = $25

The same as above, but assuming AFM 13 data fails to be clear, or breaks the modeling, and still assuming AFM 11 is successful - $18

As above, assuming less than stellar data on both 11 and 13 (low probability, under 50%) - $4 - $12, low to high depends on progress of Roche deal or lack-thereof.

Sources:

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