COMPANY NOTE

Initiating Coverage

USA | Healthcare | Biotechnology

July 15, 2014

Jefferies

Kite Pharma (KITE)

Initiate at Buy: CAR-T Readies To Revolutionize Refractory Cancer Treatment

BUY
Price target $35.00
Price $24.05

EQUITY RESEARCH AMERICAS

Key Takeaway

KITE shares hold significant promise based on PI/II data with KTE-C19 observing robust responses in patients with refractory lymphomas and leukemias and thereby supporting our favorable outlook in it's pivotal PII trial in third-line DLBCL and its potential in refractory leukemia. KITE's eACT platform should progress two additional programs (NY-ESO-1 and EGFRvIII) into the clinic by '16 and both offer upside. We initiate with a Buy rating and a $35 PT.

JEF was joint book-running manager in Kite's initial public offering on June 20, 2014.

KTE-C19 Has Observed Significant Potential in Hem Malignancies: We anticipate a strong response rate in the upcoming pivotal PII third-line DLBCL trial evaluating KTE-C19 in a single-arm design enrolling >40 patients, and is supported by the NCI PI/II trial observing an 88% objective response in eight patients with relapsed/refractory DLBCL. Data from the pivotal trial in third-line DLBCL is anticipated in mid-2016, and we estimate risk-adj peak sales of $526 million in DLBCL. Additional indications may generate risk-adj peak sales of $559 million across other refractory/relapsed lymphomas and leukemias. We estimate risk-adj sales in the EU of $457 million and believe KITE could receive a 20% royalty on topline sales. Our model does not assume potential upfront/milestone payments associated with ex-U.S. licensing agreement.

Early Stage Programs Offer Additional Upside: The company has licensed rights to a CAR-T v. EGFRvIII in glioblastoma and to TCR targeting NY-ESO-1 antigen for solid tumors. Both programs are currently in an ongoing NCI-sponsored PI/II trial with KITE-sponsored trials estimated to initiate in 2016. Clinical data with NCI's NY-ESO-1 TCR program reported a robust 67% objective response rate in fifteen patients with refractory synovial sarcoma and 53% response rate in nineteen patients with refractory metastatic melanoma. Both programs offer upside to our model, and we anticipate interim data updates from the ongoing NCI PI/II trials in the next 12-24 months.

NCI Immunotherapy Pipeline Acts As Kite's Discovery Engine: Kite's current research collaboration with NCI offers the company opportunity to license CAR-T and TCR pipeline programs based on early proof of concept PI data. NCI is currently evaluating a TCR targeting MAGE-A3 in a PI/II trial enrolling 107 patients with metastatic solid tumors.

Valuation/Risks

Our PT of $35 is DCF-based. Risks include clinical, manufacturing, competitive, regulatory, and commercial.

USD	Prev.	2013A	Prev.	2014E	Prev.	2015E	Prev.	2016E
Rev. (MM)	--	0.0	--	0.0	--	0.0	--	0.0
EPS								
Mar	--	--	--	(0.56)A	--	--	--	--
Jun	--	--	--	(0.06)	--	--	--	--
Sep	--	--	--	(0.06)	--	--	--	--
Dec	--	--	--	(0.08)	--	--	--	--
FY Dec	--	(1.18)	--	(0.76)	--	(1.13)	--	(1.11)
FY P/E		NM		NM		NM		NM

Financial Summary

Net Debt (MM):	($202.5)
Long-Term Debt (MM):	$0.0
Cash & ST Invest. (MM):	$202.5
Cash/Share:	$4.67
Cash (MM):	$202.5

Market Data

52 Week Range:	$32.65 - $22.82
Total Entprs. Value (MM):	$841.3
Market Cap. (MM):	$1,043.8
Shares Out. (MM):	43.4
Float (MM):	8.6
Avg. Daily Vol.:	NA

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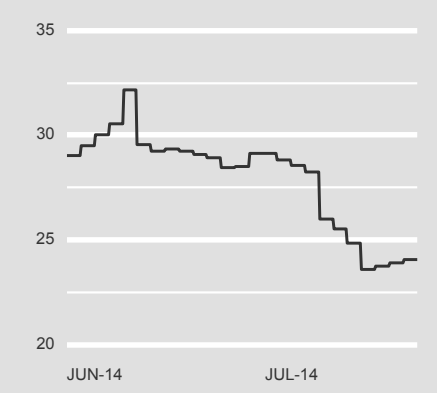
Price Performance



Kite Pharma

Buy: $35 Price Target

THE LONG VIEW

Scenarios

Target Investment Thesis

- Positive outcome from the PII registration study of KTE-C19 in DLBCL
- We expect KTE-C19approval for DLBCL in 2017 and peak sales of $526M by 2028 (risk-adj)
- We expect KTE-C19 approval for PMBCL, MCL, FL, ALL and CLL in 2019 and peak sales of $559M by 2028 (risk-adj)
- We expect peak EU royalties of $92M (risk-adj by 2028 for KTE-C19.
- DCF-based PT: $35

Upside Scenario

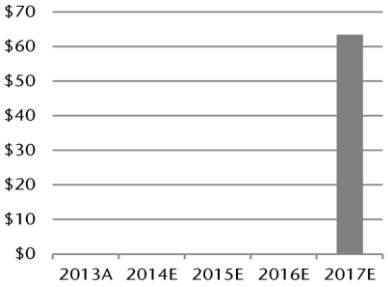
- De-risked KTE-C19 program through Phase II/III trial datasets
- DCF-based PT: $71
- Positive proof-of-concept for EGFRvIII CAR program in GBM
- DCF-based PT: an add'l $5
- Positive proof-of-concept for NY-ESO-1 TCR program in synovial and bladder cancer
- DCF-based PT: an add'l $5

Downside Scenario

- Negative outcome for KTE-C19 in DLBCL
- DCF-based PT: $13
- Negative outcome for KTE-C19 in other hem malignancies
- DCF-based PT: $16-24
- Negative outcome for KTE-C19 in all indications
- Cash-based PT: $5

Long Term Analysis

Revenue (millions)



Source: Company data; Jefferies estimates

Enterprise Value (EV)/Sales

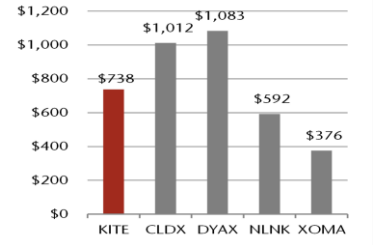
Source: Company data; Jefferies estimates

Other Considerations

We consider small-cap and mid-cap biotech companies with late-stage programs to continue to be attractive targets for partnering or M&A partnering with large-cap biotech and pharma companies, which we believe will be a driving factor for performance in the biotech sector 2014-2015.

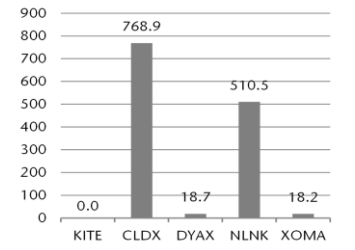
Peer Group

Group EV



Source: Factset, Jefferies estimates

Group EV/2014E Sales



Source: Factset, Jefferies estimates

Recommendation / Price Target

Ticker	Rec.	PT
KITE	**Buy**	**$35**
CLDX	Buy	$31
DYAX	Buy	$13
NLNK	Buy	$34
XOMA	Buy	$9

Catalysts

- Initiation of PII trial of KTE-C19 in DLBCL in H12015
- Initiation of NCI sponsored PI trial of SSX2 TCR in solid tumors in Q4 2014.

Company Description

Kite Pharma, Inc. is a biopharmaceutical company focused on the development and commercialization of cancer immunotherapy products involving the engineering of T-cells to express chimeric antigen receptors (CARs) or T-cell receptors (TCRs) to combat cancer. Its lead candidate is KTE-C19, a CAR-T product for the treatment of heavily pre-treated relapsed/refractory DLBCL which is set to begin a pivotal Phase I/IIb trial. KITE intends to expand KTE-C19 into other difficult-to-treat hem malignancies, including FL, MCL, PMBCL, CLL and ALL. KITE's technology has been developed in part through its collaboration with the National Cancer Institute through a cooperative research agreement (CRADA).

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Executive Summary

Kite Pharma is a biopharmaceutical company based in Santa Monica, CA, and was founded in 2009 with a focus towards the development of novel immunotherapy for cancer. The company's engineered Autologous Cell Therapy (eACT) platform is licensed from the National Cancer Institute (NCI), and trains the patient's own T cells to express two types of cancer-specific receptors (chimeric antigen receptors (CAR-T) or T-cell receptor (TCR)) which hone in on attacking tumor cells. We believe KITE has significant upside potential based on developing novel therapeutics utilizing the eACT platform. The company's lead program is KTE-C19 (CAR-T targeting CD19) in PI/II currently and in which KITE plans to initiate a pivotal Phase I/IIb trial in early 2015 in relapsed/refractory patients with diffuse large B cell lymphoma (DLBCL). KITE anticipates data from this trial by mid-2016, and filing for regulatory approval in Q4 2016 with potential approval in mid-2017. KITE plans to initiate a second trial in second line DLBCL in 2016, and will initiate in other indications such as relapsed/refractory chronic lymphocytic leukemia and acute lymphoblastic leukemia. Given the company's pipeline is licensed from NCI, KITE has also licensed rights to NCI's EGFRvIII CAR-T program which is currently in Phase I/II trials in patients with glioblastoma. We expect interim data updates from the NCI trial over the next 12-24 months, and KITE plans to initiate Phase I/II trials with this program in 2016. The company also recently licensed the NY-ESO-1 TCR program from NCI in May 2014. An IND filing is planned in late 2015 with initiation of Phase I/II trial in 2016.

- **KTE-C19 Offer Significant Promise in Relapsed/Refractory Lymphoma.** We believe promising Phase I/II data for KTE-C19 in relapsed/refractory (R/R) patients with B cell lymphomas and leukemias offers a favorable outlook for the pivotal Phase II trial in relapsed/refractory DLBCL anticipated to initiate in early 2015 with data in mid-2016. We believe the PII trial would be a single-arm design enrolling >40 patients. The PI/II interim data in the ongoing NCI trial reported 67% (n = 16/24) were in remission, and 86% (n = 19/22 evaluable) achieved an objective response. Of the twenty four patients enrolled in the trial, eight patients R/R DLBCL and 88% achieved an objective response. Duration of response was robust and 38% of all patients observed >10 month response. Patient follow-up continues, and these data likely will be updated at the American Society of Hematology meeting in December 2014. The pivotal Phase II trial in relapsed/refractory DLBCL would need to observe >50% response rate and >6 month duration of response to support an accelerated filing/approval strategy. As a proxy, we point to Spectrum Pharmaceutical's (SPPI, $7.76, Not Covered) Folotyn approved in September 2009 for treatment of R/R peripheral T-cell lymphoma with approval supported by a single arm trial observing an overall response rate of 27% and median duration of response of 9.4 months. We also highlight CTI Therapeutics (CTIC, $2.83, Not Covered) Pixuvri which received a complete response letter in April 2010 in R/R aggressive NHL in which a 37% overall response rate and 5.5 month duration of response was observed. We estimate topline data to be available mid-2016 with BLA filing Q4 2016 and accelerated approval/launch in mid-2017. We anticipate KITE will initiate a randomized PIII trial in second-line DLBCL in 2016.

- **KTE-C19 Also Has Potential in Relapsed/Refractory Leukemia.** KITE plans to investigate KTE-C19 in R/R chronic lymphocytic leukemia (CLL) and acute lymphoblastic leukemia (ALL) with clinical trials anticipated to initiate in 2016. To date, the ongoing NCI Phase I/II trial has enrolled seven patients with R/R CLL and 86% have observed an objective response and with three out of the six responders

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observing a robust duration of response of >15 mos. While patients with ALL were not enrolled in the NCI PI/II trial, we believe KTE-C19 could be effective in this indication based on other programs evaluating CAR-T CD19 in this setting in which 86% complete response was observed in pediatric ALL.

- **Early Stage Programs Offer Additional Upside:** The company has licensed rights to a CAR-T vs EGFRvIII in glioblastoma and to TCR targeting NY-ESO1 antigen for solid tumors. Both programs are currently in an ongoing NCI-sponsored PI/II trial with KITE-sponsored trials estimated to initiate in 2016. Clinical data with NCI's NY-ESO1 TCR program reported a robust 67% objective response rate in fifteen patients with refractory synovial sarcoma and 53% response rate in nineteen patients with refractory metastatic melanoma. In the Kite sponsored Phase I, the company will utilize a murine-based TCR which recognises human NY-ESO-1/HLA A2 and will not form mixed dimers with endogenous human TCR and which may have led to shortened duration of responses in the original NCI trial.

- **Additional Early-Stage Opportunities Are Available To Kite.** NCI is also conducting trials with a TCR program targeting MAGEA3 in 107 patients with metastatic tumors. MAGE-A3 is highly expressed in cancers of the lung, breast, ovary, bladder, and melanoma. Early efficacy data have been promising with five out of nine patients reporting tumor regression. However, off-target toxicity was noted in the trial, and as a result modifications were made to reduce toxicity risk.

Valuation

We arrive at our $35 price target based on a DCF valuation model, which assumes a WACC of 13%, terminal growth rate of 0% and outstanding shares of 43.4 million, driven by sales of KTE-C19. We assume market entry for KTE-C19 for third-line refractory diffuse large B-cell lymphoma (DLBCL) in 2017 based on accelerated approval on a positive data package from its pivotal Phase I/IIb trial. We estimate total U.S. peak sales of KTE-C19 of $1.8 billion (risk-unadjusted) by 2028 for multiple potential indications in hematology. Applying a 40% discount rate to reflect the risk associated with this asset, we estimate peak sales of $1.0 billion by 2028.

KITE's initial target population will be third-line relapsed/refractory diffuse large B-cell lymphoma (DLBCL). At this time, we only include the refractory DLBCL population (10%), and leave the relapsed population as upside (~30-40%). At 40% peak market penetration by 2028, we assume peak sales of $876 million (risk-unadjusted), or $526 million (risk-adjusted) using a 40% discount rate. KITE intends to expand into other lymphomas and leukemias, including PMBCL, transformed indolent NHL, FL, MCL, CLL and ALL. We estimate market expansion into FL, MCL, PMBCL, CLL and ALL in 2019 (refractory-only). We assume each reach peak market penetration of 25%, and we estimate peak sales of $214 million (FL), $39 million (MCL), and $45 million (PMBCL), $316 million (CLL) and $318 million (ALL) by 2028 (risk-unadjusted), respectively. Applying a 40% risk discount, we estimate peak sales of $128 million (FL), $24 million (MCL), and $27 million (PMBCL), $190 million (CLL) and $191 million (ALL) respectively.

We also model EU sales for KTE-C19. We model $141 million in peak sales for third-line refractory DLBCL in 2028 using a 40% risk-discount. For FL, MCL, PMBCL, CLL and ALL we model market entry in 2021 and peak sales of $35 million (FL), $7 million (MCL), $8 million (PMBCL), $131 million (CLL) and $136 million (ALL) in 2028, respectively (applying a 40% risk-discount). In total, we estimate peak sales in the EU of $457 million with a 40% risk-discount. We assume a 20% royalty, which translates to $92 million in 2028 in royalty revenue.

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At this time, we do not model sales for its EGFRvIII CAR-T program for glioblastoma or Kite's TCR targeting NYESO-1, and these represent upside to our current estimates. We assume R&D expenses of $9.1 million in 2014, with the pivotal Phase I/IIb trial expected to begin in H1 2015. We expect R&D expenses to grow to $67 million by 2028. We assume $3.0 million in SG&A expenses in 2014, and expect them to grow to $46 million by 2020 as it launches KTE-C19 in 2017 and expands into additional indications in 2019. We expect SG&A expenses to grow to $54 million by 2028.

Exhibit 1: DCF sensitivity analysis

Equity Value	Price/Share
$2,062.4	$47.55
$1,757.4	$40.52
$1,508.1	**$34.77**
$1,303.4	$30.05
$1,134.2	$26.15

Source: Jefferies estimates

Risks

Clinical Failure: As with all companies in biotechnology and pharmaceuticals developing treatments of the future, a clinical failure can lead to delays in approval or possibly discontinuation of programs.

Regulatory Failure: The FDA could determine the Biologic Licensing Application is inadequate for KTE-C19 for DLBCL and could delay approval. KITE is under the assumption that an ORR >50% with a duration of response >6 months in patients from a single-arm trial will be sufficient for accelerated approval, but the FDA could decide that is inadequate. Any delays in approval timelines could impact our earnings estimates, price target, and/or rating.

Commercial Failure: We currently assume peak sales for KTE-C19 of $1.0 billion in the U.S. (risk adjusted) and royalty revenue of $92 million (risk-adjusted) on EU sales by 2028. Our estimates may rely on the success of the company/partners to receive drug reimbursement from private/public payors.

Manufacturing Risks: KITE relies on its eACT process to manufacture its CAR-T products, and involves 1) harvesting T-cells from the patient's blood, 2) genetically engineering the T-cells to express cancer-specific receptors, and 3) increasing the number of engineered T-cells and 4) infusing the modified T-cells back into the patient. Assuming approval, KITE will require reliable commercial supplies for the materials used to manufacture and process its eACT-based candidates. KITE will need a consistent and reliable process, while limiting contamination risks, for manufacturing these candidates for the approved patient population. Any supply or manufacturing disruption could negatively impact KTE-C19 supply and sales.

Competitive Risks: Other companies are rapidly developing CAR-T product candidates in various stages of clinical development for hematological malignancies that may compete with KTE-C19. If any of these product candidates have an improved therapeutic profile over KTE-C19 and is approved, KTE-C19's growth trajectory in the marketplace, even if approved, could be adversely impacted.

Financing Risks: We expect KITE to have adequate cash to support the KTE-C19 launch in 2017, and we do not currently model any equity financing. However, KITE may need

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additional dilutive financing to fund the potential U.S. launch of KTE-C19 and its R&D programs in additional indications.

Company Overview

Kite Pharma, Inc. is a clinical-stage biopharmaceutical company focused on the development and commercialization of cancer immunotherapy products that harness the patient's immune system to combat the cancer, and involve the engineering of T-cells to express chimeric antigen receptors (CARs) or T-cell receptors (TCRs). KITE uses its engineered autologous cell therapy (eACT) that involves genetic engineering of T-cells to express CARs and TCRs. Its lead candidate is KTE-C19, a CAR-T product for the treatment of heavily pre-treated relapsed/refractory DLBCL which is set to begin a pivotal Phase I/IIb trial. KITE intends to expand KTE-C19 into other difficult-to-treat hematological malignancies, including FL, MCL, PMBCL, CLL and ALL. KITE's technology has been developed in part through its collaboration with the National Cancer Institute through a cooperative research agreement (CRADA).

Exhibit 2: Company Pipeline

Drug	Mechanism of Action	Indication	Development Phase
KTE-C19	Chimeric Antigen Receptor T-cells (CAR-T)	Heavily pre-treated r/r DLBCL, and other hematological malignancies	To initiate a pivotal Phase I/IIb trial
EGFRvIII CAR	Chimeric Antigen Receptor T-cells (CAR-T)	Recurrent glioblastoma	Phase I/II
SSX2 TCR program	T-cell receptor (TCR)	Solid tumors	Phase I
NY-ESO-1 TCR	T-cell receptor (TCR)	Solid tumors	Phase I

Source: Company data.

Exhibit 3: Key Upcoming Milestones.

Product	Indication	Event	Date
KTE-C19	**R/R DLBCL, Third-line**	Filing of IND for KTE-C19	Q4 2014
		Additional data from PI/IIa trial for hematological malignancies at ASH 2014	December 2014
		Initiation of pivotal PI/IIb trial for third-line r/r DLBCL (n>40; 1 EP: ORR, 2 EP: OS and duration of response)	H1 2015
		Preliminary data from pivotal PI/IIb trial	2015
		Enrollment completion of pivotal PI/IIb trial for third-line r/r DLBCL	Q1 2016
		Topline data for pivotal PI/IIb trial	Mid-2016
		U.S. approval/launch	H2 2017
	R/R DLBCL, Second-line	Initiation of PIII trial for second-line therapy DLBCL to support regular approval	2016
	Other lymphomas and leukemias	Initiation of clinical trials for FL, MCL, PMBCL, CLL and/or ALL	2015-2016
EGFRvIII CAR	**Glioblastoma**	Interim data from the NCI EGFRvIII CAR	2015
		KITE IND filing and initiation of Phase I/II trial	2016
		Final data from the NCI EGFRvIII CAR	2018
SSX2 TCR program	**Solid Tumors**	Initiation of PI study by NCI	Q4 2014
		Topline data for PI study	2015
		IND filing and clinical trial initiation	2016
NY-ESO-1 TCR Program	**Synovial and Bladder Cancer, Other**	Data from NCI TCR programs (various cancers)	2015
		IND filing and PI study initiation	Q4 2015
		KITE initiation of PI/II trials	2016

Source: Company estimates, Jefferies.

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KITE Set To Fly High With CAR-T

Aggressive Lymphomas and Leukemias

Chimeric Antigen Receptor T-cell (CAR-T) technology represents one of the most exciting developments in cancer immunotherapy. Early data from CAR-T therapies from Kite Pharma, Novartis, and Juno Therapeutics have shown extraordinary responses in difficult-to-treat hematological malignancies. The technology broadly involves harnessing the patient's T-cells by engineering them to express chimeric antigen receptors (CARs) to recognize and kill tumor cells. KITE's lead candidate is KTE-C19 that has been showing promising early data in heavily pre-treated aggressive NHL and leukemias, with response rates that exceed >80% and long duration of responses (>20 months in some patients). KITE plans to initiate a Phase II single-arm, multicentre trial for KTE-C19 in H1 2015 that will enroll >40 patients with DLBCL who failed two or more lines of therapy. KITE expects to use the results from the trial to support accelerated approval, with the expectation that ORR >50% and durations of responses of >6 months will be sufficient for accelerated approval. We expect data in mid-2016 and market entry in 2017.

We estimate total U.S. peak sales of KTE-C19 of $1.8 billion (risk-unadjusted) by 2028 for multiple potential indications in hematology. Applying a 40% discount rate to reflect the risk associated with this asset, we estimate peak sales of $1.0 billion by 2028. KITE's initial target population will be third-line relapsed/refractory diffuse large B-cell lymphoma (DLBCL), but KITE will follow with second-line DLBCL with a randomized trial for regular approval (to potentially start in 2016). DLBCL is the most common type of aggressive NHL subtype, making up at least ~20% of all newly diagnosed NHL cases in the U.S. At this time, we only include the refractory DLBCL population (10%), which represents ~11,200 patients. At 40% peak market penetration by 2028, we assume peak sales of $526 million for DLBCL by 2028 applying a 40% discount rate. KITE intends to expand into other lymphomas, including follicular lymphoma (FL), mantle cell lymphoma (MCL), and primary mediastinal large B-cell lymphoma (PMBCL). We estimate market expansion into these indications in 2019, and we estimate U.S. peak sales of $128 million, $24 million, and $27 million, respectively, for the refractory population using a 40% discount rate. KITE intends to expand into leukemias as well, including chronic lymphocytic leukemia (CLL) and acute lymphoblastic leukemia (ALL). For both CLL and ALL, we estimate $190 million and $191 million in U.S. peak sales by 2028, respectively, using a 40% discount rate. We also model EU sales for KTE-C19, and assuming a 20% royalty we estimate $92 million in 2028 in royalty revenue for the same indications as in the U.S.

U.S. Market Opportunity

U.S. Market Opportunity for KTE-C19 in DLBCL. The initial indication for KTE-C19 will be diffuse large B-cell lymphoma (DLBCL), the most common type of non-Hodgkin's lymphoma (NHL). According to the Surveillance Epidemiology and End Results (SEER) database, there are an estimated 70,800 new cases of NHL in the U.S. in 2014 (incidence). The Leukemia and Lymphoma Society estimates that there are approximately 558,340 patients living with NHL or are in remission in the U.S. (prevalence). We use a prevalence-based model to arrive at our KTE-C19 sales estimates. Based on SEER data which estimated only 438,325 U.S. NHL patients in 2007, we estimate a 3.4% annual growth rate.

We assume market entry for its initial target indication of third-line relapsed/refractory diffuse large B-cell lymphoma (DLBCL) in 2017. We estimate total peak sales of KTE-C19 of $1.8 billion (risk-unadjusted) by 2028 for multiple potential indications in haematological malignancies. Applying a 40% discount rate to reflect the risk associated with this asset, we estimate peak sales of $1.0 billion by 2028. Our sales breakdown is as follows:

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KITE's initial target population will be third-line r/r DLBCL. DLBCL makes up at least 20% to 30% of all newly diagnosed NHL cases in the U.S. (Source: American Cancer Society). DLBCL affects mostly middle-aged and older adults. At 20%, we estimate a prevalence of ~111,668 patients. With respect to the relapsed/refractory population (r/r DLBCL), approximately 30-40% relapse and 10% have refractory disease. Patients with r/r DLBCL have a poor outlook with a life expectancy of 3 to 4 months, if left untreated (Perry, A. R. et al., *Ann Oncol*, 1998, 9 (Suppl 1): S9-14; Fisher, R. I. et al., *NEJM*, 1993, 328, 1002-1006). At this time, we only include the refractory DLBCL population (10%), which represents ~11,200 patients. We will leave the relapsed population as upside, which represents another potential 30-40%. KITE intends to initiate a randomized trial for second-line therapy for DLBCL for regular approval in 2016, which could allow KITE to expand its target patient population to include second-line DLBCL patients and is likely to have a survival endpoint. We assume $175,000 per treatment with a gross-to-net assumption of 15% and assume no y/y price increases. At 40% peak market penetration by 2028, we assume peak sales of $876 million by 2028 (risk-unadjusted). Applying a 40% risk discount to reflect the risk associated with this asset, we arrive at peak sales of $526 million for DLBCL by 2028.

KITE intends to expand into other lymphomas, including primary mediastinal large B-cell lymphoma (PMBCL), transformed indolent NHL, follicular lymphoma (FL) and mantle cell lymphoma (MCL). PMBCL is a less common subtype of diffuse large cell lymphoma (~10% of cases of DLBCL, *NEJM* 2013, 368, 15, 1408-1416) characterized by a significant fibrosis on histology and arises in the thymus. Some indolent NHL, which makes up ~30% of all NHL (Leukemia and Lymphoma Society) can transform into aggressive NHL, known as transformed indolent NHL. FL is the most common type of indolent NHL with ~15,300 new diagnoses in the U.S. each year. We estimate market expansion into FL, MCL, and PMBCL in 2019, and we model only refractory patients. We assume each reach peak market penetration of 25%. Under the same price assumptions as DLBCL, we estimate peak sales of $214 million, $39 million, and $45 million by 2028 (risk-unadjusted), respectively. Applying a 40% risk discount, we estimate peak sales of $128 million, $24 million, and $27 million, respectively.

KITE intends to expand into leukemias and would seek to evaluate in chronic lymphocytic leukemia (CLL) and acute lymphoblastic leukemia (ALL). In the U.S., the prevalence of CLL is ~119,386 and the incidence is ~15,700, and is the most common adult leukemia in the U.S. The prevalence for ALL is ~66,030, and the incidence is ~6,020 (American Cancer Society). We model market entry into CLL and ALL in 2019 under the same assumptions in price ($175,000 per treatment) and gross-to-net discount (15%). For both CLL and ALL, we estimate peak penetration rates of 25%, resulting in $316 million and $318 million in peak sales by 2028, respectively. Applying a 40% discount rate, we estimate $190 million and $191 million in peak sales by 2028, respectively.

We also model EU sales for KTE-C19, under similar market assumptions as the U.S. but with different prevalence assumptions. We model 228,572 NHL patients in the EU. We model market entry into the EU for refractory DLBCL in 2020 at an annual price of $100,000. We model $141 million in peak sales in 2028 using a 40% risk-discount. For FL, MCL, and PMBCL, we model market entry in 2021 and peak sales of $35 million, $7 million, and $8 million in 2028, respectively, applying a 40% risk-discount. With respect to CLL and ALL, we estimate market entry in 2021 and peak sales of $131 million and $136 million in 2028, respectively, applying a 40% risk-discount. In total, we estimate peak sales in the EU of $457 million applying a 40% risk-discount. Finally, we assume a 20% royalty, which translates to $92 million in 2028 in royalty revenue.

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Planned Phase I/IIb trial

Nuts & Bolts. KITE is planning a pivotal, single arm, multicentre, U.S.-based Phase I/IIb clinical trial, and it will file an IND in Q4 2014 to initiate this trial. The trial will enroll >40 patients with chemotherapy-refractory (failed two or more lines of chemotherapy) DLBCL, including an Adriamycin-containing chemotherapy regimen (i.e. R-CHOP) and a platinum-containing regimen (i.e. R-ICE and R-DHAP), or patients who have relapsed ≤12 months or autologous transplant. Patients will receive a regimen of a single infusion of CAR T-cells following 3-day chemotherapy conditioning regimen (Cytoxan/fludarabine). The trial will use T-cells generated by the new manufacturing process. The primary endpoint will be objective response rate (ORR). Secondary endpoints will include overall survival (OS) and duration of response. KITE anticipates the trial to begin enrolling the trial in H1 2015, and with topline data in mid-2016. KITE expects preliminary data to become available in 2015.

What Will be Sufficient for Accelerated Approval? The results from this Phase II study will form the basis for accelerated approval for KTE-C19, and KITE expects to see an ORR >50% with a duration of response >6 months to be sufficient for accelerated approval. We evaluate historical examples as potential proxies for KTE-C19. We know of two examples, Velcade (bortezomib) and Folotyn (pralatrexate), which have been approved on the basis of response rate in a relapsed or refractory disease in single arm trials for an uncommon NHL subtype. Velcade, marketed by Millenium Pharmaceuticals (A Takeda Oncology Company; 4502 JP, ¥4,723, Hold), was approved in December 2006 for mantle cell lymphoma who have received at least one prior therapy. Folotyn, marketed by Spectrum Pharmaceuticals, was approved in September 2009 for relapsed/refractory peripheral T-cell lymphoma. We note that in both cases in the single arm trials, the ORR was <50%. The median duration of responses were >6 months.

Another example to highlight is CTI Biopharma's Pixuvri (pixantrone) for r/r aggressive NHL. In March 2010, the Oncologic Drugs Advisory Committee (ODAC) voted 9-0 against recommending approval for Pixuvri for relapsed/refractory aggressive NHL, and in April 2010, CTIC received a complete response letter from the FDA. CTIC filed for accelerated approval based on a Phase III study for r/r aggressive NHL. The study included mostly B-cell lymphomas but also had T-cell lymphomas as well: Grade 3 FL, transformed lymphoma, DLBCL, peripheral T-cell lymphoma, and anaplastic large cell lymphoma. Patients must have received ≥2 prior combination chemotherapy regimens. The study compared pixantrone v. comparator (n=70 each), with a primary endpoint of objective response rate (ORR). The planned sample size was 320, but the study was stopped early at an unplanned time point due to poor accrual. CTIC claimed that accrual was difficult because investigators were reluctant to enroll third-line patients because many preferred to use multi-agent chemotherapy or supportive care. In the Pixuvri group, 20% of patients had an objective response v. <6% in the comparator group (p=0.021) as determined by Independent Assessment Panel (IAP) review with duration of responses of 5.5 v. 3.4 months, respectively.

The ODAC panel voted negatively on Pixuvri on issues separate from whether the magnitude of ORR benefit or duration of response was sufficient, and therefore we believe has little read-through for KITE. Despite p<0.05 in Pixuvri's trial, the FDA reviewers noted that had just one fewer patient responded to pixantrone, the outcome would have carried a p-value of 0.036, and two fewer patients failing to respond would have resulted in a p-value of 0.06. The FDA reviewers also had concern over the re-reading of some of the scans by IAP, carrying up to 4 readings of the same scan. The FDA reviewers felt that the re-reading of scans resulted in a change in the assessment of response to CR/CRu in 2 patients on pixantrone and 1 patient in the comparator arm, leading to uncertainty in the assessment of responses. Finally, the FDA reviewers felt that the fact that no patients

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enrolled in the U.S. attained a CR or CRu was of particular concern. On safety, the FDA reviewers noted that deaths and serious adverse events were more common on the pixantrone arm than the comparator arm. Deaths in the pixantrone group were attributed to cardiac failure, infection, respiratory failure, and other causes.

Exhibit 4: Other products indicated for relapsed/refractory NHL

Disease	Indication	Trial	Findings	Approval
Rituxan (rituximab)	R/R CD20+ low grade or follicular NHL	Single arm trials in R/R low grade, CD20+ NHL	ORR: 38-57% Median duration: 11.2-15 mo	September 2006 (for low grade or follicular NHL)
Zevalin (ibritumumomab tiuxetan)	R/R CD20+ low grade or follicular NHL	Single arm trial in R/R low grade or follicular NHL	ORR: 74% Median duration: 6.4 mo	February 2002
		Zevalin v. Rituxan in R/R low grade, follicular or transformed NHL	ORR: 83% Zevalin v. 55% Rituxan Median duration: 14.3 mo Zevalin v. 11.5 mo Rituxan	
Velcade (bortezomib)	Mantle cell lymphoma, 1 prior therapy	Single arm trial in R/R mantle cell lymphoma	ORR: 31% Median duration: 9.3 mo	December 2006*
Folotyn (pralatrexate)	R/R peripheral T-cell lymphoma	Single arm trial in R/R peripheral T-cell lymphoma	ORR: 27% Median duration: 9.4 mo	September 2009*
Pixuvri (pixantrone)	R/R aggressive NHL	Single arm trial in R/R aggressive NHL	ORR: 37% Duration of CR/CRu: 5.5 mo	*Not approved*

Source: FDA ODAC Briefing Document for Pixuvri (adapted); Jefferies.
***Approved on the basis of response rate in relapsed/refractory disease in single arm trials in uncommon NHL subtypes.**

Response Rates for SOC for Aggressive NHL is only ~14%. Another takeaway from Pixuvri is the magnitude of benefit in the comparator arm, with CR/Cru/PR rate of only 14.3%, providing an indication of the limited benefit achieved from standard-of-care for aggressive NHL. KITE's early data for its CAR-T therapy which showed that 86% achieved a CR/PR and compares favorably to the comparator arm for Pixuvri's study or even Pixuvri's overall response (incl PRs) of 37%. We note that the patient populations between Pixuvri's study and KITE's study are not identical, but they are similar enough to make a broad comparison. Patients in the Pixuvri study included: Grade 3 FL, transformed lymphoma, DLBCL, peripheral T-cell lymphoma, and anaplastic large cell lymphoma (systemic, T/null cell). Patients must have received at least 2 prior combination chemotherapy regimens, and the first-line regimen must have contained an anthracycline/anthracenedione. Patients in the comparator arm had oxaliplatin (43%), ifosfamide (17%), vinorelbine (16%), etoposide (13%), mitoxantrone (6%), or gemcitabine (1.5%).

Future Directions. KITE also intends to initiate a randomized trial in second-line therapy for DLBCL for regular approval, potentially in 2016. The design of the trial has yet to be finalized pending discussions with the FDA. KITE is considering randomizing patients to KTE-C19 v. standard-of-care second-line salvage chemotherapy (R-ICE or R-DHAP) with a potential survival endpoint. The trial is expected to be ongoing at time of BLA filing for accelerated approval in third-line DLBCL. KITE also intends to investigate KTE-C19 in other lymphomas and leukemias, including follicular lymphoma (FL), mediastinal B-cell

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lymphoma (MCL), PMBCL, chronic lymphocytic leukemia (CLL), and acute lymphoblastic leukemia (ALL).

Deep Dive of CAR-T Platform Technology

The immune system has the ability to recognize and attack tumor cells based on the expression/overexpression of specific antigens. However, the immune cells often express receptors that poorly recognize antigens on tumors. To that end, significant progress has been made to design chimeric antigen receptors (CARs) that are able to recognize the tumor antigens and to modify T-cells in cancer patients to express these CARs, thereby activating a host of cell effector functions. A CAR is composed of 1) a targeting domain, 2) a hinge region, 3) a transmembrane domain, and 4) intracellular signaling components. CAR T-cells can proliferate in the patient and can infiltrate the microenvironment of solid tumors. KITE further believes that CARs can overcome mechanisms of tumor escape. When activated, the T-cells release cytokines that contribute to the killing of the tumor cells.

The Evolution of CARs. CARs have gone through several generations (Exhibit 5), with each succession building on the previous one. KITE/NCI's CAR T-cell therapy may be considered a second-generation CAR-T.

- *First-generation CARs* – First-generation CARs consist of a single signaling domain most commonly derived from the CD3ζ chain of the TCR/CD3 complex antigen binding mediated by extracellular domain. The target binding domain consists of a single-chain variable fragment (scFv) of an antibody comprising variable domains of heavy and light chains. The CAR is tethered (or "anchored") to the plasma membrane by means of transmembrane domain.

- *Second-generation CARs* – Second-generation CARs incorporate an additional intracellular signaling domain to the basic first-generation receptor configuration that provides a costimulatory signal to CD28 or CD137. First-generation CARs are not sufficient to elicit a robust cytokine response, including IL-2, and support T-cell expansion upon repeated exposure to antigen, which prompted the development of second-generation CARs. The dual-signaling receptors confer greater strength of signaling and persistence to the T-cells, resulting in greater potency which has been supported in patients treated with a mixture of T-cells transduced with either CD28/CD3ζ or CD3ζ-only CARs (Savoldo, B. et al., *J Clin Invest* 2011, 121, 1822-1826). More exhaustive comparative studies between costimulatory signals are needed. KITE/NCI's CAR T-cell therapy has only one co-stimulatory domain targeting CD28, and thus may be considered a second-generation CAR-T.

- *Third-generation CARs* – Third-generation CARs include two costimulatory domains, such as CD28, 4-1BB, and other costimulatory molecules. These CARs employ vector(s) that encode a CAR and a CAR-responsive promoter that respond upon successful signaling of the CAR by the transgenic production of cytokines (i.e. IL-2). The cytokines can recruit other components of the immune system to expand the overall anti-tumor immune response. One study utilizing a CD20-specific CD28/4-1BB/CD3ζ CAR did not show dramatic responses (Till, B. G. et al., *Blood*, 2012, 119, 3940-3950), however, as mentioned above more exhaustive comparative studies between costimulatory signals are needed.

- *Fourth-generation/TRUCKs* - TRUCKs are CAR-redirected T-cells used as vehicles to produce and release a transgenic product that accumulates in the targeted tissue, mostly a pro-inflammatory cytokine such as IL-12. The IL-12 deposit in the tumor lesion attracts innate immune cells (i.e. NK cells and macrophages),

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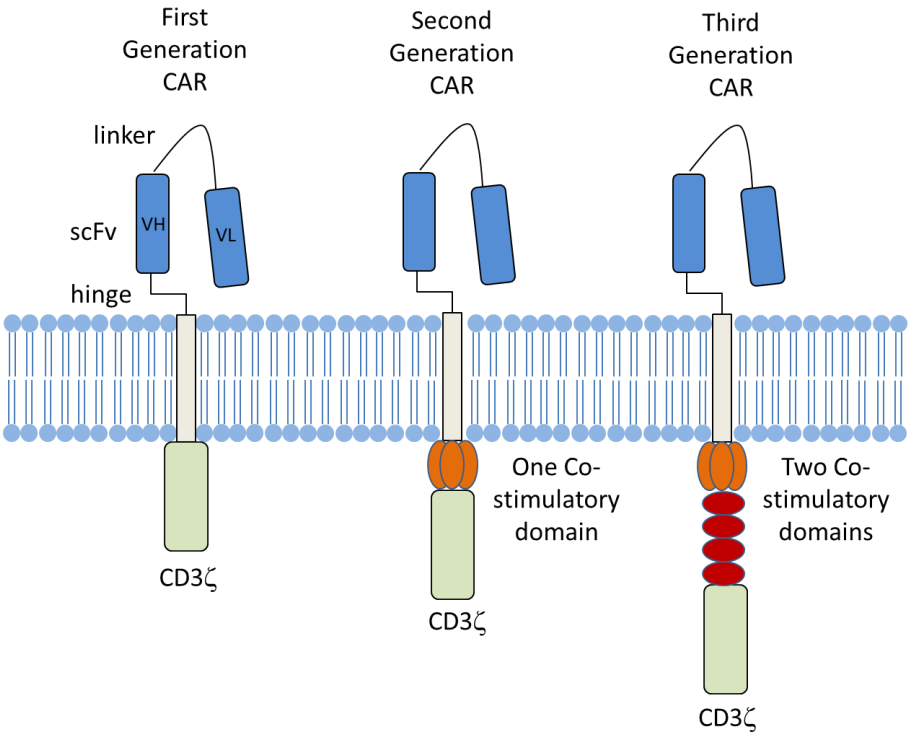
to further attack tumor cells. (Chmielewski, M. et al., *Immunological Reviews*, 2013, 83-90) IL-12 cannot normally be given at therapeutic doses given its systemic toxicity.

Exhibit 5: Diagram representation of first-, second-, and third-generation CARs



Source: Maus, M. V. et al., *Blood*, 2014, 123(17) (adapted); Jefferies.

Is There a Difference Between One or Two Costimulatory Signals? KITE has decided to move forward with a CAR with only the CD28 costimulatory domain (second-generation) into clinical development with its CD19 program v. having two costimulatory domains. Whether a clinically meaningful difference can be observed between a CAR with only a CD28 costimulatory domain v. a CAR with dual-domains (i.e. CD28/4-1BB) remains to be seen. A study by Kochenderfer, J. N. et al. (*J Immunother* 2009, 32(7), 689-702) suggests that a CAR with only the CD28 domain may be sufficient for clinical activity. Kochenderfer et al compared two CARs: 1) one with only a CD28 costimulatory signal v. 2) another with both CD28 and 4-1BB costimulatory signals. Kochenderfer measured IFN-γ in CD19+ targets, including bv173, SupB15, and CLL. As can be seen in Exhibit 6, the CD28 only-CAR produced greater IFN-γ compared to the CD28/4-1BB CAR in these CD19+ targets, suggestive of greater T-cell activation. It remains to be seen whether this result translates into clinically meaningful differences.

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Exhibit 6: IFN-γ ELISA comparing CARs with CD28 and CD28/4-1BB costimulatory domains

Effector cells	CD19+ Targets		
	bv173	SupB15	CLL
(CD28 only)-CAR	17,450	6,150	2,970
(CD28+4-1BB)-CAR	13,500	2,700	640
Non-transduced	118	57	17

Source: Kochenderfer, J. N. et al. *J Immunother* 2009, 32(7), 689-702 (adapted); Jefferies.
100,000 effector cells were cultured overnight with 100,000 target cells, and an IFN-γ ELISA was performed. Effector cells were T-cells transduced with CD28-only CAR, CD28/4-1BB CAR, and non-transduced cells.

Viruses Used to Introduce CARs into T-cells. A variety of methods to introduce CAR constructs into T-cells are available, each having advantages and disadvantages with respect to cost, safety, and level of expression. Non-viral based methods can be used because of its low cost and risk of insertional mutagenesis, but suffers due to relatively low efficiency of gene transfer. Lentiviral vectors efficiently and permanently transduce T-cells but are costly to manufacture. Many investigators use γ-retroviruses (i.e. KITE/NCI), which are relatively safe, easy to produce and efficiently and permanently transduce T-cells. There has been some general concern of γ-retroviruses regarding insertional mutagenesis given certain cases of T-cell leukemia which developed several years after gene therapy in patients with X-linked severe combined immunodeficiency (SCID-X1) were treated by gene transfer to restore missing IL-2 receptor γ gene to hematopoietic cells (Hacein-Bey-Abina, S. et al., *J Clin Invest*, 2008, 118, 3132-3142). However, a report by Scholler et al. investigating the long-term results of three clinical trials evaluating γ-retroviral CD4ζ vector-engineered CAR T-cells for HIV showed with >540-patient-years without integration-mediated toxicity (Scholler, J. et al., *Sci Transl Med* 2012, 4, 1-7). The authors commented that they had 95% confidence that the true adverse event was <0.0068 per person-year, or no more than 1 event in every ~147 years. It is possible that the previous safety issues observed in the SCID-X1 experience is not a general feature of retroviral vectors, and thus may not be applicable to CARs.

CAR-T's Have High Persistence. Scholler et al further commented in their study that the CAR T-cells were detected in 98% of the samples tested for ≥11 years after infusion, indicating a high level of persistence of CAR-T's (Scholler, J. et al., *Sci Transl Med* 2012, 4, 1-7). The authors speculate on potential mechanisms for the higher persistence, citing improved cell culture technology that promotes central memory cells, a non-immunogenic transgene, and signaling from the CD4ζ CAR moiety. Based on the observed decay rates of the CD4ζ-modified T-cells (and assuming linear decay rates going forward), the authors believe that the persistence could last decades. Early data from the different CAR-T programs targeting CD19 suggests variability in CAR-T persistence, however, it's unclear at the moment how this translates to clinical differences in duration of response.

CD19 as a Target for Hematological Malignancies. All CAR-T therapy in clinical development at this time is directed towards the CD19 antigen for hematological malignancies. CD19 is an ideal target antigen for CAR-T therapy because expression in normal tissues is restricted to B-lineage cells. Nearly all B-cell malignancies express CD19.

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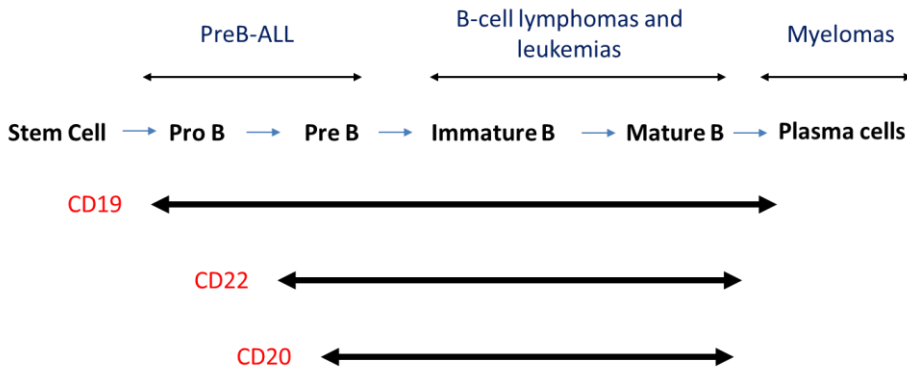
Expression is limited to B-cell malignancies, normal B cells, and a small population of other immune cells. It is not expressed on hematopoietic stem cells or other normal tissue. B-cells are considered non-essential tissue. As CAR-T technology shifts into solid tumors, other antigens targets will have to be identified, but given that many of the antigens are expressed in normal tissue at low levels, toxicity may be a key issue going forward.

Exhibit 7: CD19 expression on various B-cells



Source: Brentjens, R. presentation (adapted); Jefferies.

Applicability to Solid Tumors Remains a Challenge. All CAR-T therapy in clinical development at this time is directed towards the CD19 antigen for hematological malignancies. CD19 is an ideal target antigen for CAR-T therapy because expression in normal tissues is restricted to B-lineage cells, and nearly all B-cell malignancies express CD19. However, expanding CAR-T therapy into solid tumors will have the challenge of identifying a target antigen that is unique to the tumor. Other antigens could potentially be present in normal cells, and CAR-T therapy could therefore have significant off-target side effects ("on-target, off-tumor responses"). For instance, a recent case study of a CAR based on the widely-used Herceptin (trastuzumab) containing CD28, 4-1BB, and CD3ζ signaling moieties (Morgan, R. A. et al., *Mol Ther* 2010, 18, 843-851). As an ERBB2 (HER2/neu)-targeted CAR, this CAR was investigated in colon cancer patient who had exhibited lymphatic invasion and vascular involvement. However, within 15 minutes of infusion the patient experienced respiratory distress, and displayed a dramatic pulmonary infiltrate on chest X-ray. Despite intensive medical intervention, the patient died 5 days after treatment. Serum samples showed that she experienced CRS as demonstrated by marked increases in IFN-γ, granulocyte macrophage-colony stimulating factor (GM-CSF), TNF-α, IL-6, and IL-10. The authors believed that the CAR T-cells triggered a release of cytokines by recognition of low levels of HER2 on lung epithelial cells. Other targets, such as PSMA for castrate-resistant prostate cancer and ROR1 for certain leukemias and lymphomas, may also potentially suffer from similar problems given that these targets have also been detected in normal tissue (Silver, D. A. et al., *Clin Cancer Res* 1997, 3, 81-85; Hudecek, M. et al., *Blood*, 2010, 116, 4532-4541), however this level of toxicity observed in the Herceptin-CAR has not been reported to date.

Cytokine Release Syndrome (CRS). Cytokine Release Syndrome (CRS) is a common side effect of CAR-T therapy. In some patients, the reaction can be severe, and they exhibit high-grade non-infectious fever, drop in blood pressure (hypotension), and potential neurological changes. Whether neurological changes are a result of systemic cytokines crossing the BBB and engaging the cytokine receptors in the brain remains unclear.

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Changes so far have all been reversible. Many patients develop macrophage activation syndrome (MAS), characterized by elevated serum ferritin, IL-6, and IFN-γ, and MAS is often associated with neurologic toxicity (Maus, et al., *Blood* 2014).

At the least, CRS is an indication that the T-cells are proliferating. CRS appears to be correlated with antitumor activity, but the correlation is not conclusive. Most responding patients have some degree of CRS, but it is not yet clear whether the severity of CRS is related to antitumor activity. Severity does appear to be related to tumor burden, which triggers greater levels of T-cell activation. In many cases, the patients were given steroids which resolved the events. But steroid treatment may limit the efficacy of CAR-T's (Sentman, C. L. *Immunotherapy*, 2013, 5, 8, 783-785)

In many cases, CRS and MAS can be managed with anti-IL-6, such as Actemra or Enbrel. It is not clear which cell type produces the vast majority of the cytokines, particularly IL-6. IL-6 may be produced by the dying B-cells, dying tumor cells, or activated macrophages that are recruited to digest lysed tumor cells (Maus, M. V. et al., *Blood*, 2014, 123, 17, 2625-2635). In many cases, CRS appears treatable with tocilizumab (Actemra) or etanercept (Enbrel) both of which are anti-IL-6 monoclonal antibodies. Unlike steroids, anti-IL-6 antibodies do not appear to affect the CAR T-cell's activity or proliferation. Toxicity will necessitate the need for monitoring and interventional guidelines to identify and treat patients who are at high risk for CRS.

Other. Oddly, CAR T-cells are often found in the cerebrospinal fluid of asymptomatic patients even when there is no evidence of CD19+ disease. Some propose that the fever and IL-6 release during CRS may enhance trafficking of CAR T-cells to the cerebrospinal fluid, or it is possible that there is undetected expression of CD19 in the brain. B-cell aplasia occurs frequently and is an expected on-target result of CD19-directed therapies. It is generally manageable with treatment with γ-globulin as replacement therapy. Persistent aplasia could result in an increased risk of infection. It is not yet clear how toxicity is related to CAR-T cell dosage.

NCI KTE-C19 CAR-T Phase I/II Data

Remarkable Efficacy in Heavily Pretreated Patients. NCI's anti-CD19 CAR T-cell therapy has shown remarkable efficacy in the PI/II trial. The most updated data is presented in KITE's S-1, some of which was presented at ASH 2013. The 24 patients in the study received a total of 27 infusions of anti-CD19 CAR. Three patients were retreated upon eventual cancer progression after the first dose. One follicular lymphoma patient achieved a PR (progressed at ~8 months, received a second treatment and achieved another PR) with an impressive total duration of 55+ months. A patient with chronic lymphocytic leukemia (CLL) achieved a CR (progressed at ~22 months, received a second treatment and achieved another CR) with a total duration of remission of 45+ months. Additionally, the trial has shown remission of large solid tumor lesions in lymph nodes, abdomen and chest, and also in DLBCL-related bone metastasis. The ORR from the PI/IIa trial among 19 evaluable patients, measured by standard criteria, was 86%. KITE is compiling and will detail the data in an upcoming publication.

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Exhibit 8: NCI CD19 CAR Data.

Disease	No. of Patients	Response	Status/Commentary
Follicular Lymphoma (FL)	3	2 PR (66%)	1 progressed after ~8 mo, achieved another PR after 2nd tx; total duration 55+ mo (ongoing)
			1 ongoing after ~20+ mo
		1 not evaluable (death) (33%)	Not evaluable after ~2 mo
Chronic Lymphocytic Leukemia (CLL)	7	4 CRs (57%)	1 progressed after ~22 mo, achieved another CR after 2nd tx; total duration 45+ mo (ongoing)
			1 ongoing after ~32+ mo
			1 ongoing after ~25+ mo
			1 ongoing after ~18+ mo
		2 PRs (29%)	1 progressed after ~8 mo
			1 progressed after ~5 mo
		1 pt still ongoing without response (14%)	On tx for ~5 mo so far
Splenic Marginal Zone Lymphoma (SMZL)	1	1 PR (100%)	1 progressed after ~12 mo, achieved another PR after 2nd tx; total duration 40+ mo (ongoing)
Primary Mediastinal BCL (PMBCL)	4	2 CRs (50%)	1 ongoing after ~26+ mo
			1 ongoing after ~10+ mo
		1 progression (25%)	
		1 not evaluable (death) (25%)	Not evaluable after ~2 mo
Diffuse Large B-cell Lymphoma (DLBCL)	8	2 CR (25%)	1 ongoing after ~5 mo
			1 ongoing after ~8 mo
		5 PR (63%)	1 ongoing after ~5 mo
			1 ongoing after ~4 mo
			1 ongoing after ~4 mo
			1 ongoing after ~3 mo
			Progressed after ~4-5mo
		1 lost to follow-up (12%)	1 lost to follow-up after ~2 mo
Indolent NHL (iNHL)	1	1 PR (100%)	1 ongoing after ~9 mo

Source: Company data, Jefferies.
***JEF estimate from KITE's graphical representation**

Sixteen of the 24 treated patients were in remission, and three of the 13 patients progressed after their first treatment and re-established remission after a second course of treatment (and remain in remission).

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Two patients from the study were highlighted at ASH 2013:

- *Case Study 1*: Patient with primary mediastinal B-cell lymphoma (PMBCL). Failure of prior treatments: 6 cycles of R-CHOP: progression. XRT: CR but recurred in 5 mo. R-ICE: no response. Methotrexate + ARA-C + Rituxan: no response. Patient received a single infusion of anti-CD19 CAR T-cells/kg (5x 10^6), and achieved a CR and remains durable 23 months after treatment.

- *Case Study 2*: Patient with refractory primary mediastinal B-cell lymphoma (PMBCL). Patient had failed on 10 prior treatments: progressed on R-CHOP, R-ICE, and R-GDP. Had a good response in mediastinal disease to XRT. Referred for progressive liver and other abdominal lymphoma. Patient received a single infusion of anti-CD19 CAR T-cells/kg (2.5 x 10^6), and achieved a CR ~9 months after treatment.

Safety. All patients in the study experienced low blood count due to the chemotherapy conditioning regimen. As in most CAR-T therapies, the most common acute toxicities included fever, hypotension, kidney dysfunction, and confusion – symptoms associated with cytokine release syndrome. Some patients experienced cranial nerve dysfunction and speech impairment. These acute toxicities generally peaked during the first 8 days of therapy, resolving completely within 3 weeks. The safety profile from the 21 patients is summarized in Exhibit 9. It is not yet clear how toxicity is related to cell dosage.

Exhibit 9: Safety summary

Organ	Adverse Event	All CAR T-cell Related Adverse Events (Gr2-5)	Grade 3	Grade 4
		Number of Adverse Events (% of Total Patients)		
Cardiac	Hypotension	5 (21%)	4 (17%)	0 (0%)
	Left Ventricular Dysfunction	1 (5%)	0 (0%)	0 (0%)
Metabolic	Creatinine	4 (19%)	1 (5%)	2 (10%)
Neurology	Confusion	3 (13%)	2 (8%)	0 (0%)
	Encephalopathy	1 (4%)	1 (4%)	0 (0%)
	Agitation	1 (4%)	0 (0%)	0 (0%)
	Anxiety	1 (4%)	0 (0%)	0 (0%)
	Cranial Neuropathy	1 (4%)	1 (4%)	0 (0%)
	Motor Neuropathy	1 (4%)	1 (4%)	0 (0%)
	Pyramidal Tract Dysfunction	1 (4%)	0 (0%)	1 (4%)
	Somnolence	3 (13%)	0 (0%)	3 (13%)
	Aphasia	3 (13%)	0 (0%)	2 (8%)
Pulmonary	Dyspnea	1 (4%)	0 (0%)	0 (0%)
	Hypoxia	2 (8%)	1 (4%)	0 (0%)
Vascular	Acute Vascular Leak Syndrome	1 (4%)	1 (4%)	0 (0%)

Source: Company data; Jefferies.

Two patients died in the trial. The deaths were not attributable to the CAR-T therapy. One patient in cohort 1 died of influenza pneumonia 18 days after CAR-T therapy. The patient received the high dose conditioning regimen prior to CAR-T, and high dose IL-2 subsequently, and the death was attributable to chemotherapy conditioning regimen. The other patient from cohort 2, died of a cardiac arrhythmia after receiving the anti-CD19 CAR T-cell therapy. The patient originally presented with extensive lymphoma in her chest

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surrounding her heart, so hearth arrhythmia was presumed to be the cause of death and considered unrelated to treatment.

NCI CAR-T Also Demonstrates Efficacy in R/R ALL: Daniel W. Lee, III presented data at ASH 2013 using NCI's CD19 CAR-T cells in children with r/r ALL, even after allogeneic hematopoietic stem cell transplantation (HSCT). As an NCI CD19 CAR-T, it includes a CD28 co-stimulatory domain and uses a retroviral platform to modulate the T-cells. The primary objective of the study was safety and feasibility. Sixteen patients were enrolled in the trial to date (median age: 13). Patients enrolled in two strata: no prior transplant v. those who do (100 d after transplant, offered immunosuppression for at least 30 d). Half of the patients had at least one prior stem cell transplantation (SCT). Two patients did not meet proper dose levels due to poor T-cell expansion. The trial had a standard 3+3 format, starting at 1 million CAR transduced T-cells/kg. The maximum tolerated dose (MTD) was determined to be 1x 10^6 CAR T-cells/kg. Among patients who had Grade 3-4 adverse events, one patient had Grade 4 cytokine release syndrome, and one patient Grade 3 dysplasia. In the dose expansion phase (dose level 1): three additional patients had additional cytokine release syndrome, including one who had cardiac arrest and was successfully resuscitated. Several patients had Grade 4 neutropenia for median of 29 days, beyond what one would expect from Cytoxan/fludarabine and may require further investigation. Cytokine release syndrome associated with IL-6 and ameliorated by blocking IL-6 function (tocilizumab). The overall CR rate was 62.5%. The CR rate in ALL was 67%, despite that most patients had significant tumor burdens at time of treatment. Only three patients required tocilizumab alone or with steroids. The three refractory patients who never had remission despite multiple rounds of chemotherapy, achieved MRD(-) CR and moved onto bone marrow transplant. CAR-T therapy could therefore be used as a bridge to transplant. In patients achieving MRD(-) CR, there was a 75% leukemia-free survival in the study. There was no unexpected toxicity after transplant. In vivo, CAR-T cell expansion correlates with response and grade of cytokine release syndrome. There was no correlation of tumor burden and degree of cytokine release syndrome, although there was a trend in those who had Grade 3-4 AEs.

Manufacturing Process

eACT – General Process. KITE manufactures its CAR-T therapy by a process it calls eACT (engineered Autologous Cell Therapy). The T-cells are manufactured *ex vivo* and genetically re-directed against cancerous cells. The apheresis product is obtained from the patient with B-cell malignancy using a standard blood bank procedure. The collected cells are sent to a central processing facility, where the peripheral blood mononuclear cells (including T-cells) are separated from the other blood components. The peripheral blood mononuclear cells are stimulated to proliferate and transduced with a retroviral vector to introduce the CAR-T gene. The cells are then propagated in cell culture bags, and then washed and frozen at the cell processing site. The cells are then shipped back to the clinical center to be infused back into the patient. Typically patients receive lymphodepleting chemotherapy before CAR cell infusion with the intent of promoting homeostatic proliferation and expansion of the infused CAR-modified T cells, but it remains unknown whether pre-infusion chemotherapy is actually necessary in all patients. The total turnaround time from receipt of the patient's blood to infusion of the engineered T-cells is about two weeks.

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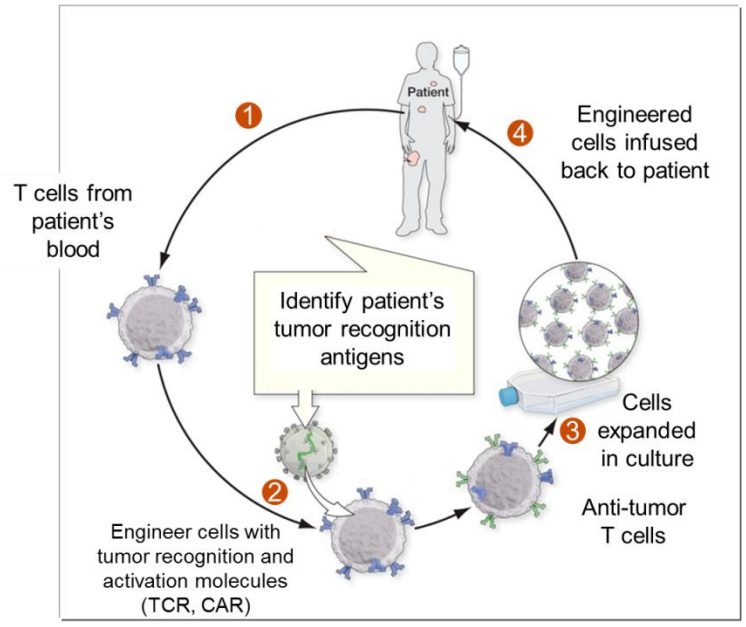
Exhibit 10: eACT (engineered autologous T-cell therapies)



Source: Company presentation; Jefferies.

RACE CAR (Rapid Cell Expansion CAR-T). KITE has modified the NCI clinical production process to simplify and improve unit operations for the CAR-T product. The new "closed" process eliminates the serum while retaining the characteristics of the CAR T-cell product. The use of serum-free medium is expected to be mandatory in the future, given the projections indicate an insufficient world supply that will peak in a few years (Maus, M. V. et al. *Blood*, 2014, 123, 17, 2625-2635; Brindley, D. a. et al., *Regen Med* 2012, 7, 1, 7-13). The new process takes <6 days in total (v. the old process that takes 10 days). The new process supports a dose of ~1-2 x 10^8 anti-CD19 CAR T-cells. Better et al. noted that T-cell phenotypes on either Day 6 or 10 are comparable between the two processes (similar percentage of naïve, central memory, effector memory, and effector T-cells). Day 6 cells are less differentiated. Better et al. also compared the bioactivity of the anti-CD19 CAR T-cells and showed that IFNγ (pg/mL) production on Day 6 was comparable between the old and new process. Also, the fold culture expansion is similar between the old and new processes in five at-scale engineering runs. The process differences are summarized in Exhibit 11. A multicenter clinical trial with aggressive non-Hodgkin lymphoma (NHL) using T-cells generated by new manufacturing process expected to begin in H1 2015.

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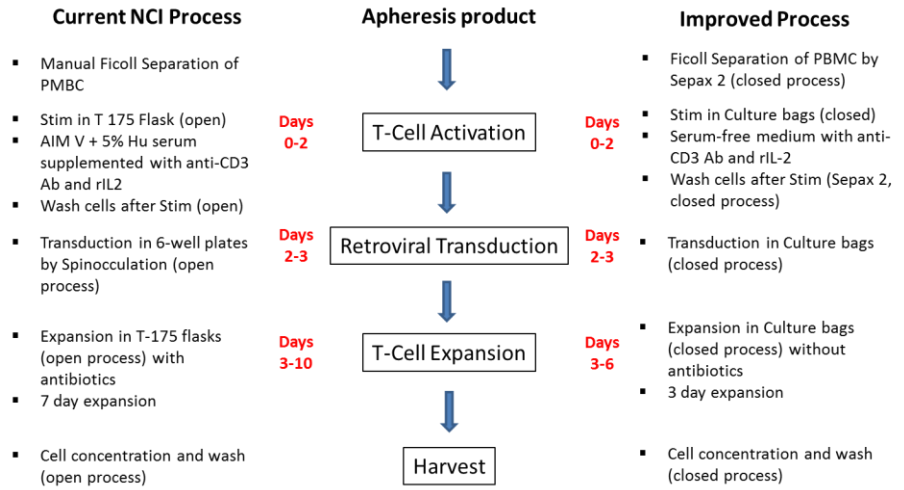
Exhibit 11: Comparison of "old" and "new" processes for manufacturing CAR T-Cells.



Source: Better, M. et al., *ASCO* 2014 (adapted); Jefferies.

Exhibit 12: Bioactivity of anti-CD19 CAR T-cells showing IFN-γ (pg/mL) production on Day 6

	CD19+ Cell Lines			CD19- Cell Lines	
	Toledo	Nalm6	CD19-K562	NGFR-K562	CEM
Untransduced Old	0	5	17	17	5
Untransduced New	0	228	56	5	17
Transduced Old	11,269	13,986	64,911	43	17
Transduced New	11,101	16,883	104,324	70	0
Control	11,942	19,818	81,165	30	5

Source: Better, M. et al., *ASCO* 2014 (adapted); Jefferies.

Competitive Landscape

We outline potential competitors in the CAR-T space in clinical development:

Novartis: Novartis' (NOVN VX, CHF 80.40, Buy) lead CAR-T candidate is CTL019, licensed from the University of Pennsylvania. CTL019 consists of an intracellular T cell receptor CD3ζ chain signaling domain that induces T-cell activation, a costimulatory 4-1BB domain, and anti-CD19 antibody fragments that bind to CD19. CTL019 has demonstrated efficacy in leukemias (CLL and ALL). On July 7, 2014, Novartis received Breakthrough Status and Fast-Track designation for CTL019. In December 2012, Novartis acquired a 16,000 square meter FDA-approved manufacturing facility in Morris Plains, NJ, from DNDN for $43 million. Novartis will utilize all fixed assets at the site, including all equipment, machinery, utilities, and cell therapy related plant infrastructure, and certain former DNDN personnel. The personnel will support both clinical and commercial production of potential new products and therapies that emerge from the Novartis-University of Pennsylvania collaboration, including CTL019. The facility space and infrastructure can also accommodate future CAR-production activities.

In their latest R&D Day in June 2014, Novartis stated that it intends to begin pivotal trials for ALL for CTL019 in 2014 with a 2016 timeframe targeted for U.S. filing. Novartis also intends to expand into DLBCL. A second-generation CTL019 that may be regulated and is

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bi-specific is being explored in Phase I trials. Finally, Novartis is exploring CAR-T's towards solid tumors, including CAR-T's directed against mesothelin in mesothelioma and pancreatic cancer in Phase I trials and EGFRvIII in glioblastoma. A CAR-T-directed against EGFR specific to glioma is anticipated to enter the clinic in Q3 2014.

Data in Pediatric ALL Presented at ASH 2013. Novartis presented data at ASH 2013 from a study of >60 patients will relapsed/refractory CLL and ALL (some after bone marrow transplant) at the University of Pennsylvania and Children's Hospital. The data presented was from the pediatric ALL cohort consisting of 22 children on treatment. Among the 22 children, 19 have gone into CR (86%). Five patients who achieved CR experienced relapse (1 had CD19(-) relapse). For all 27 pediatric and adult ALL patients, there was an 89% CR rate. There were 6 relapses altogether, with median follow-up 3.4 months (range: 2-18 months). Patients $\geq 2^{nd}$ relapse, and majority were refractory to multiple prior therapies. Sixteen patients had post-allogeneic SCT (T-cells collected 6 months post-SCT), with no GVHD to date. The modified T-cells were found to be highly proliferated (up to 10,000-fold expansion). Persistence was present in some, but not all patients. In those that responded, persistence could be as long as >145 days and ongoing (range 2 weeks to 18 months). Persistence was ~28 days in those who did not achieve CR.

There was significant cytokine secretion that seemed to correlate with toxicity, including IL-6 and IFN-γ. Median IL-6 levels were much higher in those who achieved CR v. those who did not. Patients who suffered from CRS were given tocilizumab (Actemra), which was given monthly and had well-tolerated side effects. Rapid responses were observed from tocilizumab with an immediate ablation of fever. CTL019 was found in the cerebrospinal fluid (CSF). Two patients had CSF disease at low levels that cleared after CAR therapy, but whether CTL019 was active in CNS or not was unclear. Finally, along with CRS, very high ferritin levels in patients (16k-415k ng/mL) were also observed that suggest Macrophage Activation Syndrome (MAS) or Hemaphagocytic lymphohistiocytosis (MLH). Like CRS, the effects appear to be reversible with cytokine blockade as tocilizumab. There may be a genetic predisposition for MAS or MLH.

CTL019's CR Rate Compares Favorably with Previous Agents. CTL019's CR rate of 86% compares very favorably with other agents in single drug trials in heavily pre-treated r/r ALL, including clofarabine, nelarabine, and Marqibo. These approved agents have a CR rate of only ~20-23%. Furthermore, none of these drugs have shown a survival benefit. We believe this portends a favorable outlook for CTL019 which shows data that already far surpasses these agents.

Exhibit 13: Recent trials for r/r ALL and CR rates

Registration single drug trials	Therapy	No. of Patients	CR Rate
Jeha et al., 2005	Clofarabine (≥2 prior regimens)	49	**20%**
Berg et al., 2005	Nelarabine (≥2 prior regimens)	39	**23%**
DeAngelo et al., 2005	Nelarabine (≥2 prior regimens)	28	**21%**
O'Brien et al., 2010	Marqibo (≥2 prior regimens)	101	**20%**

Source: June, C. *AACR* 2014 (adapted); Jeha, S. et al., *J Clin Oncol* 2006, 24, 1917-1923; Berg, S. L. et al., *J Clin Oncol* 2005, 23, 3376-3382; DeAngelo, D. J. et al., *Blood* 2007, 109, 5136-5142; O'Brien, S. M. ASCO *2010*; Jefferies

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Phase I Experience in Two Children with R/R ALL. The Phase I experience of two children with relapsed/refractory pre-B-cell ALL on CTL019 were detailed in an *NEJM* article (Grupp, S. A. et al., *NEJM* 2013, 368(16), 1509). These patients had poor prognosis despite use of aggressive treatments, including allogeneic hematopoietic stem-cell transplantation and bispecific CD19 antibody. The first patient was 7-year old girl with second recurrence of ALL. She had second remission after re-induction chemotherapy, but the cancer recurred 4 months later and did not respond to further intensive chemotherapy (including clofarabine, etoposide, and cyclophosphamide). She did not receive lymphocyte-depleting chemotherapy before CTL019 infusion. The second patient was a 10-year old girl with ALL who had second relapse after undergoing transplantation of umbilical-cord blood from an unrelated donor 28 months after diagnosis. She had GVHD after transplantation, which resolved with treatment. She was not given immunosuppressive treatment at time of relapse. She did not have another remission, in spite of multiple cytotoxic and biologic therapies.

These two patients received infusions of T-cells transduced with CTL019 at a dose of 1.4 x 10^6 to 1.2x10^7 CTL019 cells/kg. The CTL019 T-cells expanded to a level >1000x as high as initial engraftment level. CR was observed in both patients, and is ongoing in one patient 9 months after treatment. The other patient experienced a relapse with her blast cells no longer expressing CD19 ~2 months after treatment.

Both patients had acute toxic effects consisting of fever and cytokine-release syndrome and macrophage activation syndrome within a week after infusion. Both had substantial elevations in lactate dehydrogenase (LDH) levels, and prominent elevations in IFN-γ and IL-6. Systemic elevations of pro-inflammatory cytokines were reversible. Concomitant with peak T-cell expansion and tumor-cell elimination and consistent with on-target activity of CTL019 cells against CD19+ target cells.

- *Patient 1*: This patient experienced Grade 3 febrile neutropenia, Grade 4 hypotension, and Grade 4 acute vascular leak syndrome. Glucocorticoids were administered on day 5. There was a brief response on fever but no remission of hypotension. This patient was given anti-cytokine therapy (etanercept and tocilizumab) on day 7 with rapid effects within hours.

- *Patient 2*: This patient experienced Grade 3 febrile neutropenia, Grade 3 encephalopathy, and Grade 4 elevated AST/ALT. The febrile neutropenia resolved on day 6, and the biochemical changes were reversible.

Juno Therapeutics – A Conglomerate Of Three Academic Institutions: Juno Therapeutics, a privately-held biotech located in Seattle, was founded in Dec 2013. Three academic institutions, Fred Hutchinson Cancer Research Center, Memorial Sloan-Kettering Cancer Center, and Seattle Children's Research Institute brought their respective CAR-T platforms under one umbrella organization. Juno's technology utilizes anti-CD3/CD28 bead technology for activation and expansion of the CAR-T cells in which inert beads are covalently coupled to anti-CD3 and anti-CD28 antibodies thus driving a potential expansion of human T-cells by 100 to 1,000 fold.

Do Beads Drive Additional Benefit? Given the Novartis and Juno CAR-T platforms employ bead technology to expand T-cells whereas the Kite platform utilizes antibodies vs CD-3 in the presence of Fc receptor bearing accessory cells – we decided to evaluate the differences of both approaches and came across a study from the National Institute of Health (Li et al, *J Tran Med* 2010) and analysing the differences in CD4/CD8 T cells across both methodologies. The study evaluated cells from healthy donors in which T cells were expanded with anti-CD3/CD28 bead technology, anti-CD3 antibodies, or no stimulator. All cells received IL-2, and the beads were removed on day 7. The cells were evaluated over a 21-day period in the study. Both processes generated T-cells expansion at 50-60

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hours after stimulation. The study found greater CD4 cell expansion with the bead technology at day 7, 14, and 21 with greater CD8 cell expansion at earlier timepoints with the anti-CD3 process. The study does conclude that beads have a greater ability to expand T cells but T cell expansion may not be the only metric to compare the two platforms.

Exhibit 14: % CD8 T cells generated with bead technology vs anti-CD3 process

	Day after stimulation		
Cells stimulated with	**Day 7**	**Day 14**	**Day 21**
Anti-CD3/CD28 beads	34.0 ± 6.8	47.6 ± 6.3	48.9 ± 5.4
Anti-CD3	53.7 ± 7.0	68.0 ± 5.4	63.3 ± 4.4
	p < 0.005	p < 0.005	p < 0.05

Source: Li et al, *J Tran Med* 2010

The study also evaluated the potential to restimulate T cells. Recall, three patients in the NCI CD19 Phase I/II trial were re-treated with a second dose. Kite has plans to cryopreserve CAR-T cells for each patient in a centralized cell bank, and therefore restimulation of these CAR-T cells may be critical to re-dosing. Both the bead platform and the anti-CD3 process observed similar CD4 cell expansion, however, CD8 cell expansion was multi-fold greater with anti-CD3. The bead exposed cells were also prone to activation induced cell death of CD8 T cells. The study also observed phenotypic differences between the two methodologies with the anti-CD3 approach maintained CD45RA and CD27 expression whereas both were downregulated when exposed to beads. Based on the study's conclusions, it appears CD45RA could influence expansion into the central lymphoid tissue.

Review of the Memorial Sloan Program. Memorial Sloan Kettering Cancer Center utilized a gamma-retroviral vector to code chimeric antigen receptor T-cells and similar to the Kite program, it also utilizes a CD28-CD3zeta signalling domain. At the recent annual American Association for Cancer Research meeting in April 2014 presented data in refractory B-ALL after initial remission. Upon relapse, patients are infused CAR-T cell infusion (dose: 3 x 10^6 T cells/kg) and fifteen out of sixteen patients were able to generate sufficient T-cells to receive therapy. Average production time was 11 days with average CD3+CAR+ (or CAR positivity) was 24% in the T-cell receptor (range: 0-61%). Persistence of the CAR-T cell in the study has averaged 28.5 days. Complete response rate after CAR-T therapy was 82%, 72% were MRD negative, and 44% of all patients transitioned to allo-stem cell transplant. Average time to complete response is 24.5 days. Patients in the trial experienced fever, hypotension, hypoxia, and neurologic changes which are associated to cytokine release syndrome. Data from this trial showed cytokine release syndrome has greater likelihood in pts with greater disease burden b/c of increased CAR-T cell expansion. Two patients died within 1st 3 weeks of CAR-T infusion – one due to cardiac disease and a second patient due to persistent seizures. As a result, the trial was briefly placed on clinical hold in April 2014, and modifications to the trial include excluding patients with cardiac disease and modifying the dose based on tumor burden which should reduce the risk of cytokine release syndrome.

Fred Hutchinson CD19 Program Targets ALL. The Phase I/II trial at the Fred Hutchinson Cancer Research Center has enrolled five patients with refractory NHL and

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one patient with relapsed Ph+ ALL. Of the six patients enrolled, four patients have achieved a complete response, one patient achieved remission, and one patient was categorized as minimal response.

The Fred Hutchinson program differs from the MSKCC platform in utilizing a lentivirus for genetic integration of T-cell. One program utilizes a CD28-CD3 zeta signalling domain to target CD19, and employs a suicide gene (truncated EGFR receptor which inactivates the CAR-T cell upon binding with cetuximab). The other CAR-T program at Fred Hutchinson utilizes a CD3zeta signalling domain and targets CD19.

Cellectis – Potential for "Off-the-Shelf" CAR-T: Cellectis (ALCLS FP, €13.54, NC) is developing an allogeneic T-cell product (UCART19) which should allow for an 'off-the-shelf' CAR-T therapeutic that simplifies the manufacturing process. Cellectis is also developing next-generation CARs, and potentially combinations with checkpoint inhibitors and cancer vaccines. Recently, Pfizer (PFE, $30.24, Buy) entered into a collaboration agreement with Cellectis over its CART-T immunotherapies. PFE has exclusive rights to pursue development and commercialization of CAR-T therapies for the PFE-selected targets. The agreement also provides for a total of 12 targets selected by Cellectis (both companies will work together on preclinical research on 4 Cellectis-selected targets, and Cellectis will work independently on 8 other targets). Cellectis will be responsible for development and commercialization of CAR-T therapeutics for Cellectis-selected targets. PFE has right of first refusal to the 4 Cellectis-selected targets. Cellectis' program is ~2-3 years behind KITE. Cellectis received $80 million in an upfront payment in addition to a 10% equity investment by Pfizer, and is eligible to receive up to $185 million in milestones for each product.

Cellectis' technology is based on genome editing technology, called transcription-activator like effector nucleases (TALEN), which may be employed to silence T-cell intrinsic genes that may restrain T-cells from attacking tumor cells. TALENs are bacterial DNA-binding proteins precisely bind and cut DNA and allow the insertion of a sequence or deletion/repair of the chromosome. TALENs have the flexibility to accommodate spacers of different lengths. The company also employs a proprietary electroporation technology to generate high transfection efficacy and high cell viability, although specific percentages of gene expression and cell viability have not been disclosed. Electroporation introduces a series of voltage charges (first high voltage with short duration pulses then low voltage with longer duration pulses) to disrupt the cell membrane and thereby allowing charged molecules entry into the cell.

Cellectis believes its platform has several advantages over autologous CAR-T platforms including: off-the-shelf allogeneic productions, lower cost of goods (70-80% gross profit), potential to create multi-ligand CAR-T cells, and inclusion of a suicide gene to allow for potential elimination of the CAR-T cell. A single healthy donor may be able to generate up to 1,000 doses (10^7 cells/treatment) based on activation of 10^9 T cells leading to production of 1 x 10^{10} CAR T cells with an average production time of 17 days without release testing.

Cellectis plans to initiate Phase I trials with its lead CAR-T, UCART19 targeting CD19 receptor, in Q2 2015. In the Raji Burkitt lymphoma animal model, UCART19 administered animals observed 100% response rate with 5 out of 7 complete responses. The company also reported greater comparable/greater cytolytic activity with UCART19 vs standard CAR-T in SUPT1 leukemia cell that is expressing for CD19. However, details of the study design are unavailable to reference dosing and co-stimulatory domain(s) in the preclinical in cell line study. Other programs include UCART123 and UCART33 (treatment for AML) expected to enter preclinical studies in Q4 2014, and UCART-BCMA and UCART38 (treatment for multiple myeloma) expected to enter preclinical studies in mid-2015.

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bluebird bio/Celgene: In March 2013, bluebird bio (BLUE, $34.19, NC) and Celgene (CELG, $88.37, Buy) announced the formation of a broad and global strategic collaboration to discover, develop, and commercialize CAR-T products. BLUE/CELG are working with the Baylor College of Medicine to evaluate CAR-T cell therapies for a range of solid and hematologic malignancies. Financial terms include an upfront payment and up to $225 million per product in potential option fees, and clinical and regulatory milestones. Bluebird bio has the right to participate in the development and commercialization of any licensed products from the collaboration. They have a 50/50 co-development and profit-share in the U.S. in exchange for a reduction of milestones. If bluebird bio declines to exercise its co-development and profit-sharing rights, royalties will be paid in regions with no profit share, including the U.S.

Bellicum - Early Stage CAR-T Platform Incorporating Various Aspects Of CAR-T Cell Control. Bellicum's proprietary CAR T-cell program, CaspaCIDe, utilizes the CaspaCIDe safety switch to control CAR T-cells after they have been administered to a patient, and thereby enabling rapid elimination of administered CAR-T cells in the event of toxicity. The CaspaCIDe program utilizes a small molecule, AP1903, to drive the dimerization of CID proteins and thereby causing the activation of caspase 9 signaling, and activating caspase 3 which initiates cell destruction. Given AP1903 was first discovered in the late '90s, we're unclear on the long-term intellectual property covering this platform. We understand one of patents received by Ariad and covering AP1903's ability to regulate the biological activity of genetically engineered cells was issued in Feb 1999.

The company also is developing its GoCAR-T platform to more precisely control CAR-T expansion and activation. Again, this technology utilizes AP1903 to control T-cell proliferation, and antigen recognition is required for full activation of the T-cell, and AP1903 dosing is discontinued once the appropriate clinical response is attained. GoCAR-T is currently in preclinical development.

The CaspaCIDe technology has been demonstrated to selectively eliminate immune cells in the Graft versus Host Disease (GvHD) in stem cell transplant patients (Stasi, A. D. et al., *NEJM*, 2011, 365, 1673-83), supporting its proof-of-concept as a "kill-switch" for their CAR-T platform. In the CASPALLO investigator-sponsored trial, ten mismatched transplant recipients received CaspaCIDe-modified, ex vivo allodepleted donor T cells in which four patients developed Grade 1/2 graft versus host disease and were successfully treated with AP1903. In a separate trial of nine patients, two patients developed (GvHD) and were successfully treated with AP1903. AP1903 appear to have a fast onset with T-cell elimination within 30-60 minutes of administration.

The suicide gene iCasp9 consists of the sequence of the human FK506-binding protein, FKBP12 (with an F36V mutation), connected to the gene encoding human caspase 9. FKBP12-F36V binds to a small-molecule dimerizing agent, AP1903, with high affinity. On its own, AP1903 is biologically inert. Typically, activation of the mitochondrial apoptosis pathway requires apoptotic peptidase activating factor 1 (APAF1) which binds to cytochrome c to form an oligomeric apoptosome, which in turn binds and cleaves caspase 9 preproprotein and releases an activated form of the peptidase. The peptidase results in a caspase cascade, and ultimately, apoptosis. In transduced cells, the administration of AP1903 leads to dimerization of iCasp9 which bypasses activation of the initial mitochondrial apoptotic pathway, but nevertheless results in the caspase cascade and eventual apoptosis.

The iCaspase9.2A.ΔCD19 bicistronic transgene (i.e., one with two cistrons) comprises of the iCasp9 sequence with truncated CD19 (ΔCD19) serving as the selectable marker. The sequence cassette is then incorporated into the SFG retroviral vector.

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Amgen: Amgen is developing (AMGN, $118.98, Buy) blinatumomab, a 55 kDa investigational anti-CD19/CD3 bispecific T-cell engager (BiTE) antibody for adults with Philadelphia-negative (Ph-) r/r ALL. The antibodies to CD19 and CD3 are recombinantly joined by a short, non-glycosylated five-amino acid linker. Co-binding of CD19 and CD3 leads to T-cell activation and up-regulation of T-cell activation markers as IFN-γ, IL-2, and TNF-α. Thus, BiTE antibodies have the ability to redirect target cell lysis via T-cells, and activate the T-cells to kill the target cells. T-cell activation stimulates the proliferation of CD4+ and CD8+ cytotoxic cells. Stimulation of CD3 amplifies the T-cell signal.

In July 2014, AMGN received Breakthrough Therapy Designation for blinatumumab for ALL based on Phase II data of 189 adults with Ph- r/r B-precursor ALL treated with blinatumomab. The data was recently presented at ASCO 2014. Blinatumomab was given at 28 µg/day as a continuous infusion up to 2 cycles (for primary endpoint assessment), followed by up to 3 cycles (consolidation) if the patient responded. The primary endpoint was CR/CRh during the first two cycles. Secondary endpoints included relapse-free survival (RFS), overall survival, HSCT realization and incidence of AEs. The CR/CRh response was 43% (81/189). The RFS was 5.9 months. The median OS for all patients was 6.1 months. Among those who achieved CR/CRh on Day 77, the median OS was 9.9 months, v. those who did not whose median OS was 2.7 months. On safety, 67% had Grade 3 or 4 events and 15% had Grade 5 events (death). The most common ≥Grade 3 events included febrile neutropenia (25%), neutropenia (16%), and anemia (14%). For the patients who died, infection, disease progression, and hemorrhage were the main causes. These patients were found to have uncontrolled leukemia and did not achieve CR/CRh while on study. Nervous system/psychiatric disorder ≥Grade 3 events occurred in 13% of patients.

As in the case with CAR-T's, CRS is a common side effect in patients treated with blinatumumab with the release of inflammatory cytokines. Patients may experience fever, chills, headache, hemodynamic instability, bleeding, capillary leak syndrome, and/or respiratory compromise. Current clinical trials mandate coadministration of dexamethasone at initiation of therapy that will reduce cytokine concentration without compromising T-cell activation.

A randomized, open-label Phase III study for blinatumomab in 400 patients with r/r ALL is underway. The patients will be randomized 2:1 to receive blinatumomab or investigator's choice of standard-of-care chemotherapy. The primary endpoint is overall survival (OS). Secondary endpoints include CR rate within 12 weeks of infusion, CR duration, number of patients with MRD remission, allogeneic HSCT with or without blinatumomab treatment, adverse events, anti-blinatumomab antibody formation, event-free survival, and laboratory parameters. The study is expected to be completed in H1 2016.

Blinatumomab is also being investigated in r/r DLBCL in an open-label Phase II. Data was presented at ASH 2013. In Part I of the study, two cohorts were evaluated: 1) stepwise blinatumomab dosing of 9, 28, and 112 µg/d during weeks 1, 2, and thereafter; 2) blinatumomab at 112 µg/d throughout. After a 4-week treatment-free period, patients who achieved an objective response could receive a 4 week consolidation cycle. The ORR based on independent radiological assessment was 57% (cohort I: 1 CR, 2 PR; cohort II: 1 PR). Three patients had progressive disease (cohort I). Four patients were not evaluable for response. The most common adverse events were tremor (64%), diarrhea (46%), and fatigue (46%). Ten of 11 patients had at least one Grade ≥3 AE with two patients (both in cohort II) experiencing Grade 4 AEs. There were no Grade 5 AEs. Ten of 11 patients had central nervous system AEs, mostly tremor (64%), speech disorder (36%), and disorientation (27%). Five patients (3 patients in cohort I; 2 patients in cohort II) had Grade 3 CNS AEs (no Grade 4 or 5 CNS events). The investigators concluded that the

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stepwise dose had a better benefit/risk profile and is the recommended dose for Part II of the protocol.

Other Ongoing CAR T-cell Trials. Ongoing CAR T-cell trials for hematologic malignancies are listed in Exhibits 15 and 16.

Exhibit 15: Ongoing CAR T-cell trials in hematologic malignancies.

Antigen	Cancers	Gene transfer	CAR signaling domain	Phase	Sponsor	Cell type/selection/drug combination
CD19	Pediatric B-cell leukemia and lymphoma	Lentivirus	4-1BB-CD3ζ	I	CHOP/University of Pennsylvania	
CD19	CD19+ malignancies	Lentivirus	4-1BB-CD3ζ	I	University of Pennsylvania	
CD19	ALL (post-allo-HSCT)	Lentivirus	4-1BB-CD3ζ	I	University of Pennsylvania	T-cells from donor
CD19	CLL (randomized to 1 or 2 doses)	Lentivirus	4-1BB-CD3ζ	II	University of Pennsylvania	
CD19	CLL	Retrovirus/ lentivirus	CD28-CD3ζ; 4-1BB-CD3ζ	I/II	MSKCC/ University of Pennsylvannia	
CD19	ALL		CD28-CD3ζ	I	MSKCC	
CD19	Auto-HSCT for NHL followed by T-cell infusion	Retrovirus	CD28-CD3ζ	I	MSKCC	
CD19	Relapsed ALL post-allo-HSCT	Retrovirus	CD28-CD3ζ	I	MSKCC	CD19 CAR-transduced EBV-specific CTLs from donor
CD19	CLL (residual disease following upfront pentostatin/cyclophosphamide/ Rituxan	Retrovirus	CD28-CD3ζ	I	MSKCC	
CD19	Pediatric relapsed B-cell ALL	Retrovirus	CD28-CD3ζ	I	MSKCC	
CD19	NHL, CLL	Retrovirus	CD28-4-1BB-CD3ζ, and CD28-CD3ζ	I	Baylor College of Medicine	2 CARs at the same time
CD19	ALL, CLL, NHL	Retrovirus	CD28-CD3ζ	I	Baylor College of Medicine	Yervoy in low-grade disease (2 wk after T-cells)
CD19	ALL, CLL, NHL post-allo-HSCT (prophylaxis or therapy)	Retrovirus	CD28-CD3ζ	I/II	Baylor College of Medicine	CD19 CAR-transduced trivirus-specific CTLs (CMV, EBV, and adenovirus) from donor
CD19	CLL	Transposon	CD28-CD3ζ	I	MD Anderson Cancer Center	
CD19	Leukemia/lymphoma post-cord blood HSCT	Transposon	CD28-CD3ζ	I	MD Anderson Cancer Center	
CD19	B-cell malignancies post-allo-HSCT	Transposon	CD28-CD3ζ	I	MD Anderson Cancer Center	Donor derived
CD19	B-cell malignancies post-allo-	Transposon	CD28-CD3ζ	I	MD Anderson Cancer Center	Low- and high-dose cohorts with and without IL-2
CD19	Pediatric leukemia and	Retrovirus	CD28-CD3ζ	I	National Cancer Institute	
CD19	CLL, small lymphocytic lymphoma; MCL, FL, LCL	Retrovirus	CD28-CD3ζ	I/II	National Cancer Institute	IL-2

Source: Maus, M. V. et al., *Blood*, 2014, 123(17) (adapted); Jefferies.

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Exhibit 16: Ongoing CAR T-cell trials in hematologic malignancies (continued).

Antigen	Cancers	Gene transfer	CAR signaling domain	Phase	Sponsor	Cell type/selection/drug combination
CD19	B-cell malignancies relapsed post-allo-HSCT	Retrovirus	CD28-CD3ζ	I	National Cancer Institute	T-cells from donor
CD19	Auto-HSCT for NHL followed by T-cell infusion (day 2 or 3)	Lentivirus	CD3ζ	I/II	City of Hope	T_{cm}-enriched CD8+ T-cells
CD19/EGFRt	Auto-HSCT for NHL followed by T-cell infusion (day 2 or 3)	Lentivirus	CD28-CD3ζ	I	City of Hope	T_{cm}-enriched T-cells (cetuximab as possible suicide system)
CD19/EGFRt	Pediatric ALL	Lentivirus	CD28-CD3ζ	I	Seattle Children's Hospital	
CD19	Relapse/refractory CLL, NHL, or ALL	Lentivirus	CD3ζ	I/II	Fred Hutchinson Cancer Research Center	
CD19/EGFRt	ALL, DLBCL, MCL, NHL, CLL relapsed post-allo-HSCT	Lentivirus	CD28-CD3ζ	I/II	Fred Hutchinson Cancer Research Center	Donor-derived, CMV- or EBV-specific CD62L+ T_{cm}
CD19	Pediatric ALL post-allo-HSCT	Retrovirus	CD3ζ	I/II	University College, London	CD19 CAR-transduced EBV-specific CTLs from donor; 2nd cohort adds vaccination with irradiated EBV-LCL
CD19	ALL, CLL, NHL	Retrovirus	CD137-CD3z and CD3ζ	I/II	Chinese PLA General Hospital	
CD30	NHL, HL	Retrovirus	CD28-CD3ζ	I	Baylor College of Medicine	
CD30	NHL, HL	Retrovirus	CD28-CD3ζ	I	Baylor College of Medicine	CD30 CAR-transduced EBV-specific CTLs
CD30	Mycosis funcoides/CTCL	Retrovirus	CD28-CD3ζ	I	University of Cologne	
Ig κ light chain	Lymphoma, myeloma, leukemia	Retrovirus	CD28-CD3ζ	I	Baylor College of Medicine	
CD20	CD20+ leukemia and	Retrovirus	4-1BB-CD3ζ	I/II	Chinese PLA General Hospital	
CD33	Relapsed/refractory CD33+ AML	Retrovirus	CD137-CD3ζ, CD3ζ	I/II	Chinese PLA General Hospital	
CD138	Relapsed and/or chemotherapy	Retrovirus	CD137-CD3ζ, CD3ζ	I/II	Chinese PLA General Hospital	
Lewis-Y	AML, MDS, multiple myelmoa	Retrovirus	Anti-Lewis-Y-CD28-CD3ζ	I	Peter MacCullum Cancer Centre, Australia	

Source: Maus, M. V. et al., *Blood*, 2014, 123(17) (adapted); Jefferies.

Background

About NHL

Lymphoma is a cancer of the lymphatic system characterized by the formation of solid tumors in the immune system, and affects the lymphocytes (white blood cells). It is the most common blood cancer and has two main forms: Hodgkin's lymphoma (HL) (~10%) and non-Hodgkin lymphoma (NHL) (~90%). NHL is the most prevalent hematologic malignancy in the U.S., representing 4% of all malignancies in incidence and deaths per year (*Cancer Control* 2012).

The two main types of lymphocytes are B-cells and T-cells, and each performs different functions within the immune system. Both types can develop into lymphoma cells, but B-cell lymphomas are much more common in the U.S. than T-cell lymphomas. Lymphoid tissue can found in the spleen, thymus, adenoids and tonsils, digestive tract, bone marrow. The causes of lymphoma are unknown.

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NHL can be further classified as "indolent" (low-grade) or "aggressive" (high-grade), and the different subtypes are outlined in Exhibit 17. Some indolent NHL, which makes up ~30% of all NHL (Leukemia and Lymphoma Society) can transform into aggressive NHL, known as transformed indolent NHL.

Exhibit 17: Aggressive- and Indolent-NHL subtypes, including B-cell and T-cell lymphomas

Aggressive NHL-subtypes	Indolent NHL-subtypes
Diffuse large B-cell lymphoma (DLBCL)	Follicular lymphoma
Primary mediastinal large B-cell lymphoma	Lymphoplasmacytic lymphoma (Waldenstrom macroglobulinemia)
Follicular large cell lymphoma	Marginal zone lymphoma
Anaplastic large cell lymphoma	Splenic marginal zone lymphoma
Extranodal NK-/T-cell lymphoma	Primary cutaneous anaplastic large cell lymphoma
Lymphomatoid granulomatosis	
Angioimmunoblastic T-cell lymphoma	
Peripheral T-cell lymphoma	
Enteropathy-type intestinal T-cell lymphoma	
Intravascular large B-cell lymphoma	
Burkitt lymphoma/diffuse small noncleaved-cell lymphoma	
Lymphoblastic lymphoma	
Adult T-cell leukemia/lymphoma	
Mantle cell lymphoma	
Polymorphic posttransplantation lymphoproliferative disorder	
True histiocytic lymphoma	
Primary effusion lymphoma	

Source: National Cancer Institute; Jefferies.

According to the Surveillance Epidemiology and End Results (SEER) database, there are an estimated 70,800 new cases of NHL in the U.S. in 2014 (incidence). The Leukemia and Lymphoma Society estimates that there are approximately 558,340 patients living with DLBCL or are in remission in the U.S. (prevalence). Patients with relapsed/refractory DLBCL have a short life expectancy of only ~3-4 months (Pfreundschuh, M. et al., *Lancet Oncol*, 2006, 7, 379-91). DLBCL affects mostly middle-aged and older adults; the median age at diagnosis for NHL is 66 years of age (SEER 2007-2011). The 5-year survival of those with NHL is ~69.3%.

Diffuse large B-cell lymphoma (DLBCL) is the most common type of aggressive NHL subtype, making up ~20-30% of all newly diagnosed NHL cases in the U.S. DLBCL affects mostly middle-aged and older adults. Overall, DLBCL is aggressive but potentially curable with intensive combination chemotherapy. Primary mediastinal large B-cell lymphoma (PMBCL) is a less common subtype of diffuse large cell lymphoma (~10% of cases of DLBCL, *NEJM* 2013, 368, 15, 1408-1416) characterized by a significant fibrosis on histology and arises in the thymus. Some indolent NHL, which makes up ~30% of all NHL (Leukemia and Lymphoma Society) can transform into aggressive NHL, known as transformed indolent NHL. The Bastion study of 220 patients with follicular lymphoma (FL) showed a 22% and 31% transformation rate at 5 and 10 years, respectively. The probability of transformation in this study was relatively constant for the first 5 years after the initial FL diagnosis (Berstein, S. H. et al., *Hematology* 2009, 532-541 and ref therein).

DLBCL

Signs & Symptoms/Diagnosis. The first signs of DLBCL include painless rapid swelling in the neck, armpit, or grown, caused by enlarged lymph nodes. Swelling can eventually be painful. Sometimes, DLBCL doesn't begin in the lymph nodes but in other parts of the body (extranodal disease), including the stomach or bowel. Other symptoms include

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drenching night sweats, unexplained fevers (at least 100 ºF or 38 ºC), and weight loss (≥10% over the previous 6 months). Only 20-30% of patients have evidence of DLBCL in the marrow.

At physical examination, swollen lymph nodes will be felt. The physician may feel around the abdomen to examine the spleen and liver. If lymphoma is suspected, a biopsy will be performed. The most common forms of biopsy procedure include excisional biopsy (surgeon cuts through the skin to remove an entire lymph node for analysis) and incisional biopsy (surgeon removes only part of a large suspected lymph node). If lymphoma is confirmed, further testing is carried out to determine the stage of the cancer. There are four stages:

- Stage I (tumor is localized)
- Stage II (limited spread)
- Stage III (regional spread to an area or organ near primary lymph node tumor)
- Stage IV (lymphoma has spread beyond the lymphatic system to distant parts of the body)

The International Prognostic Index (IPI) was developed to predict response based on a retrospective analysis of >2,000 patients with aggressive lymphomas. There have two modifications that have been implemented that include an age-adjusted model for patients <60 yrs of age, and a revised IPI that consolidates five prognostic risks into 3: tumor stage, performance status, and lactate dehydrogenase (LDH) level. The age-adjusted model is accepted in standard practice.

Exhibit 18: Variables in the International Prognostic Index (IPI) and Age-Adjusted IPI

IPI	Age-adjusted IPI
Age >60 yrs	Advanced stage of disease (III, IV)
Advanced stage of disease (III, IV)	Elevated lactate dehydrogenase (LDH)
Extranodal involvement >1 site	Poor ECOG performance status ≥2
Elevated lactate dehydrogenase (LDH)	
Poor ECOG performance status ≥2	

Source: Cultrera, J. et al., *Cancer Control* 2012, 19, 3, 204-213; Jefferies.

Treatment. DLBCL is an aggressive lymphoma. It advances very quickly, and requires immediate treatment. However, aggressive types of NHL including DLBCL are often curable with intensive combination chemotherapy, commonly involving Rituxan. Rituxan is a monoclonal antibody targeting CD20, a common B-cell marker present on majority of malignant lymphoma cells in DLBCL. It was approved for DLBCL in February 2006. The most widely used treatment is R-CHOP: Rituxan + cyclophosphamide, doxorubicine, vincristine, and prednisone. R-CHOP has proved successful in curing 50-60% of patients with DLBCL and is the SOC for advanced-stage disease (Oncology/Hematology 2013 Review). Beyond R-CHOP, the following options may also be used as first-line therapy (NCCN Guidelines 2014):

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First-line therapy:

- R-CHOP = Rituxan (rituximab), cyclophosphamide, doxorubicin, vincristine, prednisone
- Dose-adjusted EPOCH = VP-16 (etoposide), prednisone, vincristine, cyclophosphamide, doxorubicin + Rituxan
- R-mini-CHOP (for patients >80 years of age with comorbidities)

First-line therapy for patients with poor left ventricular function or very frail:

- R-CEPP = Rituxan, cyclophosphamide, VP-16, prednisone, procarbazine
- R-CDOP = Rituxan, cyclophosphamide, liposomal doxorubicin, vincristine, prednisone
- R-CNOP = Rituxan, cyclophosphamide, mitoxantrone, vincristine, prednisone
- DA-EPOCH = VP-16, prednisone, vincristine, cyclophosphamide, doxorubicin + Rituxan
- R-CEOP = Rituxan, cyclophosphamide, VP-16, vincristine, prednisone

Approximately 30-40% relapse and 10% have refractory DLBCL among those who achieve and maintain complete remission after first-line therapy. Patients with r/r DLBCL have a poor outlook with a life expectancy of 3 to 4 months, if left untreated (Perry, A. R. et al., *Ann Oncol*, 1998, 9 (Suppl 1): S9-14; Fisher, R. I. et al., *NEJM*, 1993, 328, 1002-1006). The general treatment paradigm from the NCCN Guidelines (2014) for r/r DLBCL is outlined in Exhibit 19. Second-line therapy typically involves a platinum-based chemotherapy regimen with Rituxan, such as R-ICE or R-DHAP. If a second response is achieved, the patient may be eligible for stem cell transplant. If no response is achieved or the patient relapses again, the patient will receive best supportive care.

Other examples for second-line therapy are listed below (NCCN Guidelines 2014):

Second-line therapy:

- R-ICE = Rituxan ± ifosfamide, carboplatin, etoposide;
- R-DHAP = Rituxan ± dexamethasone, cisplatin, cytarabine;
- R-ESHAP = Rituxan ± etoposide, methylprednisolone, cytarabine, cisplatin;
- R-GDP = Rituxan ± gemcitabine, dexamethasone, cisplatin;
- R-GemOx = Rituxan ± gemcitabine, oxaliplatin;
- R-MINE = Rituxan ± mesna, ifosfamide, mitoxantrone, etoposide;
- R-CEPP = Rituxan ± cyclophosphamide, etoposide, prednisone, procarbazine;
- R-CEOP = Rituxan ± cyclophosphamide, etoposide, vincristine, prednisone;
- R-DA-EPOCH = Rituxan ± etoposide, prednisone, vincristine, cyclophosphamide, doxorubicin.

Patients who respond to second-line therapy may go on to receive hematopoietic cell transplantation (HCT). Patients who do not respond to second-line therapy or relapse after HCT may move onto salvage therapy. Salvage chemotherapy typically involves

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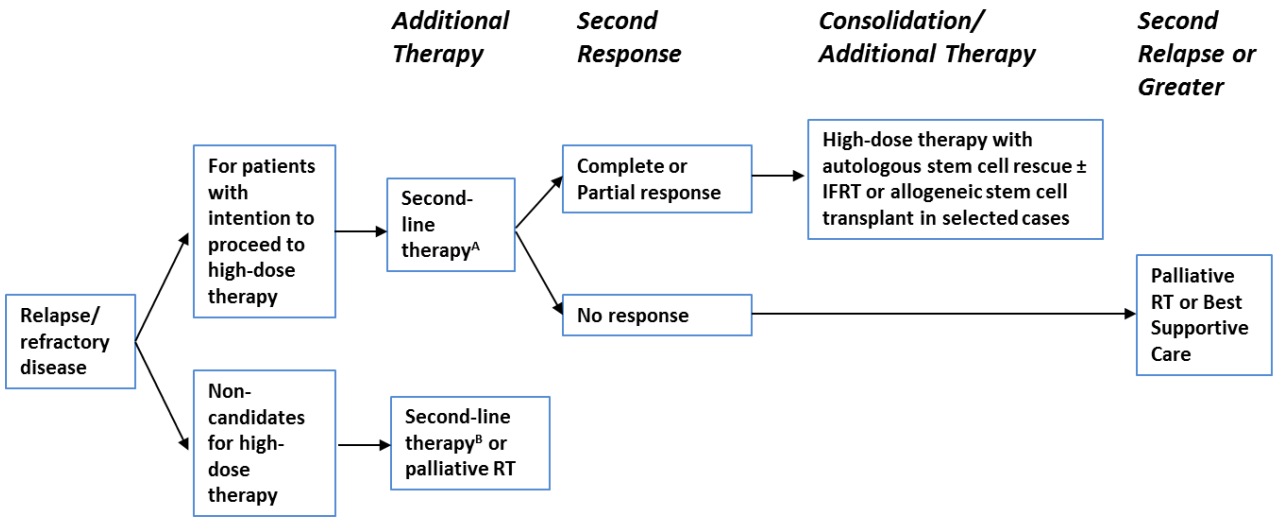
combination chemotherapy +/- Rituxan. Response rates vary from 30-60% but there are frequent relapses (Cultrera, J. et al., *Cancer Control* 2012, 19, 3, 204-213).

Exhibit 19: General treatment algorithm for relapsed/refractory DLBCL.



Second-line therapy (A): May include DHAP, ESHAP, GDP, GemOx, ICE, MINE, all ± Rituxan

Second-line therapy (B): May include bendamustine, CEPP, CEOP, DA-EPOCH, GDP, GemOx, Revlimid, all ± Rituxan

Source: National Comprehensive Cancer Network (NCCN) Guidelines (2014) (adapted), Jefferies.

Abbreviations: DHAP = dexamethasone, cisplatin, cytarabine; ESHAP = etoposide, methylprednisolone, cytarabine, cisplatin; GDP = gemcitabine, dexamethasone, cisplatin; GemOx = gemcitabine, oxaliplatin; ICE = ifosfamide, carboplatin, etoposide; MINE = mesna, ifosfamide, mitoxantrone, etoposide; CEPP = cyclophosphamide, etoposide, prednisone, procarbazine; CEOP = cyclophosphamide, etoposide, vincristine, prednisone; DA-EPOCH = etoposide, prednisone, vincristine, cyclophosphamide, doxorubicin.

Chronic lymphocytic leukemia (CLL)

Chronic lymphocytic leukemia (CLL) is a cancer of the B-cell lymphocytes in the blood and bone marrow. The leukemia cells build slowly over time in the bone marrow, and begin to crowd out normal cells. As a result, CLL patients do not make enough red blood cells, properly functioning lymphocytes, and platelets. Because it is slow, symptoms may not occur for several years. Leukemia cells can eventually invade other parts of the body including lymph nodes, liver, and spleen. It is the most common leukemia in the Western world with an incidence of 4.2/100,000 per year (*SEER Clinical Statistics Review*, 1975-2007). The American Chemical Society estimates 15,720 new cases of CLL and 4,600 deaths in 2014. CLL mainly affects older adults with the average age at time of diagnosis ~72 years. It is rarely seen in people <40 years of age. The exact causes of CLL are unknown. Certain chemical exposures, family history, and race/ethnicity may be risk factors. Signs/symptoms are often vague and commonly associated with other diseases, and include weakness, fatigue, weight loss, fever, night sweats, enlarged lymph nodes, and/or pain in the belly as a result of an enlarged spleen. In the U.S., CLL is staged using the Rai system, which divides CLL into 5 stages (0-IV). Stage 0 is considered low risk, stages I-II are considered intermediate risk, and stages III-IV are considered high risk.

First- and second-line therapies are listed in Exhibit 20, and the choice of therapies depends on age (NCCN Guidelines 2014) and genetics (with or without del(11q) or del(17p)). Patients with multiple r/r CLL have a poor prognosis with few effective

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treatment options (Brown et al, *Novel Treatments for Chronic Lymphocytic Leukemia*, 2014, ASCO Educational Book).

Exhibit 20: Recommended Treatments for CLL in first- and second-line.

Line of Therapy	Patient	Recommended Therapy
First-line Therapy (CLL without del(11q) or del(17p)	Age >70 years patients with comorbidities	Gazyva + Leukeran
		Treanda ± Rituxan
		Cyclophosphamide, prednisone ± Rituxan
		Rituxan
		Fludara ± Rituxan
		Cladribine
		Leukeran
	Age <70 years patients without significant comorbidities	FCR (fludarabine, cyclophosphamide, Rituxan)
		FR (fludarabine, Rituxan)
		PCR (Penostatin, cyclophosphamide, Rituxan)
		Treanda ± Rituxan
		Obinutuzumab ± Leukeran
Second-line Therapy (CLL without del(11q) or del (17p)	Long response	Retreat as in first-line therapy until short response
	Short response for age ≥70 years	Imbruvica
		Reduced-dose FCR
		Reduced-dose PCR
		Treanda ± Rituxan
		High-dose methylprednisolone (HDMP) + Rituxan
		Rituxan + Leukeran
		Arzerra
		Revlimid ± Rituxan
		Campath ± Rituxan
		Dose-dense Rituxan
	Short response for age <70 years	Imbruvica
		FCR
		PCR
		Treanda ± Rituxan
		Fludara ± Campath
		R-CHOP
		OFAR
		Arzerra
		Revlimid ± Rituxan
		Campath ± Rituxan
		HDMP + Rituxan
First-line Therapy (CLL with del(17p))		Campath ± Rituxan
		FCR
		FR
		HDMP + Rituxan
		Gazyva + Leukeran
Second-line Therapy (CLL with del(17p))		Campath ± Rituxan
		R-CHOP
		CFAR
		HDMP ± Rituxan
		Imbruvica
		Revlimid ± Rituxan
		Arzerra
		OFAR

Source: National Comprehensive Cancer Network (NCCN) Guidelines (2014) (adapted), Jefferies.

Acute Lymphoblastic Leukemia (ALL)

As in CLL, acute Lymphoblastic Leukemia (ALL) similarly describes a cancer of the lymphocytes that begins in the bone marrow, but "acute" term means that the leukemia

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can progress very quickly. The leukemia cells can invade the blood and spread to the lymph nodes, liver, spleen, central nervous system, and testicles. If not treated, ALL is likely to be fatal within a few months. The exact cause of ALL is not known, but some potential risk factors include radiation exposure, certain chemical exposure, certain viral infections, and race/ethnicity. Like CLL, ALL is several non-specific symptoms such as weight loss, fever, night sweats, fatigue, and loss of appetite. Leukemia cells may build up in the liver and spleen, causing a general swelling in the abdomen. ALL may also exhibit enlarged lymph nodes and bone or joint pain.

The U.S. prevalence for ALL is ~66,030 and the incidence is ~6,020 per year (American Cancer Society). The risk of developing ALL is highest in children <5 years of age, then declines slowly until mid-20's. About ~1/3 of ALL cases are adults. Age is one of the most important prognostic factors in ALL patients. The long-term survival rates are ~80% in children but only <30% in adults (Pui, C. H. et al, *NEJM* 2006, 354, 166–178). Patients with ALL can further categorized into Philadelphia chromosome-positive ALL (Ph+) (20-30% of adults and 2-3% of children with ALL) and Philadelphia chromosome-negative ALL (Ph-). The Ph chromosome results from a reciprocal translocation between chromosomes 9 and 22, which produces a fusion gene on chromosome 22 (BCR-ABL). BCR-ABL fusion proteins alter multiple signaling pathways which contribute to tumor growth and proliferation (Koo, H. H. *Korean J Pediatr* 2011, 54(3), 106-110).

Treatment will depend on what Ph category of ALL, and can include Oncovin or Marqibo, Cerubidine (daunorubicin), Adriamycin (doxorubicin), Cytosar (cytarabine), Elspar, Cytoxan, Decadron, or VP-16 (etoposide). Standard therapy fails in 10-20% of newly diagnosed patients (Hunger, S. P. et al., *J Clin Oncol* 2012, 30(14), 1663-1669). In adults, r/r B-cell acute lymphoblastic leukemia (B-ALL) have a very poor prognosis with an expected median overall survival of <6 months (Davila, M. et al., *Sci Transl Med* 2014, 6, 1-10; Fielding, A. K. et al., *Blood* 2007, 944-950; Gökbuget, N. et al., *Blood* 2012, 120, 2032-2041) For Ph-positive ALL patients, relapsed/refractory regimens can include dasatinib, nilotinib, bosutinib, and ponatinib. For Ph-negative ALL patients, r/r regimens can include clofarabine-containing regimens, cytarabine-containing regimens, or alkylator combination regimens (NCCN Guidelines 2014).

EGFRvIII-CAR-T

Kite is developing a CAR-T which targets the EGFRvIII protein over expressed in 30-50% of glioblastomas. EGFRvIII is a deletion mutant of EGFR (epidermal growth factor receptor) that leads to constitutively active EGFR and leads to enhanced tumorigenicity, and confers resistance to radiation and chemotherapy. EGFRvIII-CAR-T is currently being evaluated in a Phase I/II trial conducted in collaboration with NCI in recurrent glioblastoma patients who have failed on chemoradiation. The trial is an open label study and will enroll 160 patients with safety and PFS rate at 6 months as the primary end point. Secondary end points of the study include determination of survival percentage of engineered CAR cells in the patients and evaluate radiographic changes post treatment. Patients will have leukapheresis about a month before treatment and will have chemotherapy one week prior to treatment. EGFRvIII-CAR cells will be given along with IL-2 and will be infused every 8 hrs up to 15 doses. The trial will involve T3+3 design with a single patient representing a cohort for the first 3 cohorts. NCI plans to infuse 10^7, $3*10^7$ and 10^8 in the 1st, 2nd and 3rd cohorts respectively. If no DLTs are observed then dose escalation cohorts (up to 8 cohorts with 3pts/cohort) will be initiated and will be infused with $3*10^8$ to $3-6*10^9$ EGFRvIII CART-T cells/patient. Inclusion criteria for the trial includes patients >18-66yrs who have failed on prior standard treatment, have Karnofsky score >60 and exhibit expression of EGFRvIII. Exclusion criteria for the trial includes patients who had Gliadel implantation within six months and FEV1 $\leq$60%. EGFRvIII CAR vector used for this study is

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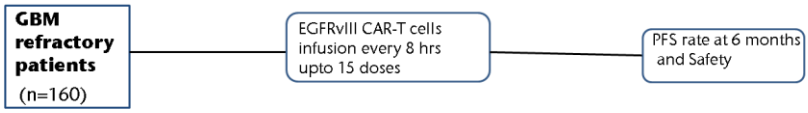
a lentiviral vector which co-expresses co-stimulatory molecules CD28, 4-1BB and CD3. Final data from the trial is expected in 2018 with interim data updates expected to be released at key annual oncology medical meetings. Kite is going to file an IND and initiate clinical trials in 2016.

Exhibit 21: Phase I/II design of EGFRvIII CAR in GBM



Endpoints
Primary:
- PFS rate at 6 months
- Safety

Secondary:
- Overall survival
- In vivo survival rate of CAR-T cells

Inclusion Criteria:
- EGFRvIII expressing refractory GbM
- >18-66 yrs
- Karnofsky score >60
- 3 months treatment with NSAI in metastatic setting

Exclusion Criteria:
- Pts who had gliadel transplantation
- FEV1 ≤60%

Source: Clinicaltrials.gov; Jefferies

NCI is conducting the phase I/II trial based on encouraging results observed in the preclinical studies where EGFRvIII-CAR-T cells was able to reduce/eradicating the glioma tumors burden in both in vitro and in vivo models. In vitro studies have shown that EGFRvIII CAR vector when transduced into T cells from normal and glioblastoma patients were able to recognise and become activated when challenged with EGFRvIII glioma cell lines as judged by cytokine production and cytolytic activity. EGFRvIII cells were not activated when challenged with non EGFRvIII expressing cells suggesting the specificity of EGFRvIII CAR-T cells.

In addition EGFRvIII CAR-T cells were able to block the formation of neurospheres (Morgan RA et al *Hum Gen Therap* 2012; Choi BD et al, *J Clin Neuroscience* 2013). Neurospheres are mass of glioma cells formed from division of individual glioma stem cell. EGFRvIII CAR T cells prevented neurosphere formation, and were able to destroy preformed neurospheres suggesting that EGFRvIII CAR-T cells are also able to kill glioma stem cells. These favorable preclinical data of EGFRvIII CAR-T suggest a possible favorable outcome for EGFRvIII CAR-T cells in GBM in clinical trials.

Other studies also back up the efficacy of CAR-T cells in GBM with several investigators reporting anti-GBM activity of CAR T cells in preclinical models specific for EphA2, IL-13Rα2, or HER2. These CAR-T cells recognized GBM cell lines or primary GBM samples in an antigen dependent manner as judged by cytokine production and cytolytic activity. In vivo, autologous CAR T cells had potent antitumor activity in human glioma xenograft models after local T-cell injection as judged by the prolonging of survival of mice when compared to mice not treated with CAR-T cells. (Chow KKH et al Mol Therap 2013; Ahmed N et al *Clin Cancer Res* 2010; Kong S et al *Clin Cancer Res* 2012; Brown CE et al Clin *Cancer Res* 2012; Morgan RA et al *Mol Therap* 2010).

So far clinical experience with CAR-T cell therapy for patients with GBM is limited. The safety and efficacy of intratumoral injection of T cells expressing a first generation IL-

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13Rα2-specific CAR (IL13-Rα2-CAR T cells) has been evaluated in one clinical study (Briown CE et al, Mol Therap 2011). Infusion of IL13-Rα2-CAR T cells was well tolerated and associated with clinical benefit in 3 patients. Though no adverse events have been reported in GBM, however, there was a patient death reported with HER2 CAR cells in metastatic colon cancer. The death was mostly attributed to off-target effect of recognition of low levels of Her2 expressed on lung epithelial cells (Morgan RA et al *Mol Therap* 2010).

EGFRvIII GBM Market. Despite being the most common form of primary brain tumor, GBM remains a significant unmet medical given limited treatment options with somewhat poor prognosis including OS of less than two years (albeit there has been some improvement in recent years). Based on the Surveillance, Epidemiology and End Registry (SEER) database, we estimate a prevalence of brain and CNS tumors of ~145K in the U.S. in 2012, of which ~75% or 109K patients have primary tumors (versus secondary brain/CNS metastases from other cancers). Based on the National Comprehensive Cancer Network (NCCN) estimates, we estimate 65% or 71K are brain tumors, among which ~50% or 36K are GBM. The EGFRvIII subset, for which rindopepimut is being developed, accounts for 25-30% or 9-11K of GBM patients, comprising ~60% (5,400-6,600) newly diagnosed, ~25% (2,250-2,750) Avastin-naïve recurrent and ~15% (1,350-1,650) Avastin-refractory patients. We currently don't model EGFRvIII into our financial valuation and represents upside.

Competitive CAR-T in GBM

Novartis EGFRVIII CAR-T: Similar to Kite, Novartis is also developing EGFRvIII CAR-T cells for GBM. Novartis has licensed this technology from University of Pennsylvania and plans to initiate the phase I trial in Q4 2014. Novartis is proceeding with the EGFRvIII CAR vector which had shown promising results in preclinical murine glioma models. Mice injected with NVS CAR-EGFRvIII cells completely eradicated the tumor in a dose dependent manner and were able to generate resistance to EGFRvIII –ve gliomas (Sampson JH et al, *Clin Cancer Res* 2013).

Her2 CAR-T: Baylor College of Medicine is conducting a Phase I study evaluating the Her2 CAR-T cells in GBM. Approximately 80% of the GBMs express Her2. The trial will enrol 18 patients with dose limiting toxicity and safety of dose escalation as primary end points. Secondary end points of the study include the reduction of tumor burden. In vivo autologous HER-2 CAR-T developed from GBM patient has shown to significantly prolong the survival of mice engrafted with human glioma.

EGFRvIII Mutant. The mutant EGFRvIII expression is currently understood to be limited to cancerous cells of certain tumors including GBM and virtually absent in normal cells/tissues. An estimated 30-50% of GBM patients are believed to have tumors overexpressing EGFRvIII on the cell surface, potentially enabling therapeutic approaches targeted at EGFRvIII. Biologically, EGFRvIII is considered a constitutively active mutant of the normal EGFR with increased activity, conferring the ability for cells to become cancerous (i.e. transforming). EGFRvIII overexpression may also confer resistance to TMZ, predicting a poor outcome following RT/TMZ+TMZ. In GBM cells, EGFRvIII exists as extra chromosomal fragment; however when GBM is treated with radiation and chemotherapy the extra chromosomal fragment integrates into chromosomes and provides resistance to the GBM cells. Overall, EGFRvIII is a negative prognostic factor in GBMs and patients with EGFRvIII mutant have shown to have worse survival than patients with wild type EGFRvIII. Proportion of cells overexpressing EGFRvIII within a tumor can be highly variable, ranging 10% to >90%. EGFRvIII overexpressing cells are theorized to exist in discrete "islands" within the tumor with potentially stem cell-like functions driving proliferation, as well as, secretory functions driving growth and/or survival of neighboring cells through paracrine growth/survival factor signaling.

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NY-ESO-1 TCR – Re-engineering T-cell Receptor To Target Cancer

Kite has licensed re-engineered T-cell receptor technology from NCI that targets NY-ESO-1 (New York oesophageal squamous cell carcinoma-1) antigen that is over expressed in multiple cancers but not in normal cells. NY-ESO-1 one has been found to be over expressed in 20-80% of cancers that include melanoma, GBM and synovial cancers. In engineered TCRs, a retroviral vector encoding antigenic specific T-cell receptor is expressed in T-cells. These T-cells recognize the antigen on the tumor in MHC (major histocompatibility) specific manner and kills the tumor cells. MHC is antigen presenting molecules and without MHC, T-cells cannot recognise the antigens. There are two types of MHC; MHC I and MHC II, which are encoded by human leukocyte antigens genes (HLA). In a Phase I/IIa trial conducted by NCI, NY-ESO-1 TCR exhibited encouraging results in NY-ESO-1/HLA A2 positive melanoma and synovial sarcoma. HLA 2 encodes for MHC I that mostly activate CD8 T cells. Kite is going to file an IND for a single arm phase I in synovial and bladder cancer in Q4 2015 and initiate the Phase I in early 2016.

NY-ESO-1 TCR Shows Encouraging Results in Phase I/IIa: In a NCI conducted Phase I/IIa study in 19 melanoma and 15 synovial sarcoma patients, treatment with NY-ESO-1 TCR led to encouraging objective responses. In the melanoma patients, objective responses were found in 53% of patients with 6 PR and 4 CR and the duration of response lasting 5-50+ months in CR and 3-9+ months for PR. In the synovial sarcoma patients, objective responses were found in 67% of patients with 9 PR and 1 CR and the duration of response lasting 5+ months in CR and 3-31+ months for PR. MRI scans of these patients showed complete tumor regression in patients with a CR. Majority of the objective responses were detected within one month of TCR cells infusion. Neutropenia and thrombocytopenia were the transient toxicities observed but were not attributed to the administered T-cells.

Exhibit 22: Summary of responses observed in NY-ESO-1 TCR treated melanoma and synovial cancer patients

A

Cancer type	Sample size	CR (%; duration of response)	PR (%; duration of response)	PD (%)
Refractory melanoma	19	4 (21%; 5-50+ months)	6 (32%; 3-9+ months)	9 (47%)
Refractory synovial sarcoma	15	1 (6%; 5+ months)	9 (61%; 3-31+ months)	5 (33%)

Source: Company presentation; Jefferies

Patients enrolled in the trial were >18 yrs and had high expression of NY-ESO-1 and received 2-4 prior treatments, which included radiation, surgery, chemotherapy and immunotherapy. All the patients presented with multiple metastatic lesions at various anatomic sites that include lymph nodes, lung and bone. Patients received a median of $5*10^{10}$ NY-ESO-1 TCR cells after initial chemotherapy (range: $16\text{-}130*10^{9}$). A median of 78% and 65% of the transferred CD8 T and CD4 T cells respectively, bound the NY-ESO-1 tetramer, and a median of 92% of total CD3 cells bound an anti-V-13.1 antibody, which was reactive with the β chain of the transduced TCR. Approximately 1 month after transfer, between 2-60% of the CD8 T cells and 4-45% of CD4 T cells from peripheral blood stained bound to NY-ESO-1 tetramer. Similarly between 3-85% of CD8 T cells and between 3-63% of CD4 T cells expressed Vβ13.1 (Robbins PF et al *JCO* 2011).

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Exhibit 23: Baseline patient characteristics of patients and NY-ESO-1 TCRs at administration and after one month

					NY-ESO-1 tetramer positive at infusion			NY-ESO-1 tetramer positive after 1 month	
Patients	Age	No: of prior treatments	NY-ESO-1 TCR cells infused (*10^9)	No: of IL-2 doses	% of CD8	% of CD4	Vβ13.1 positive cells	% of CD8	% of CD4
Melanoma									
1	52	3	130	6	86	64	94	13	14
2	60	2	71	6	76	53	90	60	38
3	30	3	47	1	80	65	91	2	<1
4	56	3	50	7	80	74	94	3	<1
5	32	3	64	4	85	76	94	11	<1
6	38	2	51	7	87	79	94	<1	<1
7	47	3	23	7	70	58	90	18	7
8	39	4	38	8	78	70	94	11	19
9	51	3	31	10	83	69	96	39	26
10	61	4	16	8	79	56	92	<1	<1
11	46	3	37	6	63	58	85	7	7
Synovial cancer									
12	20	4	83	5	77	64	91	45	45
13	37	3	50	8	78	78	93	6	6
14	47	3	56	8	81	76	91	24	24
15	19	4	16	5	67	63	89	12	12
16	30	2	59	5	74	57	88	4	4
17	40	3	52	5	78	69	92	<1	<1

Source: Robbins PF et al JCO 2011; Jefferies

KITE Proceeds with Murine TCR Targeting NY-ESO-1 in Phase I: In the NCI sponsored Phase I/IIs several partial responses spanning short durations were observed and one of the potential reasons for this short term duration of response is the formation of mixed TCR dimers formed between endogenous and transduced TCRs that have less affinity for NY-ESO antigen. In mice it has been shown that mixed TCR leads to self-reactive TCRs leading to cytokine driven autoimmune responses. Therefore it's important to create a TCR which doesn't form mixed dimers with endogenous TCR. In the Kite sponsored phase I, the company will utilize a murine TCR which recognises human NY-ESO-1/HLA A2 and will not form mixed dimers with endogenous human TCR. In preclinical models, mTCR showed high avidity for the human NY-ESO-1 antigen. Peripheral blood cells from donors transduced with NY-ESO-1 mTCR maintained TCR expression in both short term and long term cultures with efficiency ranging from 50-90% in 7-11 days after stimulation and 46-82% in 10-20 days after restimulation. In addition, mTCR exhibited better or similar efficacy in recognising the NY-ESO-1 tetramers or Vβ13.1 in preclinical models (Rosati SF wt al J Immunotherapy 2014).

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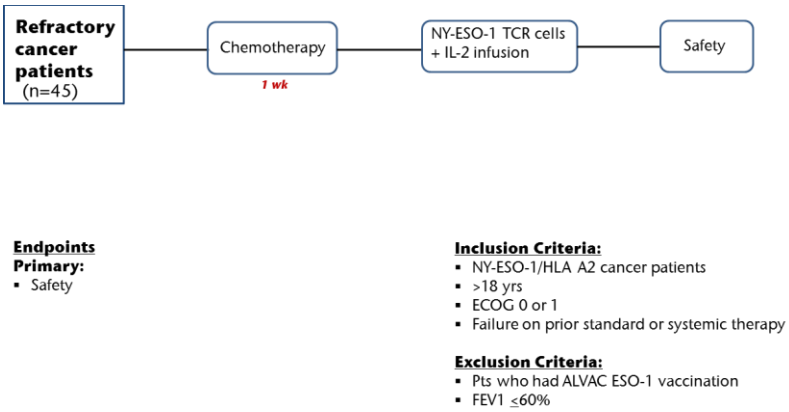
NCI Sponsored Phase I/II design: The NCI sponsored NY-ESO-1 TCR study is an open label 45 patient study with safety as the primary end point. Patients will have chemotherapy for 1 week followed by infusion of hTCR cells and IL-2. IL-2 will be dosed at 720K IU/kg every 8 hrs with a maximum of 15 doses. Patients will have a recovery time of 1-2 weeks following the infusions and return to clinic monthly for follow-ups. Thrombocytopenia and neutropenia has been observed following chemotherapy and T-cell infusion hence patients are required to remain in the hospital for 1-2 weeks post-infusion to monitor for safety. Inclusion criteria for the trial includes patients >18yrs who have failed on prior standard treatment, have ECOG of 0 or 1, exhibit NY-ESO-1 expression with HLA A2 MHC and have not had prior systemic or IPI treatment within the last 4-6 weeks of enrolment. Exclusion criteria for the trial includes patients who had prior ALVAC ESO-1 vaccine treatment, FEV1 ≤60% or immunodeficiency.

Exhibit 24: NCI sponsored NY-ESO-1 TCR Phase I/II design



Source: Clinicaltrials.gov; Jefferies

Other NCI Sponsored TCR Programs

NCI Conducting a Phase II Trial Using NY-ESO-1 TCR Transduced into CD62+ T-Cells: NCI is conducting a 22 patient open label Phase II study in refractory melanoma evaluating NY-ESO-1 TCR transduced into CD62L+ T-cells with ORR as primary endpoint. Patients will have chemotherapy for 1 week followed by infusion of hTCR cells and IL-2. IL-2 will be dosed at 720K IU/kg every 8 hrs up to maximum of 15 doses. Patients will have a recovery time of 1-2 weeks following the infusions and return to clinic monthly for follow-ups. Inclusion criteria for the trial includes patients >18yrs who have failed on prior standard treatment, have ECOG of 0 or 1, exhibit NY-ESO-1 expression with HLA A2 MHC and have not had prior systemic or IPI treatment within the last 4-6 weeks of enrolment. Exclusion criteria for the trial includes patients who had prior ALVAC ESO-1 vaccine treatment, FEV1 ≤60% or immunodeficiency. CD62L is a marker which is expressed by naive T-cells that have not been exposed to antigens. Following encounter with antigens, T-cells experience an expansion and then a contraction phase, which leads to a long-lived pool of memory cells. Central memory T-cells express high CD62L and cause the release of cytokines whereas effector memory expresses low CD62L (Sallusto F et al *Nature* 1999; Richard H et al *J Immunology* 2008). CD62+ central memory cells display a capacity for self-renewal, and have been reported in animal studies to lead to sustained engraftment and potent anti-tumor efficacy in adoptive immunotherapy (Berger C et al J Clin Invest 2008; Klebanoff CA et al *PNAS* 2005). Hence NCI is using these cells for the clinical trial to

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see if transducing NY-ESO in CD62L+ve cell can lead to higher ORR when compared to NY-ESO-1 transduced in unsorted T-cells.

GD2-CAR-T Phase I Trial Initiated In Pediatric Sarcoma. NCI have begun treating up to 36 pediatric patients with osteosarcoma and other non-neuroblastoma GD2-expressing solid tumors. Positive expression is defined as a 2+ expression in >50 percent of tumor cells. A third generation CAR-T utilizing OX40-CD28 costimulatory domains to drive T-cell expansion. The NCI employed a third generation construct based on preclinical studies observing T-cell exhaustion may impact CAR-T efficacy, and a CAR-T targeting GD2 and only utilizing a CD28 signaling domain may induce early exhaustion whereas the addition of a co-stimulatory domain such as 4-1BB or OX40L may mitigate T-cell exhaustion. Data from animal models with GD2-28Z CAR-T observed tumor growth post day 10. Further preclinical observations suggest lack of anti-tumor efficacy may be related to increased expression of PD1/TIM3/LAG3 when compared to CD19-CD28Z CAR-T construct.

The trial consists of 4 cohorts of at least 3 patients each with an expanded group of at least 12 patients, including at least 6 with osteosarcoma, receiving the highest dose. The Phase I dose-escalation will administer four potential doses- 1 x 10^5, 1 x 10^6, 3 x 10^6, and 1 x 10^7 cells/kg. Patients will receive escalating doses of autologous T-cells engineered to express a third-generation CAR equipped with Bellicum's CaspaCIDe safety switch. Patients with a PR or SD may receive a second cycle of cells at the next higher dose level after a minimum of 60 days after completion of the 1st cycle.

Patients experiencing a >Gr3 toxicity, or a Grade 2 toxicity that is believed to be causing substantial risk to the patient, will receive an infusion of AP1903 (a total of 6 doses may be administered). All patients will be measured for tumor response. Those receiving AP1903 will be evaluated for its effects. The study could require 2-3 years to finish enrolment, and therefore data could be available in late '16/early '17.

MART-1 TCR Program: MART-1 TCR was first genetically engineered TCRs tested in clinical trials. In a Phase I trial conducted by NCI, 13% (4 PR) response rate was found in 31 metastatic melanoma patients treated with MART-1 TCR T cells (Morgan RA et al Science 2006). The poor response rate was attributed to low avidity of the MART-1 TCR. In a second clinical trial conducted by NCI using second generation gene modified MART-1 TCR in 21 patients, ORR was observed in 30% of the patients. Several toxicities were observed with second generation MART-1 TCR, which included skin, eye and ear rash, hearing loss and uveitis which required steroid administration (Johnson LA et al Blood 2009). In a collaboration trial of NCI and Jonsson Comprehensive Cancer Center in 13 metastatic melanoma patients, treatment with MART-1 TCR cells and MART-1 DC vaccine resulted in an ORR of 69% (Chodon T et al Clin Can Res 2014).

MAGE A3 TCR Programs: NCI is conducting two Phase I/II trials evaluating MAGE-A3 in metastatic tumors: 1) a 107 patient trial for MAGE A3 and HLA-DP0401/0402; and 2) 107 patient trial for MAGE A3 and HLA-A*01. The primary objective of the study is identifying safe dose and ORR. Both trials will start with initial doses of 10^7 cells and will eventually test doses up to 10^{11} cell/patient depending on the safety profile. Patients will have chemotherapy for 1 week followed by infusion of hTCR cells and IL-2. IL-2 will be dosed at 720KIU/kg every 8 hrs with maximum of 15 doses. MAGE-A3 expression is found in 30-80% of tumor patients with expression range depending on tumor indication. MAGE-A3 is expressed in a variety of common epithelial malignancies including cancers of the lung, breast, ovary, bladder, and melanoma. In a small pilot study using MAGE A3/12 in 9 metastatic tumors, tumor regression was observed in 5 patients. However the study was terminated due to severe neural toxicities resulting in the death of two patients. Autopsy of the dead patients revealed necrotizing leukoencephalopathy in brain. Another patient

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developed Parkinson-like symptoms which resolved after 4 weeks (Morgan RA et al J Immunotherapy 2013). In another study conducted by Adaptimmune and University of Pennsylvania, two patients who received MAGE-A3 TCR died of cardiac failure. It was later identified that cardio toxicity was attributed to the similarity of the antigen epitope shared by titin and MAGE-A3 protein that MAGE-A3/12 TCR was targeting (Cameron BJ et al Sci Trans Medicine 2013).

gp100 TCR Program: In a Phase I trial conducted by NCI in 16 metastatic melanoma patients evaluating gp100 TCR T cells, objective responses were found in 19% of the patients (Morgan RA et al *Science* 2006). The trial was terminated owing to low ORR and severe toxicities observed in the study. Toxicities included skin, eye and ear rashes, uveitis and hearing loss. Many of the patients with uveitis toxicity required steroid administration (Johnson LA et al Blood 2009).

P53 TCR Program: p53 gene is found mutated/deregulated in nearly 50% of tumors and makes it an ideal candidate for adoptive immunotherapy. p53 functions includes cell cycle regulation, apoptosis induction, DNA repair, hence mutation/deregulation of P53 leads to tumorigenesis. In a Phase I trial conducted by NCI ORR of 11% was observed in 9 metastatic cancer patients treated with gp100 TCR T cells (www.clinicaltrials.gov). No severe adverse events were found in this study.

Exhibit 25: Summary of completed and ongoing NCI TCR Programs

TCR Antigen	Clinical trial ID	Tumor indication	Sample size	Response rate	Study status
NY-ESO-1 in CD62+ve T cells	NCT02062359	Metastatic melanoma		Awaiting results	Ongoing
MART-1	NCT00509288	Metastatic melanoma	31	13%	Completed
MART-1	NCT00923195	Metastatic melanoma	21	30%	Completed
MART-1 + MART-1 DC vaccine	NCT00910650	Metastatic melanoma	13	69%	Ongoing
MAGE-A3	NCT01273181	Metastatic cancer	9	56%	Terminated due to toxicities
MAGE-A3/HLA-DP4	NCT02111850	Metastatic cancer	107	Awaiting results	Ongoing
MAGE-A3/HLA-A1*01	NCT02153905	Metastatic cancer	102	Awaiting results	Ongoing
gp100	NCT00509496	Metastatic melanoma	16	19%	Terminated due to toxicities
P53	NCT00393029	Metastatic cancer	9	11%	Completed

Source: Jefferies

Competitive NY-ESO-1 TCR

Adaptimmune NY-ESO-1 TCR: Adaptimmune (Private, NC), a UK-based company, is developing NY-ESO-1 TCR for solid and liquid tumors based on the technology licensed from University of Pennsylvania. Adaptimmune has an individual NY-ESO-1 TCR technology and combination of NY-ESO-1 and LAGE-1 TCR technology. In a phase I/II clinical trial with NY-ESO-1/LAGE-1 combination TCR in 21 multiple myeloma patients, a best response rate of 77% was observed at 100 days and 65% of these responses are continuing till date. Adaptimmune is conducting a phase I/II using individual NY-ESO-1 TCR in 6 refractory melanoma patients results of which are due in early 2015. In June 2014, Adaptimmune entered into a strategic deal with GSK for the development and commercialisation of its NY-ESO-1 TCR program. Adaptimmune suggested that the deal also involves other target candidates but did not disclose them.

NY-ESO-1 Background. The promise of NY-ESO-1 as a candidate for specific immune recognition of cancer comes from its restricted expression in normal tissues but frequent occurrence in cancer. Although originally described from the complementary DNA sequences of an esophageal tumor, NY-ESO-1 has shown a much more widespread incidence in a number of other tumor types. Expression of NY-ESO-1 protein has been

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observed in approximately 10-50% of melanoma, breast, prostate, lung, ovarian, thyroid, and bladder cancers and has been suggested to lead to poor prognosis and presents as an early marker for recurrence (Chen YT et al *PNAS* 1997; Barrow C et al *Clin Can Res* 2006; Goydos JS et al J surg Res 2001; Gure AO et al *Clin Can Res* 2005). In melanoma it has been shown that NY-ESO-1 is found only in metastatic patients but is not seen in early stage patients.

Other TCR Programs

SSX2 TCR

Kite is developing an engineered TCR therapy targeting SSX2 (synovial sarcoma X chromosome breakpoint) antigen for refractory solid tumors. Similar to NY-ESO-1, SSX2 is silent in most normal adult tissues and expressed in various malignant tumors. Expression of SSX2 in tumors has been associated with advanced stages and worse patient prognosis (Taylor BJ et al *J Immunotherapy* 2005). Although the precise function of SSX2 remains unknown, its expression has been associated with stem cell migration, suggesting a potential biologically important role to the metastatic phenotype (Cronwright G et al *Cancer Res* 2005). Kite is licensing the SSX2 TCR technology from NCI. In preclinical experiments, engineered TCR T cells were very specific in targeting SSX2 and didn't show any off target binding (Abate-Daga D et al *Plos One* 2014). NCI is initiating phase I clinical trials of SSX2 TCR in solids tumors in Q4 2014 with the results expected in 2015. Kite will file for IND and initiate clinical trials in early 2016.

Engineered TCRs - Background

TCR is expressed on the surface of T lymphocytes and recognizes antigens presented by major histocompatability complex (MHC) proteins. Antigen specificity is determined by the TCR heterodimer, which is composed of two chains, either αβ or γδ. Genes encoding the TCR α and β chains are molecularly cloned into retroviral or lentiviral vectors and then transduced into autologous T-cells. Identification of large numbers of tumor specific antigens such as NY-ESO-1, MAGE A-1/3, SSX2 have been essential to the development of TCR based immunotherapy. Several studies have demonstrated that transfer of a tumor antigen-specific TCR into T cells results in an antigen-specific T cell population (Morgan RA et al *J Immunology* 2003; Schaft N et al *J Immunology* 2003). Preclinical studies and clinical studies have shown that engineered TCR T cells secrete immunostimulatory cytokines (IFN-γ, IL-2, and TNFα) and exhibit antigen-specific cytotoxicity upon encounter with antigen positive tumor cells (Zhao Y et al *J Immunology* 2005; Chinnasamy N et al *J Immunology* 2011).

One of the potential problems in ectopic expression of tumor-specific TCR is the mixed dimers between formation between endogenous and introduced TCR chains resulting in less binding to target or novel reactivity that may be deleterious to the host. Hence several strategies are being developed to overcome this problem. The first strategy focuses on developing murine-human hybrid TCRs in which the constant region of the human TCR chains was replaced by their murine counterparts (Cohen CJ et al *Can Res* 2006). Another approach to increase the specific pairing of TCR chains was to introduce additional cysteine residues within the constant region of the TCR α and β chains or add leucine zipper motif to hybrid TCR (Cohen CJ et al *Can Res* 2007; Voss RH et al J Immunology 2008). These strategies demonstrated to enhance TCR expression and therefore improve the functional activity by inducing higher levels of cytokine release and cytolytic activity. Some studies have developed TCRs use γδ dimers instead of αβ, however, the function and persistence of γδ T cells in adoptive cell therapy are not well studied. Recently a group has developed complete murine TCR that recognizes human antigen (Rosati SF wt al *J Immunotherapy* 2014).

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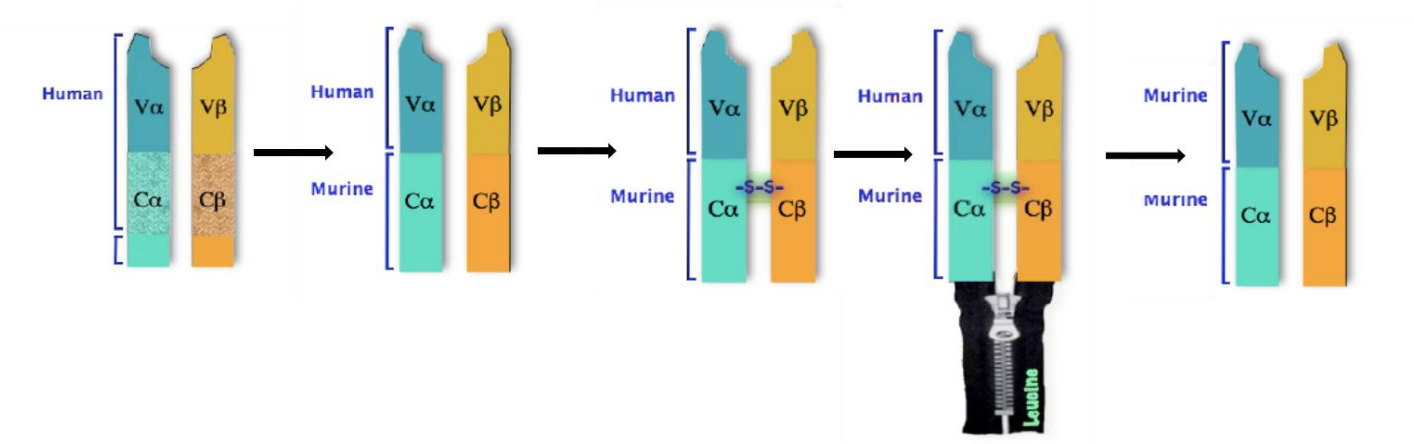
Exhibit 26: Evolution of engineered TCR technology to avoid mispairing of TCR chains



Source: NIH Immunotherapy conference; Jefferies

Differences between TCR and CAR-T Technology: Though both CAR-T and engineered TCR technologies are characterized into adoptive T cell immunotherapy, however significant differences exist between these technologies which are mentioned below. TCRs require the MHC expression for activation and formation of TCR complementarity to the MHC presented antigen, which defines the potential for signal amplification. The greater the affinity of binding of TCR to MHC presented antigen, the greater the signal amplification and efficacy. However CAR-T cells do not require MHC expression and its signal is completely dependent on the antigen and the co-stimulatory molecules being expressed by the vector. TCRs can recognize internal proteasome due to the MHC presentation of antigens, whereas efficacy of CAR-T cells depends on the expression of antigens on cell surface. Because of the clonal evolution of the engineered TCRs, memory T-cells recognizing the antigen is formed leading to lifelong persistence of the TCR T-cells whereas CAR-T cells have shorter persistence in the body.

Exhibit 27: Differences between CAR and TCR T cells technologies

Engineered TCR technology	CAR technology
Requires MHC expression	MHC independent
Requires HLA matching on tumor cell	HLA independent
Life long persistence	Decade long persistence
Low avidity	Avidity controllable
Signal amplification derived by evolution	Signal amplification dependant on synthetic biology
Toxicity difficult to predict	Toxicity can be predicted
Targets intracellular proteome	Targets surface structures

Source: Jefferies

Capital Structure

KITE currently has approximately $202.5 million in cash and equivalents. As of March 31, 2014, KITE had $18.6 million in cash and equivalents. In April 2014, KITE entered into a

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note purchase agreement with investors for the sale of $50.0 million in convertible promissory notes. The 2014 notes accrue interest at a rate of 6.0% per annum and will become payable on the earlier of October 25, 2016 or the occurrence of default. On June 20, 2014, KITE announced the pricing of its initial public offering of 7,500,000 shares of common stock at a price of $17/share, netting a $133.9 million in net proceeds.

Collaborations

KITE has entered into a five-year CRADA research collaboration with the National Cancer Institute expiring on August 30, 2017. Under the collaboration, KITE provides $1M annually in R&D funding to NCI. The collaboration provides KITE an option to negotiate licenses from NIH relating to CAR-T and TCR programs, and may exercise a right to negotiate a license by providing four months written notice after either 1) KITE receives a patent application covering the invention or 2) the date KITE files a patent application for an invention. KITE has a 10-month period to negotiate the license. Upon first market approval for a CAR-T/TCR program, KITE is obligated to pay $6 million milestone to NCI. KITE pays a $50,000 payment on start of KITE's first sponsored human trial of a licensed product in the U.S. KITE is also obligated to make milestone payments of up to $7 million on net sales of up to $1 billion, and royalties in the mid-single digits.

Management Team

Arie Belldegrun, M.D., FACS – Chairman, President & Chief Executive Officer

Dr. Belldegrun has been closely involved with the founding and advancement of several successful private and public biopharmaceutical companies. In 1996 he founded Agensys, Inc., a biotechnology company, and served as its founding Chairman of the board of directors and as a board member until 2007, when it was acquired by Astellas Pharma Inc. Dr. Belldegrun was also the founding Vice-Chairman of the board of directors and Chairman of the scientific advisory board of Cougar Biotechnology, Inc., a biotechnology company, from 2003 to 2009 when it was acquired by Johnson & Johnson. Dr. Belldegrun is Professor of Urology, holds the Roy and Carol Doumani Chair in Urologic Oncology, and Director of the UCLA Institute of Urologic Oncology at the David Geffen School of Medicine at UCLA. He was at the National Cancer Institute/NIH, as a research fellow in surgical oncology and immunotherapy. Dr. Belldegrun completed his M.D. at the Hebrew University Hadassah Medical School in Jerusalem, his post graduate studies in Immunology at the Weizmann Institute of Science, and his residency in Urologic Surgery at Harvard Medical School. He has authored several books and more than 400 scientific and medical papers related to urological cancers, immunotherapy, gene therapy, and cancer vaccines. He is certified by the American Board of Urology, and is a Fellow of the American College of Surgeons and the American Association of Genitourinary Surgeons (AAGUS).

Cynthia M. Butitta – Chief Operating Officer, Chief Financial Officer

Ms. Butitta has over 20 years of leadership experience in both the biotechnology and high-technology industries. She was most recently Senior Vice-President and Chief Financial Officer of NextWave Pharmaceuticals, Inc., which was acquired by Pfizer in 2012. Prior to NextWave, Ms. Butitta served as Chief Operating Officer from 2001 to 2010 and Chief Financial Officer from 1998 to 2010 of Telik Inc., a public biotechnology company focused on the development of small molecule drugs for the treatment of cancer. While at Telik, Ms. Butitta was responsible for securing over $450 million through an IPO and subsequent follow-on offerings. She also was responsible for SEC reporting, financial controls, investor relations, information technology, manufacturing, quality, project management and commercial operations. Ms. Butitta received a B.S. degree with honors in Business and Accounting from Edgewood College in Madison, Wisconsin and a MBA degree in Finance from the University of Wisconsin, Madison.

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Margo R. Roberts, Ph.D. - Chief Scientific Officer

Dr. Roberts has more than 23 years of biomedical research experience in both biotechnology and academia. In her capacity as Principal Scientist and Director of Immune and Cell Therapy at Cell Genesys, Inc. (1990-1998), Dr. Roberts led the development of chimeric antigen receptor (CAR) technology encompassing CAR design and function in T cells and stem cells, and related methodologies for T cell engineering. She oversaw the implementation of the CAR technology to HIV disease and under her leadership the CD4z CAR T cell research program culminated in the first CAR T cell clinical trial initiated in 1994. Dr. Roberts is inventor on the first set of CAR patents including 2nd generation CAR constructs that incorporate the costimulatory domains of receptors such as CD28 aimed at improving CAR T-cell survival and function. Dr. Roberts joined the University of Virginia for 13 years starting in 1999. She pursued interdisciplinary research in the area of immunity and inflammation. She is author on more than 25 scientific publications and inventor on 13 issued US patents and three published US patent applications related to CAR T-cell technology and tumor vaccine therapies. Dr. Roberts was a postdoctoral fellow at Yale University and at the Laboratoire de Génétique Moléculaire des Eucaryotes (LGME) of the CNRS in Strasbourg, France. She received both her B.Sc. with honors and her Ph.D. from the University of Leeds in England.

Jeffrey S. Weizorek, M.D., M.S. – Vice President, Clinical Development

Prior to joining Kite Pharma, Dr. Wiezorek held roles of increasing responsibility over 9 years at Amgen. His most recent position was as Executive Medical Director, Global Development, he had global oversight of the clinical strategy for the immunotherapy (including blinatumomab and talimogene laherparepvec), angiogenesis, and denosumab oncology product areas. Previously, he served as Global Development Leader for multiple programs encompassing phase 1 through phase 4 trials. In these roles, Dr. Wiezorek worked closely with global regulatory agencies, and he was the development lead for the approval of Vectibix in combination with chemotherapy in Europe. He received his B.A. degree in biophysics from the University of Pennsylvania and his M.D. degree from Columbia University. Dr. Wiezorek trained in internal medicine at Stanford University and also completed a fellowship in oncology at UCLA. Prior to joining Amgen, he investigated the role of nuclear factor-kappaB in cellular proliferation and cancer pathogenesis in the laboratory of Dr. David Baltimore at the California Institute of Technology.

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Exhibit 28: KITE Income Statement

Kite Pharma, Inc.

Quarterly Income Statement

(All values in $MM except EPS and average shares)

	2012A	2013A	2014E					2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
	FY	FY	1QA	2QE	3QE	4QE	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY
Revenue:																		
CD19 CAR-T U.S. Sales	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	63.4	185.1	393.4	635.5	805.4	933.7	964.7	996.5	1029.2
CD19 CAR-T EU Royalty	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	3.8	24.3	50.4	66.3	82.8	85.2
Total revenue, net	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**63.4**	**$ 185.1**	**$ 393.4**	**$ 639.4**	**$ 829.7**	**$ 984.1**	**$ 1,031.0**	**$ 1,079.3**	**$ 1,114.3**
Costs and expenses:																		
Cost of goods sold	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	46.3	98.3	158.9	193.3	214.8	212.2	209.3	205.8
Research & development	18	5.1	2.1	2.0	2.0	3.0	9.1	47.0	47.0	49.4	518	54.4	56.0	57.7	59.5	60.6	619	63.1
Selling, general & administrative	0.8	13	11	0.7	0.8	0.8	3.0	3.2	3.4	26.5	39.8	43.7	45.9	46.8	47.8	48.7	49.7	50.7
Total operating expenses	**2.6**	**6.4**	**3.2**	**2.7**	**2.8**	**3.8**	**12.1**	**50.2**	**50.4**	**75.9**	**137.9**	**196.5**	**260.8**	**297.9**	**322.0**	**321.6**	**320.8**	**319.6**
Income (loss) from operations	**(2.6)**	**(6.4)**	**(3.2)**	**(2.7)**	**(2.8)**	**(3.8)**	**(12.1)**	**(50.2)**	**(50.4)**	**(12.4)**	**47.3**	**196.9**	**378.5**	**531.9**	**662.2**	**709.4**	**758.5**	**794.7**
Other income (expense):																		
Miscellaneous (expense) income	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Interest income	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Interest expense	(0.0)	(0.0)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other	(0.0)	(14)	(0.6)	0.0	0.0	0.0	0.0	0.0	0.0	(6.0)	0.0	0.0	(10.0)	0.0	0.0	0.0	0.0	0.0
Net profit (loss) before income taxes	**(2.6)**	**(7.9)**	**(3.7)**	**(2.7)**	**(2.8)**	**(3.8)**	**(12.1)**	**(50.2)**	**(50.4)**	**(18.4)**	**47.3**	**196.9**	**368.5**	**531.9**	**662.2**	**709.4**	**758.5**	**794.7**
Income tax expense (benefit)	(0.0)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	19.7	36.9	186.2	231.8	248.3	265.5	278.2
Income tax (%)	*0.0%*	*0.0%*	*0.0%*	*0.0%*	*0.0%*	*0.0%*	*0.0%*	*0.0%*	*0.0%*	*0.0%*	*0.0%*	*10.0%*	*10.0%*	*35.0%*	*35.0%*	*35.0%*	*35.0%*	*35.0%*
Net Income (GAAP)	**(2.6)**	**(7.8)**	**(3.7)**	**(2.7)**	**(2.8)**	**(3.8)**	**(12.1)**	**(50.2)**	**(50.4)**	**(18.4)**	**47.3**	**177.2**	**331.7**	**345.7**	**430.4**	**461.1**	**493.0**	**516.6**
Adjusted Items (Non-GAAP)																		
Stock options	0.0	0.0	0.0	0.3	0.3	0.3	0.9	3.0	5.0	5.0	7.0	10.0	10.0	10.0	12.0	12.0	12.0	15.0
Other	0.0	14	0.6	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Net Income (Non-GAAP)	**(2.6)**	**(6.4)**	**(3.1)**	**(2.4)**	**(2.5)**	**(3.5)**	**(11.2)**	**(47.2)**	**(45.4)**	**(13.4)**	**54.3**	**187.2**	**341.7**	**355.7**	**442.4**	**473.1**	**505.0**	**531.6**
EPS, GAAP	**(0.49)**	**(1.18)**	**(0.56)**	**(0.06)**	**(0.06)**	**(0.08)**	**(0.76)**	**(1.13)**	**(1.11)**	**(0.40)**	**1.00**	**3.68**	**6.76**	**6.91**	**8.43**	**8.86**	**9.28**	**9.54**
Basic	5.3	5.5	5.6	43.4	43.5	43.6	34.0	44.4	45.3	46.2	47.2	48.1	49.1	50.0	51.0	52.1	53.1	54.2
Diluted	**5.3**	**5.5**	**5.6**	**43.4**	**43.5**	**43.6**	**34.0**	**44.4**	**45.3**	**46.2**	**47.2**	**48.1**	**49.1**	**50.0**	**51.0**	**52.1**	**53.1**	**54.2**
EPS, Non-GAAP	(0.49)	(0.22)	(0.11)	(0.06)	(0.06)	(0.08)	(0.30)	**(1.06)**	**(1.00)**	**(0.29)**	**1.15**	**3.89**	**6.96**	**7.11**	**8.67**	**9.09**	**9.51**	**9.81**
Basic	5.4	29.2	29.3	43.4	43.5	43.6	39.9	44.4	45.3	46.2	47.2	48.1	49.1	50.0	51.0	52.1	53.1	54.2
Diluted	**5.4**	**29.2**	**29.3**	**43.4**	**43.5**	**43.6**	**39.9**	**44.4**	**45.3**	**46.2**	**47.2**	**48.1**	**49.1**	**50.0**	**51.0**	**52.1**	**53.1**	**54.2**

Source: Jefferies estimates, company data

Biren Amin, Equity Analyst, (212) 284-8162, bamin@jefferies.com

Exhibit 29: KITE Balance Sheet

Kite Pharma, Inc.

Balance Sheet

(All values in $MM)

	2013A FY	2014E FY	2015E FY	2016E FY	2017E FY	2018E FY	2019E FY	2020E FY	2021E FY	2022E FY	2023E FY	2024E FY	2025E FY	2026E FY	2027E FY	2028E FY
Current assets:																
Cash and cash equivalents	22.4	193.6	141.0	87.7	65.9	109.2	282.0	608.7	949.0	1,374.0	1,829.7	2,317.3	2,828.4	3,353.3	3,885.5	4,425.0
Cash and investments	**22.4**	**193.6**	**141.0**	**87.7**	**65.9**	**109.2**	**282.0**	**608.7**	**949.0**	**1,374.0**	**1,829.7**	**2,317.3**	**2,828.4**	**3,353.3**	**3,885.5**	**4,425.0**
Other	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2
Total current assets	**22.6**	**193.9**	**141.3**	**87.9**	**66.1**	**109.5**	**282.2**	**609.0**	**949.3**	**1,374.2**	**1,829.9**	**2,317.5**	**2,828.6**	**3,353.5**	**3,885.7**	**4,425.2**
Other	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4
Total assets	**23.0**	**194.2**	**141.6**	**88.3**	**66.5**	**109.9**	**282.6**	**609.4**	**949.7**	**1,374.6**	**1,830.3**	**2,317.9**	**2,829.0**	**3,353.9**	**3,886.1**	**4,425.6**
Current liabilities:																
Accounts payable	0.4	2.4	2.4	2.4	2.4	2.4	2.4	2.4	2.4	2.4	2.4	2.4	2.4	2.4	2.4	2.4
Accrued expenses	0.9	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Other	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total current liabilities	**1.4**	**2.6**	**2.6**	**2.6**	**2.6**	**2.6**	**2.6**	**2.6**	**2.6**	**2.6**	**2.6**	**2.6**	**2.6**	**2.6**	**2.6**	**2.6**
Deferred rent, excluding current portion	0.0	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5
Total Liabilitiy	**1.4**	**5.1**	**5.1**	**5.1**	**5.1**	**5.1**	**5.1**	**5.1**	**5.1**	**5.1**	**5.1**	**5.1**	**5.1**	**5.1**	**5.1**	**5.1**
Total stockholders' equity	**21.6**	**189.1**	**136.5**	**83.2**	**61.4**	**104.7**	**277.5**	**604.2**	**944.5**	**1,369.5**	**1,825.2**	**2,312.7**	**2,823.9**	**3,348.8**	**3,881.0**	**4,420.5**
Total liabilities and stockholders' equity	**23.0**	**194.2**	**141.6**	**88.3**	**66.5**	**109.9**	**282.6**	**609.4**	**949.7**	**1374.6**	**1830.3**	**2317.9**	**2829.0**	**3353.9**	**3886.1**	**4425.6**

Source: Jefferies estimates, company data

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Exhibit 30: KITE Cash Flow Statement

Kite Pharma, Inc.

Cash Flow Statement

(All values in $MM)

	2013A FY	2014E FY	2015E FY	2016E FY	2017E FY	2018E FY	2019E FY	2020E FY	2021E FY	2022E FY	2023E FY	2024E FY	2025E FY	2026E FY	2027E FY	2028E FY
Cash flows from operating activities:	(6.4)	(12.1)	(50.2)	(50.4)	(18.4)	47.3	177.2	331.7	345.7	430.4	461.1	493.0	516.6	530.3	537.6	544.9
Net income																
Adjustments to reconcile cash by operating activities:																
Depreciation and amortization expense	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Compensation expense	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Other	(0.0)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Changes in operating assets and liabilities:																
Prepaid expenses	(0.0)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Due to related party	0.0	2.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other assets	(0.1)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Accounts payable	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Accrued expenses and deferred rent	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5
Net cash provided by operating activities	**(5.7)**	**(9.3)**	**(49.3)**	**(49.5)**	**(17.6)**	**48.1**	**178.1**	**332.5**	**346.6**	**431.3**	**461.9**	**493.9**	**517.4**	**531.2**	**538.4**	**545.8**
Cash flows from investing activities:																
Short-term investments	0.0	(2.5)	(3.0)	(3.5)	(4.0)	(4.5)	(5.0)	(5.5)	(6.0)	(6.0)	(6.0)	(6.0)	(6.0)	(6.0)	(6.0)	(6.0)
Change in fixed assets	(0.3)	(0.3)	(0.3)	(0.3)	(0.3)	(0.3)	(0.3)	(0.3)	(0.3)	(0.3)	(0.3)	(0.3)	(0.3)	(0.3)	(0.3)	(0.3)
Net cash (used in) provided by investing activities	**(0.3)**	**(2.8)**	**(3.3)**	**(3.8)**	**(4.3)**	**(4.8)**	**(5.3)**	**(5.8)**	**(6.3)**	**(6.3)**	**(6.3)**	**(6.3)**	**(6.3)**	**(6.3)**	**(6.3)**	**(6.3)**
Cash flows from financing activities:																
Cash received from loans	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Issuance of common stock, net of offering costs	0.0	133.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Issuance of preferred stock	19.6	50.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Net cash (used in) provided by financing activities	**19.6**	**183.4**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**
Effect if exchange rate changes on cash/equivalents																
Increase (decrease) in cash and cash equivalents	13.6	171.3	(52.6)	(53.3)	(21.8)	43.4	172.8	326.7	340.3	425.0	455.7	487.6	511.1	524.9	532.2	539.5
Cash and cash equivalents at beginning of period	8.7	22.3	193.6	141.0	87.7	65.9	109.2	282.0	608.7	949.0	1,374.0	1,829.7	2,317.3	2,828.4	3,353.3	3,885.5
Cash and cash equivalents at end of period	**22.3**	**193.6**	**141.0**	**87.7**	**65.9**	**109.2**	**282.0**	**608.7**	**949.0**	**1,374.0**	**1,829.7**	**2,317.3**	**2,828.4**	**3,353.3**	**3,885.5**	**4,425.0**

Source: Jefferies estimates, company data.

Biren Amin, Equity Analyst, (212) 284-8162, bamin@jefferies.com

Exhibit 31: KITE DCF Analysis

Kite Pharma, Inc.

Discounted Cash Flow Analysis

(All values in $MM)	*2012A*	2013A	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E
Sales	*0.0*	0.0	0.0	0.0	0.0	63.4	185.1	393.4	639.4	829.7	984.1	1,031.0	1,079.3	1,114.3	1,143.2	1,159.5	1,176.0
Operating Expenses	*7.7*	9.4	12.1	50.2	50.4	75.9	137.9	196.5	260.8	297.9	322.0	321.6	320.8	319.6	327.2	332.4	337.7
EBIT	(7.7)	(9.4)	(12.1)	(50.2)	(50.4)	(12.4)	47.3	196.9	378.5	531.9	662.2	709.4	758.5	794.7	815.9	827.1	838.4
(-): Taxes	0.0	0.0	0.0	0.0	0.0	0.0	0.0	19.7	36.9	186.2	231.8	248.3	265.5	278.2	285.6	289.5	293.4
EBIAT	(7.7)	(9.4)	(12.1)	(50.2)	(50.4)	(12.4)	47.3	177.2	341.7	345.7	430.4	461.1	493.0	516.6	530.3	537.6	544.9
(+):Depreciation	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
(+):FAS-123 Options	0.3	0.3	0.9	3.0	5.0	5.0	7.0	10.0	10.0	10.0	12.0	12.0	12.0	15.0	15.0	20.0	20.0
(-): Capital expenditures	0.0	0.0	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
(-): Changes in w orking capital	0.0	0.0	2.7	0.7	0.7	0.3	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7
Unlevered free cash flow	***(7.4)***	***(9.1)***	***(14.1)***	***(48.1)***	***(46.3)***	***(7.9)***	***53.3***	***186.2***	***350.7***	***354.7***	***441.4***	***472.1***	***504.0***	***530.6***	***544.4***	***556.6***	***564.0***

Source: Jefferies estimates, company data

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Exhibit 32: CD19 CAR-T U.S. Revenue Build

CD19 CAR-T U.S. Sales	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028
U.S.												
Total Net Sales of CD19 CAR-T in DLBCL ($M)	**$105.7**	**$308.6** 192%	**$467.4** 51%	**$577.7** 24%	**$687.4** 19%	**$721.2** 5%	**$756.1** 5%	**$792.1** 5%	**$829.2** 5%	**$858.9** 4%	**$867.5** 1%	**$876.2** 1%
Risk discount	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%
Total Net Sales of CD19 CAR-T in DLBCL - risk adj. ($M)	**$63.4**	**$185.1**	**$280.4**	**$346.6**	**$412.5**	**$432.7**	**$453.7**	**$475.2**	**$497.5**	**$515.4**	**$520.5**	**$525.7**
Total Net Sales of CD19 CAR-T in Hem Malignancies ex-DLBCL ($M)			**$188.2** #####	**$481.6** 156%	**$654.9** 36%	**$835.0** 28%	**$851.7** 2%	**$868.7** 2%	**$886.1** 2%	**$901.0** 2%	**$916.1** 2%	**$931.4** 2%
Risk discount			40%	40%	40%	40%	40%	40%	40%	40%	40%	40%
Total Net Sales of CD19 CAR-T in Hem Malignancies ex-DLBCL - risk adj. ($M)			**$112.9**	**$288.9**	**$392.9**	**$501.0**	**$511.0**	**$521.2**	**$531.7**	**$540.6**	**$549.6**	**$558.9**
Total Net Sales of CD19 CAR-T - risk adj. ($M)	**$105.7**	**$308.6**	**$655.6** 112%	**$1,059.2** 62%	**$1,342.4** 27%	**$1,556.2** 16%	**$1,607.8** 3%	**$1,660.8** 3%	**$1,715.3** 3%	**$1,759.9** 3%	**$1,783.6** 1%	**$1,807.6** 1%
Risk discount	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%
Total Net Sales of CD19 CAR-T - risk adj. ($M)	**$63.4**	**$185.1**	**$393.4**	**$635.5**	**$805.4**	**$933.7**	**$964.7**	**$996.5**	**$1,029.2**	**$1,055.9**	**$1,070.1**	**$1,084.6**

Source: Jefferies estimates, company data

Exhibit 33: CD19 CAR-T EU Revenue Build

CD19 CAR-T EU Sales	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028
EU												
Total Net Sales of CD19 CAR-T in DLBCL ($M)				**$31.8** #####	**$91.9** 189%	**$137.8** 50%	**$168.7** 22%	**$200.8** 19%	**$210.6** 5%	**$218.7** 4%	**$226.8** 4%	**$235.1** 4%
Risk discount				40%	40%	40%	40%	40%	40%	40%	40%	40%
Total Net Sales of CD19 CAR-T in DLBCL - risk adj. ($M)				**$19.1**	**$55.1**	**$82.7**	**$101.2**	**$120.5**	**$126.4**	**$131.2**	**$136.1**	**$141.1**
Total Net Sales of CD19 CAR-T in Hem Malignancies ex-DLBCL ($M)				**$0.0** #####	**$110.7** #####	**$282.2** 155%	**$383.7** 36%	**$489.3** 28%	**$499.1** 2%	**$508.2** 2%	**$517.6** 2%	**$527.1** 2%
Risk discount				40%	40%	40%	40%	40%	40%	40%	40%	40%
Total Net Sales of CD19 CAR-T in Hem Malignancies ex-DLBCL - risk adj. ($M)				**$0.0**	**$66.4**	**$169.3**	**$230.2**	**$293.6**	**$299.4**	**$304.9**	**$310.6**	**$316.3**
Total Net Sales of CD19 CAR-T - risk adj. ($M)				**$31.8** #####	**$202.5** 537%	**$420.0** 107%	**$552.5** 32%	**$690.1** 25%	**$709.7** 3%	**$726.9** 2%	**$744.4** 2%	**$762.3** 2%
Risk discount				40%	40%	40%	40%	40%	40%	40%	40%	40%
Total Net Sales of CD19 CAR-T - risk adj. ($M)				**$19.1**	**$121.5**	**$252.0**	**$331.5**	**$414.0**	**$425.8**	**$436.1**	**$446.7**	**$457.4**
Royalty rate				20%	20%	20%	20%	20%	20%	20%	20%	20%
Total Royalty of CD19 CAR-T - risk adj. ($M)				**$3.8**	**$24.3**	**$50.4**	**$66.3**	**$82.8**	**$85.2**	**$87.2**	**$89.3**	**$91.5**

Source: Jefferies estimates, company data

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Company Description

Kite Pharma, Inc. operates as a clinical stage biotechnology company which engages in the development of novel cancer immunotherapeutic products with focus on engineered autologous T cell therapeutics targeted to different tumor types. In addition, the company is advancing a novel therapeutic cancer vaccine aimed to trigger potent and specific immunity against multiple epithelial cancers, which has the potential to complement its eACT programs.

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I, Biren Amin, certify that all of the views expressed in this research report accurately reflect my personal views about the subject security(ies) and subject company(ies). I also certify that no part of my compensation was, is, or will be, directly or indirectly, related to the specific recommendations or views expressed in this research report.

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The expected total return (price appreciation plus yield) for Buy rated stocks with an average stock price consistently below $10 is 20% or more within a 12-month period as these companies are typically more volatile than the overall stock market. For Hold rated stocks with an average stock price consistently below $10, the expected total return (price appreciation plus yield) is plus or minus 20% within a 12-month period. For Underperform rated stocks with an average stock price consistently below $10, the expected total return (price appreciation plus yield) is minus 20% within a 12-month period.

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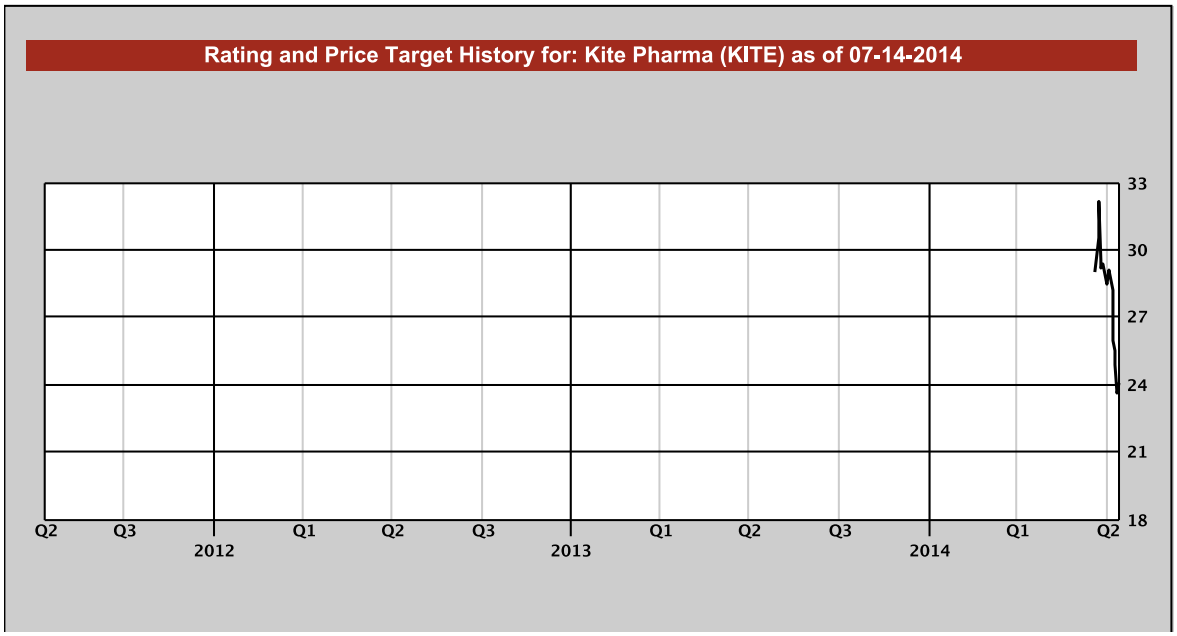
Other Companies Mentioned in This Report

- Amgen, Inc. (AMGN: $118.98, BUY)
- Celldex Therapeutics, Inc. (CLDX: $14.40, BUY)
- Dendreon Corporation (DNDN: $2.31, UNDERPERFORM)
- Dyax (DYAX: $8.98, BUY)
- Kite Pharma (KITE: $24.05, BUY)
- Newlink Genetics Corp. (NLNK: $23.06, BUY)
- Pfizer, Inc. (PFE: $30.24, BUY)
- XOMA Ltd. (XOMA: $4.15, BUY)



Distribution of Ratings

Rating	Count	Percent	IB Serv./Past 12 Mos. Count	IB Serv./Past 12 Mos. Percent
BUY	952	51.52%	246	25.84%
HOLD	748	40.48%	119	15.91%
UNDERPERFORM	148	8.01%	8	5.41%

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