USA | Healthcare | Biotechnology

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Immune Design (IMDZ) Initiate With A Buy: A Unique Approach To **Cancer Immunotherapy**

Key Takeaway

With the first in vivo dendritic cell activation technology, IMDZ offers a unique way to activate T cells to combat cancer. CMB305 has shown promising efficacy in pre-clinical studies and will undergo PI evaluation in Q1 2015. G100 in orphan Merkel cell carcinoma has observed a preliminary efficacy signal in a PI trial. IMDZ's widely partnered GLAAS platform for infectious disease vaccines provides upside.

Jefferies was joint book-running manager for IMDZ's IPO on July 29, 2014.

CMB305 Boosts T-Cells In Pre-clinical Tests And Will Initiate A PI Trial In Q1 2015: CMB305, a combination therapy of NY-ESO-1 dendritic cell vaccine (LV305) and G305 a NY-ESO-1 protein fused TLR-4 agonist will undergo evaluation in PI trial in solid tumors in Q1 2015. Assuming positive data in PI, IMDZ plans to proceed to PII in NSCLC and synovial sarcoma. LV305 primes dendritic cells and has shown to eradicate lung tumors in a preclinical model. LV305 + immune booster G305 are synergistic and have shown to boost the number of tumor eradicating cytotoxic T cells in preclinical models. G305 and LV305 are currently under PI evaluation in solid tumors and IMDZ plans to initiate the CMB305 Plb on assessing the data observed from these studies. Assuming positive data expected in Q1 2015 for LV305/G305 and successful dev't for CMB305, we estimate CMB305 approval in 2022. We anticipate peak U.S. revenues of \$360M for CMB305 (risk-adjusted).

Topline data from G100 PI trial in MCC in H1 2015: In a preliminary PI analysis, G100, the TLR-4 agonist has shown encouraging efficacy with 20% ORR observed in 5 merkel cell carcinoma (MCC) pts. IMDZ plans to combine radiation therapy to G100 in the second phase of PI study to observe better response. The P1 trial is enrolling 10 MCC pts with safety as EP and immunogenicity as secondary EP. Topline data from PI study is expected in H1 2015. Assuming positive data expected H1 2015 and in the subsequent pivotal PII/III trial, we estimate G100 approval in 2021. We anticipate peak U.S. revenues of \$30M (risk-adjusted).

Partnered GLAAS Platform Provides Upside: IMDZ has licensed its GLAAS platform molecules as an adjuvant for three vaccine vs infectious diseases to Medimmune (AZN, 4092p, Hold) and for a food allergy indication to Sanofi (SAN FP, €78.00, Buy). We currently don't model these milestones/royalties into our PT.

Valuation/Risks

Our \$15 PT is DCF-based. Risks to our thesis include clinical, regulatory and commercial risks.

USD	Prev.	2013A	Prev.	2014E	Prev.	2015E	Prev.	2016E
Rev. (MM)		1.6		0.8		2.0		2.0
EV/Rev		91.3x		NM		73.0x		73.0x
EPS								
Mar				(0.81)A				
Jun				(0.30)A				
Sep				(0.31)				
Dec				(0.29)				
FY Dec		(2.28)		(1.71)		(1.58)		(1.22)
FY P/E		NM		NM		NM		NM

Price target \$15.00 Price \$12.41

Financial Summary
Net Debt (MM):
Long-Term Debt (MM):
Cash & ST Invest. (MM):
Cash/Share:
Cash (MM):
Market Data
52 Week Range:
Total Entprs. Value (MM):
Market Cap. (MM):
Shares Out. (MM):
Float (MM):
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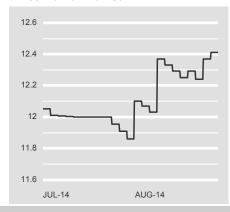
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Price Performance



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Buy: \$15 Price Target

Scenarios

Target Investment Thesis

- Expect success of G100 in MCC with US approval expected in2021. We estimate peak risk-adj sales of \$30M in 2032 in US.
- Expect success of CMB305 in NSCLC with US approval expected in 2022. We estimate peak risk-adj sales of \$314M in 2032 in US.
- Expect success of CMB305 in synovial sarcoma with US approval expected in 2022. We estimate peak risk-adj sales of \$46M in 2032 in US.
- DCF-based PT: \$15

Upside Scenario

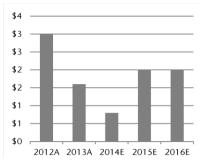
- Greater than expected adoption of CMB305 in NSCLC- \$19 PT (DCF-based)
- Expanded utility of CMB305 in other solid tumors - \$22 PT (DCF-based)

Downside Scenario

- Failure of G100 in MCC \$12PT (DCF-based)
- Failure of CMB305 in synovial sarcoma -\$12PT (DCF-based)
- Failure of CMB305 in NSCLC \$5 PT (DCF-based)
- Failure of all clinical programs \$4 PT (cash-valuation)

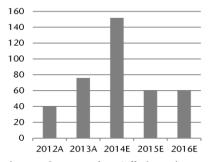
Long Term Analysis

Revenue (millions)



Source: Company data; Jefferies estimates

Enterprise Value (EV)/Sales



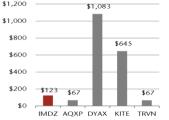
Source: Company data; Jefferies estimates

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.

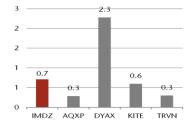
Peer Group

Group EV



Source: Factset, Jefferies estimates

Group EV/2024E Sales



Source: Factset, Jefferies estimates

Recommendation / Price Target

Ticker	Rec.	PT
IMDZ	Buy	\$15
AQXP	Buy	\$16
DYAX	Buy	\$13
KITE	Buy	\$35
TRVN	Buy	\$11

Catalysts

- Preliminary efficacy data from PI trial of G100 in MCC in H2 2014
- IND filing for CMB305 in H2 2014 and Initiation of Plb in solid tumors in Q1 2015
- Preliminary data from PI study of LV305 in solid tumors in H2 2014
- Topline data from the from PI study of LV305 in solid tumors in Q1 2015

Company Description

Immune Design is a biotechnology company that is Immunotherapy based developing treatments against cancers, infectious diseases and allergy. IMDZ's lead product is CMB305, combination therapy of NY-ESO-1 dendritic cell vaccine (LV305) and a NY-ESO-1 protein fused TLR-4 agonist (G305) and will undergo evaluation in Plb trial in solid tumors in Q1 2015. Regado's second product G100 (GLA-SE), a TLR-4 agonist for the treatment of merkel cell carcinoma is in Pl evaluation. IMDZ is also using the GLA-SE (G100) platform for developing TLR-4 adjuvants for the treatment of infectious diseases and allergy.

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Executive Summary

Immune Design is a Seattle-based biotech company that focuses on the development of immunotherapy for cancer, infectious disease and allergy. IMDZ's lead pipeline product is CMB305, a combination therapy of NY-ESO-1 dendritic cell vaccine (LV305) and a NY-ESO-1 protein fused TLR-4 agonist (G305) for the treatment of solid tumors. CMB305 will undergo evaluation in PIb trial in Q1 2015 in 4 solid tumors (sarcoma, NSCLC, melanoma and ovarian) and in phase II the company plans to further evaluate CMB305 in NSCLC and synovial sarcoma. Based on the promising pre-clinical data observed with LV305, w/ G305 alone and with combination of LV305 vector ZVex and G305 in eliciting humoral immune responses, we believe CMB305 has the strongest odds of success in NSCLC and synovial sarcoma and if successful could enter the US market in 2022. The topline data from the PIb study of CMB305 is expected by 2H '15 and PII initiation in expected in 2016. Preceding the CMB305 Plb trial, IMDZ is conducting Pl trials of LV305 and G305 separately in 5 solid tumors (sarcoma, NSCLC, melanoma, ovarian and breast cancer) to further understand the clinical safety profile of each therapeutic and which would further identify a more precise dose range for studies in the combination CMB305 program. Topline data from both LV305 and G305 PI studies is expected in H1 2015. IMDZ is also developing G100, a TLR-4 agonist synthetic molecule Glucopyranosyl Lipid A (GLA-SE) for the treatment of Merkel cell carcinoma (MCC). G100 is in PI trials with encouraging data observed in preliminary efficacy data. We estimate G100 could be approved in US market in 2021. IMDZ's other pipeline products include the development and licensing of GLA-SE platform (TLR-4 agonist) as adjuvant for the treatment of infectious diseases and allergy. IMDZ has licensed its GLA-SE molecule as an adjuvant to MedImmune (AZN LN, 4092.50, Hold) for treatment of three infectious disease and Sanofi (SAN FP, €78.00, Buy) for the treatment of undisclosed food allergy.

CMB305 Primes and Boosts In Vivo Cytotoxic T-cells. We believe CMB305 in vivo approach of dendritic cell activation could observe efficacy signals in its clinical trials in solid tumors due to the favourable pre-clinical data observed to date with CMB305 backbone of LV305 and G305. In mouse models of lung metastases, a single dose of LV305 reduced metastatic lung tumors in a dose dependent manner with the highest dose nearly eradicating the tumor. Since LV305 doesn't use autologous approach of administration, its bio distribution in mouse models was observed in draining lymph nodes, and studies suggest that localization of activated dendritic cells within the draining lymph node is critical for the prevention the tumor cells escape and metastasis. Several clinical trials with autologous dendritic cell vaccines have failed or have observed mediocre efficacy due to localization of dendritic cells outside the draining lymph nodes. Further, it was shown that ZVex (LV305 backbone vector) and GLA-SE (G305 backbone vector) when given individually were able to induce only 3.16% and 0.05% of CD8 T cells and 0.05% and 0.75% of CD4 T cells respectively. However, the combination of ZVex+GLAAS was able to show synergistic effect and was able to induce 15.7% and 2.82% of CD8 and CD4 T cells, respectively. Studies suggest that cytotoxic CD8+ T cell responses generated by lentivectors are CD4+ T cell-dependent which is activated by TLR-4 adjuvants (Xiao L et al Vaccine 2011) and is important in immune response against tumors. In addition, in a clinical study using prime and boost approach of immunity with recombinant vaccinia virus expressing NY-ESO-1 antigen resulted in an ORR of

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14% and CBR of 72% was observed in 25 stage III/IV melanoma patients. LV305 and G305 are currently undergoing PI trials in solid tumors currently and we believe that both these trials should see encouraging safety and efficacy. Based on these results, IMDZ plans to initiate the PIb trial in 4 solid tumors (sarcoma, NSCLC, melanoma and ovarian) in Q1 2015 with preliminary data expected by YE 2015.

encouraging efficacy observed with G100 in loco-regional merkel cell carcinoma in the PI could suggest the program has a path forward in this orphan indication. In preliminary phase I data released to date, treatment with G100 observed a response rate of 20% in 5 MCC patients (2 localised and 3 metastatic patients). Though no response rates were observed in the metastatic patients, we believe the next phase of PI study where IMDZ will evaluate the combination of G100 + radiation therapy to treat the MCC patients may offer better prospects in metastatic MCC patients. These studies are supported by preclinical data where G100 was able to activate dendritic cells leading to production of cytokines responsible for boosting innate immunity against cancer. The topline data from the phase I study is expected in H1 2015.

Valuation

We arrive at our \$15 PT based on a DCF valuation model which assumes a WACC of 13%, patent expiry for CMB305 in 2032, and outstanding shares of 17.4 million. We estimate risk-adjusted peak U.S. CMB305 sales of \$360 million and risk-adjusted US G100 sales of \$30 million. We estimate US approval and launch of G100 and CMB305 in 2021 and 2022, respectively. We have not included EU sales of CMB305 or G100 or licensing royalties received from the infectious disease and allergy programs into our model and presents upside.

Exhibit 1: DCF sensitivity analysis

Disc Rate	Price/Share
9%	\$24.20
11%	\$19.00
13%	\$14.99
15%	\$11.90
0%	\$9.49

Source: Jefferies estimates

Risks

Clinical Failure: As with all companies in biotechnology that are investing in the development of preclinical/clinical programs, trial failures can lead to delays in projections for market entry or possibly discontinuation of programs.

Regulatory Failure: The FDA could determine the new drug application is inadequate for CMB305 and G100 and could delay approval. Any delays in approval timelines could impact our earnings estimates, price target, and/or rating.

Commercial Failure: We currently project U.S. sales of \$360 million (risk-adjusted) for CMB305 and \$30 million (risk-adjusted) for G100. Our estimates may rely on the success of the company/partners to receive drug reimbursement from private/public payors.

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Financing Risks: We estimate IMDZ may need additional financing(s) in 2016-2017 to develop CMB305 and G100 and the pipeline and fund a potential U.S. launch of CMB305 and G100. The company may offset the need to raise additional capital by potentially licensing ex-U.S. rights to CMB305 and G100 and licensing royalties from infectious disease and allergy program. We currently have not modelled any potential upfront payments from a licensing collaboration.

Company Overview

Immune Design is focused on the development of immunotherapeutics targeting against cancers, infectious diseases and allergy. IMDZ's lead product is CMB305, combination therapy of NY-ESO-1 dendritic cell vaccine (LV305) and a NY-ESO-1 protein fused TLR-4 agonist (G305) and will undergo evaluation in Plb trial in solid tumors in Q1 2015. IMDZ's second product G100 (GLA-SE), a TLR-4 agonist for the treatment of merkel cell carcinoma is in PI evaluation. IMDZ is also using the GLA-SE (G100) platform for developing TLR-4 adjuvants for the treatment of infectious diseases and allergy.

Drug	Mechanism of Action	Indication	Development Phase
LV305	NY-ESO-1 dendritic cell vaccine	NSCLC, sarcoma, melanoma, ovarian and breast cancer	Phase I
G305	NY-ESO-1 vaccine and TLR-4 agonist	NSCLC, sarcoma, melanoma, ovarian and breast cancer	Phase I
CMB305	NY-ESO-1 vaccine and TLR-4 agonist	NSCLC and synovial sarcoma	Preclinical
G100	TLR-4 agonist	Merkel cell carcinoma	Phase I
GLA-SE (MedImmune partnered	TLR-4 agonist	3 infectious diseases	Preclinical
GLA-SE (Sanofi partnered)	TLR-4 agonist	Food allergy vaccine	Preclinical
G103	TLR-4 agonist	HSV-2	Preclinical

Source: Company reports

Product	Indication	Event	Date
G100	Merkel cell carcinoma	Preliminary efficacy data from PI trial	H2 2014
		Topline results for PI trial	H1 2015
		Topline PIII data	2020
		US regulatory filing and approval	2021
LV305	Solid tumors	Preliminary data from the PI trial in sarcoma, NSCLC, melanoma, ovarian and breast cancer	H2 2014
		Topline data from the PI trial	Q1 2015
G305	Solid tumors	Topline data from the PI trial in sarcoma, NSCLC, melanoma, ovarian, and breast cancer	Q1 2015
CMB305	Solid tumors	IND filing	H2 2014
		Initiation of PIb in sarcoma, NSCLC, melanoma and ovarian cancer	Q1 2015
		Preliminary efficacy data from Plb in solid tumors	2H 2015
		Topline data from the Plb in solid tumors	H1 2016
	NSCLC and synovial	Initiation of PII trial	YE 2015/ early 2016
	sarcoma		
		Topline PIII data in 3rd/4th line NSCLC and synovial sarcoma	2021
		US regulatory filing and approval	2022

Source: Company estimates; Jefferies.

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G100: Endogenous Antigen Approach To Boost The Innate Immune Sytem

G100 is a Glucopyranosyl Lipid A stable emulsion (GLA-SE) synthetic molecule that is a TLR4 agonist involved in the activation of dendritic cells. TLR-4 agonists have been shown to activate the dendritic cells leading to maturation by production of inflammatory cytokines, type I interferon and chemokines. Mature dendritic cells then boosts the activation of the CD4 T against endogenous tumor antigens. G100 is currently in Phase I trial in MCC. G100 has shown encouraging results in the preliminary phase I results in MCC. Topline data from the Phase I trial is expected in 1Q 2015. Assuming a positive Phase III trial, we estimate G100 could launch in 2021 for MCC with a market opportunity of \$30M in the U.S. by 2032 (on a risk-adjusted basis). Additional upside for G100 may be derived as a potential treatment for Non-Hodgkin's Lymphoma.

Market Opportunity

Market Opportunity for G100. Though IMDZ plans to evaluate G100 in Non-Hodgkins Lymphoma in the near-term, we have only accounted for MCC in our model. According to the Surveillance Epidemiology and End Results (SEER) database, there are an estimated 1,500 new cases of MCC in the U.S. in 2014 (incidence). We use a prevalence-based model to arrive at our G100 sales estimates. Based on our estimate we expect prevalence of 2,500 cases of MCC in 2015 and a 2% annual growth rate. We believe that G100 will have adoption in the loco-regional and metastatic MCC. Based on cancer.gov, 31% and 9% of the MCC patients represent loco-regional and metastatic MCC patients. We believe G100 could be approved in 2021 and will achieve a peak market penetration of 45% in 2028. We estimate peak market sales of \$120M for G100 in 2032. As G100 is in Phase I we have taken a 75% risk discount and our risk-adjusted peak sales are \$30M.

Encouraging Efficacy Observed G100 Phase I study

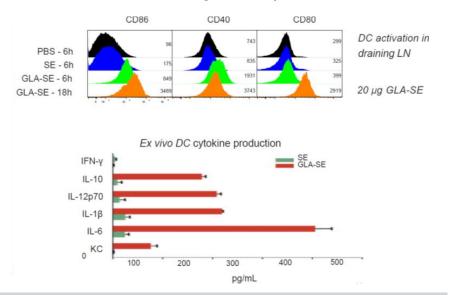
Complete Response Observed in Phase I study: In the preliminary results of 5 patients involving 2 loco-regional and 3 metastatic MCC patients, 1 complete response was observed in a loco-regional MCC patient. However, no response was observed in the other MCC patients treated to date. To improve the efficacy further, IMDZ is planning to combine G100 administration with local radiation. We believe this approach could offer potentially better efficacy vs metastatic MCC disease.

These encouraging results are corroborated by preclinical work where administration of G100 has shown production of cytokines responsible for activation and maturation of dendritic cells from the draining lymph nodes ex vivo. G100 was able to induce IL-6, IL-10, IL-12p70 and IL-1β, which lead to activation and complete maturation of dendritic cells in 18 hrs.

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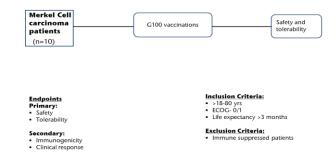
Exhibit 4: Preclinical data showcasing the efficacy of G100 in DC activation



Source: Source: company presentation; Jefferies

G100 Phase I Design. G100 is in an open-label phase I trial that will enroll 10 patients of MCC and measure the safety and tolerability of G100 as a primary endpoint. Secondary endpoint of the study includes evaluating immunogenicity and clinical responses of G100. Final top line data from the study is expected in H1 2015. The trial will enroll patients who are between 18-80 yrs, ECOG 0/1 and have life expectancy greater than 3 months. Exclusion criteria for the trial include patients who are immune suppressed or had prior pre-chemotherapy or major surgical procedure within 3 weeks or radiotherapy within last 2 weeks prior to study.

Exhibit 5: G100 PI study design



Source: www.clinicaltrials.gov

Merkel Cell Carcinoma Background. Merkel cell carcinoma (MCC), sometimes referred to as a neuroendocrine carcinoma of the skin, arises from the uncontrolled growth of Merkel cells in the skin. It is a rare skin cancer with incidence of 1500 cases per year in the US, and 80% of MCC is cause by merkel cell polyomavirus. MCC has the potential to be lethal, and thus prompt aggressive treatment is warranted. Diagnosis of MCC is made with a skin biopsy, where special stains are used to distinguish MCC from other forms of cancer, such as small cell lung cancer (SCLC), lymphoma, and small cell melanoma. MCC will stain for low molecular weight cytokeratins (CAM 5.2 or AE1/AE3),

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CK 20 and neuron-specific enolase (NSE) but will not stain for CK7 or thyroid transcription factor 1 (TTF-1) leukocyte common antigen (LCA) and S100.

MCC patients with loc-regional disease (stage I, II and III) have a better prognosis than metastatic patients (stage IV). Median survival of localized (stage I and II), regional and metastatic MCC is > 10 years, > 6 years and < 2 years, respectively. Treatment for MCC is generally based on the stage of the disease and includes the following options: 1) surgical excision of the primary lesion; 2) lymph node surgery; 3) radiation therapy; and 4) chemotherapy. For most cases of localized MCC excision of the primary lesion with a greater than or equal to 2 cm margin may be a treatment option. It is important for almost all cases in which there are no obvious lymph node disease to also undergo sentinel lymph node biopsy at the time of wide surgical excision to determine whether or not microscopic disease is present. In loco-regional disease, radiation of the affected nodes, or surgical removal of the remaining lymph nodes in a draining lymph node basin may be indicated and results in a high cure rate for the affected nodal region. Metastatic disease is treated with radiotherapy and/or chemotherapy. Stage IV disease is palliative and is given to relieve symptoms, such as pain, and to help patients live more comfortably.

Several targeted therapies have been tested in last few years with very little success in advanced MCC. Imatinib (TKI inhibitor) was tested in unresectable or metastatic MCC and an ORR of 4% was observed with a median PFS of 1-2 months. Separately, a phase II trial evaluating oblimersen, an antisense vs Bcl-2, was tested in 12 patients w/ recurrent or metastatic MCC, and reported no objective responses and stable disease in three patients. Bcl-2 expression was later found to not play a role w/ Bcl-2 negative patients exhibiting worse prognosis in a separate analysis of 166 MCC tumor specimens. The only targeted therapy that exhibited any efficacy in MCC was GSK's (GSK LN, 1392.00p, Hold) Votrient (pazopanib) which observed a complete response after two months of treatment in a single MCC patient. Votrient is currently being tested in a broader population of neuroendocrine tumors which include MCC and is targeting to enrol 165 patients w/ data expected in YE '16.

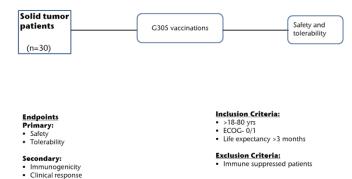
G305: Specific Antigen Approach To Boost CD4 T cells

G305 is a modified version of G100, where instead of boosting immunity through endogenous antigen, a full length antigen NY-ESO-1 along with G100 is used to stimulate dendritic cells/CD4 T cells leading to the targeting of NY-ESO-1 specific tumors. G305 is in a 30-patient open-label phase I trial that is evaluating G305 in unresectable metastatic NSCLC, ovarian, melanoma, sarcoma and breast cancer. The trial will involve a T3+3 design and G305 will be administered intradermally to patients. The primary endpoint of the study is to evaluate safety and tolerability of G305 whereas secondary endpoints include immunogenicity and clinical response. Topline data from this trial is expected in H1 2015. The trial will enroll patients who are between 18-80 years of age, exhibit expression of NY-ESO-1, ECOG 0/1 and have life expectancy greater than 6 months. Exclusion criteria for the trial include patients who had prior NY-ESO-1 targeting immunotherapies, have brain metastases and administration of investigational therapy 3 weeks prior to enrollment.

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Exhibit 6: G305 PI study design



Source: www.clinicaltrials.gov

TLR4 in Dendritic Cell Activation and Maturation. Activation of innate immune system through toll like receptors has been a well-established pathway for combating infectious disease and more recently research efforts have been made to study this pathway for combating cancer. In humans, 11 different TLRs have been identified. Different subsets of dendritic cells (DCs) could express different TLRs. For example, mouse spleen CD8+ DCs are shown to express TLR-2, 3, 4, and 9 and have a better antigen presentation capacity as compared to CD8- DCs that express TLR-2, 3, 4, 5, 7, and 9. TLR4 has been shown to activate dendritic cells leading to Th1 responses which activate the CD4 T cells. Preclinical evidence has shown that a TLR4 agonist specifically promoted the production of the Th1-inducing cytokine interleukin (IL) 12 p70 and the chemokine interferon-gamma inducible protein (IP)-10, which is also associated to Th1 responses (Re F et al JBC 2001, Behzad H Clin Cancer Res 2012). TLR4 is the only TLR receptor capable of inducing two distinct signalling pathways: 1) myD88 activates NF-Kappa signalling and induces production of cytokines; and 2) the TRIF pathway which induces the production of Type I interferons.

CMB305: Priming and Boosting Of In Vivo Cytotoxic T-cells

CMB305 is a combination of LV305 (NY-ESO-1 dendritic cell vector) and G305 (TLR4 agonist + NY-ESO-1 protein) that will undergo Plb trials in solid tumors in Q4'14. NY-ESO-1 is a cancer-associated antigen that has been found to be over expressed in 20-80% of solid cancers that include melanoma, GBM and synovial cancers and CMB305 aims to target this protein by enhancing in vivo cytotoxic T-cells through dendritic cells. The Phase lb trial of CMB305 will be preceded by separate phase I trials of LV305 and G305 in NSCLC, breast cancer, ovarian cancer, melanoma and sarcoma. Based on the results of these studies, IMDZ will file an IND in H2 2014 and will initiate CMB305 phase lb/II trial in 4 solid tumors which include NSCLC, melanoma, sarcoma and ovarian cancer in H1 2015. IMDZ currently plans to conduct the phase II trial of CMB305 in NSCLC and synovial sarcoma patients. Assuming a positive Phase III trial, we believe CMB305 can launch in 2022 in both synovial sarcoma and NSCLC with market opportunity of \$360M in the U.S. by 2032 (on a risk-adjusted basis).

Market Opportunity

Market Opportunity for CMB305. We currently have only modelled CMB305 for two cancer indications: NSCLC and synovial sarcoma. According to the Sarcoma Foundation

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of America, there are an estimated 900 new cases of synovial sarcoma in the U.S. in 2013 (incidence). We use a prevalence-based model to arrive at our G100 sales estimates. Based on our estimate we expect prevalence of 1,700 cases of synovial sarcoma in 2015 and a 1% annual growth rate. We believe CMB305 will be approved in 2022 and will achieve a peak market penetration of 50% in 2027. We estimate a peak market of \$184M for CMB305 in synovial sarcoma in 2032.

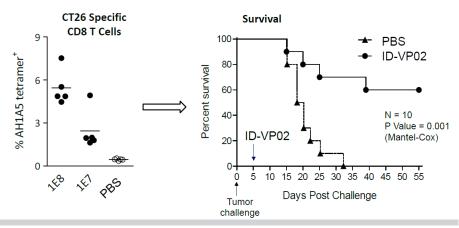
According to the American Lung Association, there are estimated ~224K new cases of lung cancer patients in the U.S. in 2014 (incidence) of which 85% are NSCLC patients. Our prevalence based estimate of NSCLC patients is ~379K cases with a 3% annual growth rate. CMB305 will be tested in 3rd/4th line NSCLC patients, which represents 20% of NSCLC patients or 57K patients. We believe CMB305 will be approved in 2022 and will achieve a peak market penetration of 7% in 2031. We estimate a peak market of \$1.2B for CMB305 in 2032. Due to early nature of CMB305 clinical trials, we have taken a 75% risk discount and our risk-adjusted peak sales are \$46M for synovial sarcoma and \$295M for NSCLC in 2032.

LV305 Exhibits Superior Efficacy in Preclinical Models

LV305 Doesn't Require Autologous Transduction of Dendritic Cells: One of the biggest issues facing dendritic cell vaccines is the requirement of generating autologous antigen primed DCs ex vivo and then transplanting it back to the patient. Ex vivo DCs have low migration capacity from blood to secondary lymphatic system leading to inefficient generation of CTLs. To overcome this hurdle, LV305 is derived from lentiviral vector called ZVex that expresses DC-SIGN, a protein that is expressed only on the surface of dendritic cells and macrophages. ZVex also contains VPX gene that binds to SAMHD1 gene on DCs and helps in its replication. It has been shown that dendritic cell transduction with ZVex is DC-SIGN and VPX dependant and blocking DC-SIGN or VPX abrogates the transduction of dendritic cells by ZVex.

ZVex expressing AH1A5 tumor antigen was able to activate the CTLs (CD8 T cells) which was dose dependant and was able to provide reduction in death by 60% in a mouse colon carcinoma model.

Exhibit 7: ZVex expressing AH1A5 tumor antigen induces CTLs



Source: Company reports; Jefferies

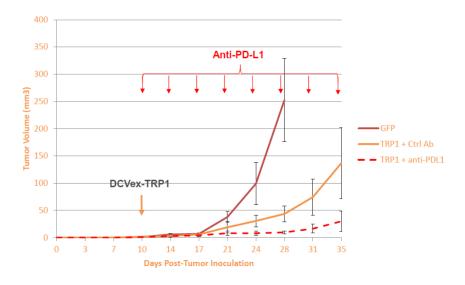
These results can be further confirmed by using other tumor antigens. ZVex expressing self TRP1 (melanocyte antigen) was able to significantly reduce tumor growth of B16 murine tumor model. In addition it has been shown that DVex-TRP1 exhibited synergistic

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effects when combined with immune check point inhibitors (PD-1 inhibitors) leading to significant eradication of tumor in mouse melanoma model. IMDZ plans to evaluate in combination w/ PD-1 inhibitors once CMB305 data are available, and more recently several companies have started to evaluate cancer vaccines w/ immune checkpoint inhibitors.

Exhibit 8: ZVex expressing TRP1 has synergistic effect with PDL-1 in eradicating tumor



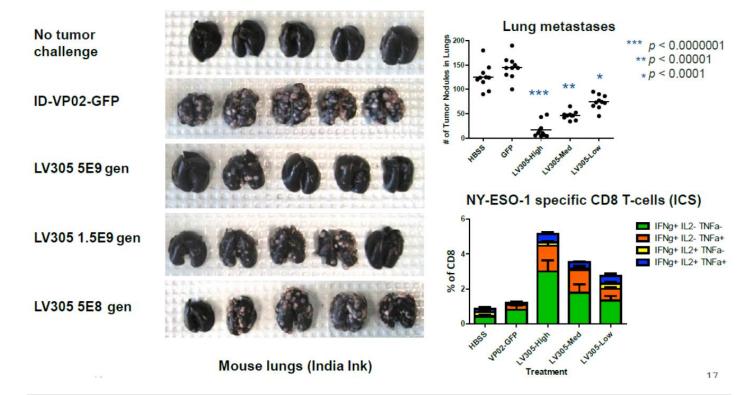
Source: Company reports; Jefferies

LV305 Reduces Lung Metastases in Preclinical models: LV305 is a dendritic cell antigen delivery system that targets the NY-ESO-1 antigen. NY-ESO-1 is tumor specific antigen and is not expressed in normal cells. LV305 is lentiviral vector based antigen delivery system of NY-ESO-1 gene into dendritic cells. The activated dendritic cells lead to increased production of cytotoxic T cells and thereby causing the eradication of tumor cells. In preclinical mouse models of lung metastases, a single dose of LV305 on day 3 post induction of lung metastases was able to reduce metastatic lung tumors in a dose dependant manner. Near eradication of lung tumor nodules was observed with higher doses of LV305. A similar effect was observed with induction of CD8 CTLs by LV305 in a dose dependent manner where higher doses resulted in induction of CD8 T CTLs.

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Exhibit 9: Preclinical Activity of LV305 in Lung Tumors



Source: Company presentation, Jefferies

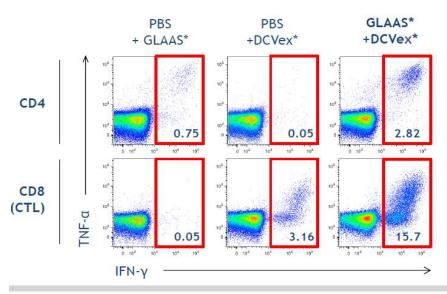
No off target transduction of LV305 was found in mice as biodistribution was limited to the injection site and the draining lymph nodes where dendritic cells are located. Ovaries, bone marrow, heart, lung, kidney, intestine and non-draining lymph nodes all tested negative for LV305. However, low levels of off target transduction were observed after 4th vaccination of LV305. These data suggests potential limited off-target toxicity of LV305, however, human clinical data need to further confirm these preclinical observations.

CMB305 Primes and Boosts the Humoral Responses and Innate Immunity: CMB305 is a combination of LV305 and G305, where LV305 acts as a primer for the activation of humoral response (CTLs) through dendritic cells whereas G305 acts as booster for enhancing innate immunity. This combination was devised based on the preclinical data where synergistic effects were observed when ZVex was combined with GLAAS (GLA agonists). In preclinical models it was shown that ZVex and GLA-SE when given individually were able to induce only 3.16% and 0.05% of CD8 T cells and 0.05% and 0.75% of CD4 T cells, respectively. However, the combination of ZVex+GLAAS was able to show a synergistic effect and was able to induce 15.7% of CD8 T cells and 2.82% CD4 T cells. These results are supported by a study where it was observed that cytotoxic CD8+ T cell responses generated by lentivectors is CD4+ T cell-dependent which is activated by TLR-4 adjuvants and TLR-4 based activation of dendritic cells is dependent on MYD88 and TRIF pathways (Xiao L et al Vaccine 2011). Therefore we believe that CMB305 should have significant effect in reducing the tumor burden in clinical models and enhancing the survival of patients. Due to the in vivo stimulation and generation of CTLs, we believe that safety profile of CMB305 should exhibit benign safety profile.

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Exhibit 10: CMB305 Exhibits Induces Greater Production of CD8 and CD4 T-Cells



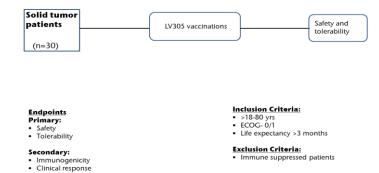
Source: Company reports; Jefferies

LV305 Phase I Design. LV305 is in an open-label, Phase I trial that began in April 2014 and will be comprised of two parts: Phase I (dose-escalation) and Phase Ib (expansion). The trial will involve a T3+3 design (3 cohorts) and will enrol 36 patients from 5 solid tumors that include NSCLC, breast, ovarian, melanoma and sarcoma. In the dose expansion portion, IMDZ expects only to enrol patients from melanoma and breast cancer. The primary endpoint of the study is to evaluate the safety and tolerability of LV305 w/ the secondary endpoints following clinical response. Preliminary data from the cohorts 1 and 2 study is expected in H2 2014 with final topline data expected in H1 2015. The trial will enrol patients who are between 18-80 years, exhibit expression of NY-ESO-1, ECOG 0/1 and have life expectancy greater than 6 months. Exclusion criteria for the trial include patients who had prior NY-ESO-1 targeting immunotherapies, have brain metastases and administration of investigational therapy 3 weeks prior to enrollment.

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Exhibit 11: LV305 PI study design



Source: www.clinicaltrials.gov

Other NY-ESO-1 Vaccines- Prime and Boost Approach With NY-ESO-1 Shows Efficacy: Since NY-ESO-1 is not expressed in normal cells and only expressed by tumor cells, many clinical trials are under way with NY-ESO-1 to target solid tumors with mixed results. In a clinical study using recombinant vaccinia virus expressing NY-ESO-1 antigen, an ORR of 14% and CBR of 72% was observed in 25 stage III/IV melanoma patients. The median PFS of the patients was 9 months and OS was 48 months. At 31 months follow up, 52% of the patients were still alive. Using the same vaccine, a PFS of 21 months and OS 48 months was observed in 22 ovarian cancer patients and the trial could not measure response rate because no measurable disease activity was observed in these patients at the time of study entry. High titers of CD4 and CD8 T cells were observed post-vaccination in both melanoma and ovarian cancer patients (Odunsi K et al PNAS 2012).

Exhibit 12: PI results using NY-ESO-1 vaccinia virus

Cancer type	Patient size	ORR	CBR	PFS (mos)	OS (mos)
Stage III/IV melanoma	25	14%	72%	9	48
Stage III/IV Ovarian	22	NA	NA		48

Source: Odunsi K et al PNAS 2012; Jefferies

In a PI clinical trial involving 12 prostate patients and NY-ESO-1 vaccine and CpG adjuvant, humoral responses of high CD4/CD8 T cells was observed in 69% of the patients. Similarly, in a PI trial involving NY-ESO-1 vaccine (CHP-NY-ESO-1) improved survival of patients was observed in a dose dependent manner. The trial observed a clinical benefit rate (CBR) of 23% in the low dose 100 ug arm vs a 50% CBR in the 200 ug arm. However, studies show that the efficacy of NY-ESO-1 response depends completely on the activation of humoral response. In a PI clinical trial using mixed bacteria vaccine of NY-ESO-1 in 12 solid tumors, response rate was observed in only 1 bladder cancer patient (8%). Upon analysis of the humoral response it was observed that no difference in CD4/CD8 T cells was observed between pre and post vaccination in this study (Karbach J et al Clin Cancer Res 2012).

Deficiencies of Current Dendritic Cell Vaccines: The efficacy of dendritic cell vaccines in clinical trials has been mixed. Current dendritic cell based vaccines in clinical

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trials and Provenge (DNDN, \$1.40, Underperform) utilize autologous administration, where monocytes from patients are taken and developed into dendritic cells, and the DCs are primed with a tumor antigen and then re-administered into the patient. The problem with the autologous approach is that these dendritic cells have to relocate to the draining lymph nodes and activate the humoral responses and due to ex vivo culture of these cells, several cell features might have changed that don't direct these cells to draining lymph nodes within the lymphatic system. Studies suggest that localization of activated dendritic cells outside or inside of draining lymph nodes is critical for the prevention the tumor cells escape and metastasis (Ochsenbein AF et al Nature 2001; Zinkernagel RM Nature 2014). Autologous approach also uses storage of dendritic cell vaccines in freezer which can reduce the efficacy of DC vaccines. However with CMB305/LV305, all these problems are mitigated and we may observe better clinical efficacy than these approaches. To overcome this hurdle, LV305 is derived from lentiviral vector called ZVex that expresses DC-Sign, a protein that is expressed only on the surface of dendritic cells and macrophages. ZVex also contains VPX gene that binds to SAMHD1 gene on DCs and helps in its replication. In addition, LV305/CMB305 does not require activation of dendritic cells ex vivo and the viral particles can be injected directly into the body where these LV305/CMB305 viral particles will be directly taken up by the DCs cell in vivo in the draining lymph nodes and thereby activate the immune system against the tumor.

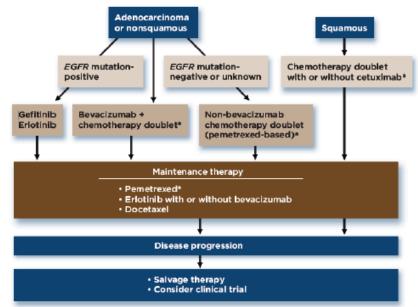
NSCLC Background. Lung cancer remains the leading cause of cancer-related deaths with 221,130 new cases and 156,940 deaths estimated by the National Cancer Institute in 2011. The majority of the patients with lung cancer have been found to harbor multiple mutations in the crucial development and signaling pathways. In patients diagnosed with early-stage NSCLC, surgery may be curative. However, surgery and radiation are not options in patients with advanced NSCLC (stage IIIB/IV). Advances in chemotherapy with doublet regimens have led to increased median survival times of 11 months. Platinumbased doublet therapies are the standard of care in NSCLC with negative EGFR status. Chemotherapies that may be combined with platinum agents include Gemzar, Navelbine, Taxotere, Taxol, and Alimta.

Targeted therapies such as Avastin, Tarceva, and Iressa are approved in advanced NSCLC. Avastin may be used in combination with platinum doublet therapy in patients with epidermal growth factor receptor (EGFR)-mutation negative/unknown status whereas Tarceva and Iressa may be prescribed in patients with EGFR-mutation positive status. Avastin's ECOG-4599 trial evaluated the potential of a combination with platinumdoublet therapy, and observed a significantly longer overall survival (12.3 months versus 10.3 months; p = 0.003, HR = 0.79). Response rates and PFS were also significantly better with the Avastin-based regimen. EGFR mutations are an important target and are associated with sensitivity to Iressa/Tarceva. Iressa, first approved in 2004, through an accelerated approval pathway, improved response rates by 19% in 105 patients with NSCLC. However, a subsequent trial failed to observe a benefit in overall survival, and thereafter, Iressa's label is restricted to patients who have previously benefited from it. Tarceva, approved in 2004, is indicated in patients who have progressed on at least 1 chemotherapy, and more recently was approved for maintenance therapy in patients with locally advanced/metastatic NSCLC whose disease had progressed after 4 cycles of platinum-based therapy. Data from a 731- patient Phase III trial observed a significantly longer overall survival by 2 months compared to placebo in patients who progressed on at least 1 chemotherapy. The SATURN trial in 884 patients reported an improvement in PFS with Tarceva compared to placebo (12.3 weeks versus 11.1 weeks; HR = 0.71, p < 0.0001). The EGFR family serves as mediators of cell signaling by extracellular growth factors and binding of ligands at the receptor sites may promote tumor growth. Approximately 10% of all NSCLC have EGFR positive mutations. About 50-60% of patients progress to second line and another 50% progress to third line.

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Exhibit 13: Treatment algorithm - NSCLC



Source: Clinical Oncology: Jefferies

Synovial Sarcoma Background. Synovial sarcoma is a type of soft-tissue sarcoma that usually occurs near to the joints of the arm, neck or leg. It is a rare cancer with only 1-3 cases in a million people diagnosed each year. Synovial sarcoma seems to have a slight preference for males, with 12 male patients for every 10 female patients. Nearly all cases of synovial sarcoma carry the SS18-SSX fusion gene. The diagnosis of synovial sarcoma can be suspected via X-ray or imaging, made via biopsy, and confirmed via cytogenetic studies that show a translocation (an exchange of material) between the X chromosome and chromosome 18 in the tumor cells. The key treatment is surgery to remove the entire tumor, nearby muscle, and lymph nodes. Radiation, chemotherapy, or a combination of treatment methods may follow surgery.

There are currently very few targeted therapies that have been successful in clinical trials in synovial sarcoma. The only targeted therapy approved in synovial sarcoma is Votrient from Glaxo Smith Kline which targets broad range of tyrosine kinase inhibitors. In a randomised phase II study involving 390 soft tissue sarcoma patients, 10% of the patients had synovial sarcoma. The median PFS in metastatic synovial sarcoma patients treated with Votrient was 3.8 month v. 1.6 months for placebo (HR = 0.43; 95% Cl: 0.19-0.98). The median OS was 12.6 months for Votrient arm v. 10.7 months for placebo (HR = 0.87; 95 % Cl: 0.67-1.12). Synovial sarcomas have also been shown to express EGFR as well as HER2/neu. A phase II trial of gefitinib was also disappointing, with only 13% of patients having prolonged disease stabilization (Blay J et al J Clin Oncol 2006). Similarly, neither efficacy nor a significant response rate was observed in metastatic synovial sarcoma patients who have been treated with Herceptin (Her2 antibody), R1507 (IGF-IR antibody), Gleevec (TKI inhibitor), or Afinitor (mTOR inhibitor). There are several studies that that are currently being conducted in sarcoma which include synovial sarcoma.

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 observed in approximately 10-50% of melanoma, breast, prostate, lung, ovarian, thyroid,

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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.

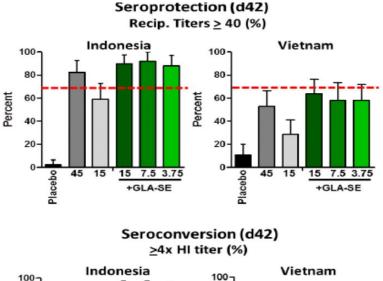
Infectious Disease/ Allergy Immunotherapy Collaboration Programs

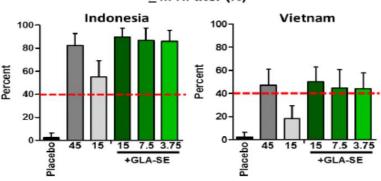
GLA-SE/G100 Augments the Efficacy of Influenza Vaccine: IMDZ has partnered several programs within its GLAAS platform as adjuvant immunotherapy for the treatment of infectious diseases or for food allergy. GLA molecules when combined with infectious disease antigens have been shown to boost pre-existing T cells and trigger a broad antibody response allowing for diverse antigen recognition. Clinical trials involving GLA + infectious disease antigen in over 1,000 volunteers have shown to enhance innate immunity and increasing the antibody response to disease antigens. Peripheral blood monocytes taken from 12 healthy volunteers when challenged with split-virus influenza +GLA-SE significantly increased the proportion of dendritic cells which resulted in the production of TNF-α, IL-6 and IL-12, IL-10, Granzyme B. Previous studies have shown that IL-10 and Granzyme B have been shown to correlate with protection from influenza virus. Clinical trials conducted with GLA-SE have corroborated these preclinical findings. In 290 subjects of a randomized PII trial conducted by Medicago, GLA-SE + VLP influenza antigen combination not only reduced the dosage of VLP antigen but also showed statistical significance in increased seroconversion and seroprotection when compared to VLP antigen alone. GLA-SE when combined with 3.75, 7.5, or 15 ug of VLP antigen showed equal level of seroprotection and seroconversion suggesting that GLA-SE reduces the dosage of VLP antigen required for activation of innate immunity. Seroprotection and seroconversion are approved FDA end points for approval of influenza vaccines.

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Exhibit 14: Summary of PI results of GLA-SE + VLP influenza antigen





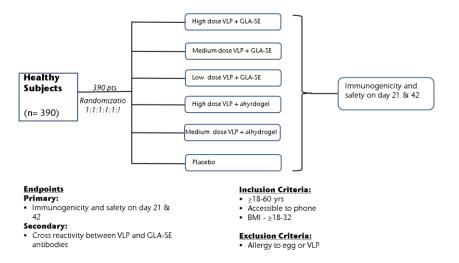
Source: Company presentation; Jefferies

GLA-SE+VLP antigen Phase II Design: Medicago's GLA-SE+VLP antigen combination is in a randomised, observer blind phase II trial that will enrol 390 subjects and measure the measure the immunogenicity and safety of GLA-SE+VLP antigen v. VLP antigen+PBO on day 21 and 42 as primary endpoint. Secondary endpoint of the study includes measuring the cross reactivity of VLP and GLA-SE. The patients will be divided into six arms :1) high dose VLP+GLA-SE; 2) medium dose VLP+GLA-SE; 3) low dose VLP+GLA-SE; 4) high dose VLP+PBO; 5) medium dose VLP+ PBO and 6) PBO only. Final top line data from the study is expected in H2 2014. Two doses of vaccine combinations or PBO controls will be given 21 days apart in this study. The trial will enroll patients who are between 18-60 yrs, accessible by phone, and BMI between ≥18-32. Exclusion criteria for the trial includes patients who are allergic to egg, VLP antigen and vaccine treatment within last 30 days.

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Exhibit 15: VLP+GLA-SE PII study design



Source: www.clinicaltrials.gov

MedImmune Collaboration: In October 2010, IMDZ granted three separate license agreements for the use of GLA to MedImmune for three different infectious disease indications and received a \$4.5M upfront payment. Upon reaching various developments, regulatory and commercial milestone, IMDZ will receive additional aggregate payments of \$62.9-\$76.0 million. IMDZ will also receive non-royalties in the low double-digit percentage from sub-licensees and a mid-single-digit royalty on net sales of licensed products, which royalty is subject to reduction under certain circumstances. ImDZ will continue to receive royalty from MedImmune for 10 years on a country-by-country basis, after the first commercial sale of the first licensed product in the applicable country and will continue on a country-by-country and product-by-product basis, for the life of the licensed patents that cover the sale of the applicable product in the applicable country.

Sanofi Collaboration: In August 2014, IMDZ and Sanofi (SAN entered into an agreement for the use of IMDZ's GLAAS platform for development of treatments for food allergy. Nearly 15M people in US and 200-250M people worldwide suffer from food allergy. IMDZ received an undisclosed amount of upfront milestone payment and will receive an additional \$168M upon achieving development and commercialization milestones, as well as tiered royalties on sales of approved products.

IDRI Collaboration: IMDZ has acquired worldwide exclusive licensed for the GLA molecule and know how technology of TLR-4 agonists from Infectious Disease Research Institute (IDRI), and the licensing covers all of IMDZ's GLAAS platform products for the treatment, prevention or diagnosis of any disease or condition. The license, however, does not retain rights for certain infectious diseases (HIV, pneumonia) found in low-income countries and IMDX has limited rights in specific territories, indications, and/or a narrow subset of synthetic TLR4 agonist-containing products. IMDZ has non-exclusive rights for two other synthetic TLR4 agonists that are limited to cancer and specific infectious diseases, which are within IMDZ's core focus. Under the agreement, IMDZ has paid a \$1.4M upfront payment and IMDZ issued shares covering the annual fees, sublicensing fees and financial support of continuing research on GLA. IMDZ is obligated to make additional \$2.4M and \$1.3m payments upon reaching certain developmental

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and regulatory milestones for the first GLA product, and for each subsequent GLA product, respectively. Upon commercialization, IMDZ will pay IDRI additionally a low single-digit royalty on net sales of licensed products.

GLAAS Platform for Infectious diseases

IMDZ's GLAAS platform uses a small synthetic molecule, Glucopyranosyl Lipid A (GLA) as a TLR-4 agonist resulting in strong antibody responses for infectious disease or allergy antigens. IMDZ's GLA technology/TLR-4 agonist technology has been licensed from Infectious Disease Research Institute (IDRI). Stable emulsion of GLA (GLA-SE) has been shown to induce strong Th1 responses that cause the activation of CD4 T cells and providing immunity against tuberculosis and influenza. In preclinical mouse models, mice that were given GLA-SE followed by H5N1 influenza virus challenge were protected from the influenza and weight loss vs. mice administered only a stable emulsion followed by a virus challenge (Clegg CH et al PNAS 2012). Similar results were observed with challenges of mycobacterium tuberculosis. GLA-SE +ID93 tuberculosis antigen vaccination was able to protect the mice or guinea pigs from the infection of tuberculosis, while the combination of only SE+ID93 was not able to protect the mice from tuberculosis as assessed by reductions in bacterial burden, survival, and pathology (Baldwin SL et al J Immunology 2012). Based on these findings we believe that GLA-SE would be an effective adjuvant for treatment of infectious diseases.

IP/Litigation

The patent for GLA and ZVex both expire in 2027; however, the patent for modified version of ZVex vector made by IMDZ lasts until 2032. IMDZ holds the patent for CMB305 and it lasts until 2032.

Litigation with Theravectys: In July 2015, Theravectys, a French-based company, filed a lawsuit against IMDZ in the Delaware Court of Chancery alleging that IMDZ induced its lentiviral manufacturer Henogen SA to breach its exclusivity agreement with Theravectys for the manufacturing lentiviral vectors. Under the lawsuit, Theravectys claims that IMDZ misappropriated Theravectys' trade secrets and violated its contractual relationship with Henogen. IMDZ has disclosed that it currently is not using Henogen to manufacture its lentiviral vectors and IMDZ states it has developed its own manufacturing process. We don't expect the lawsuit will pose a hurdle to IMDZ initiating its clinical trials. Under a worst-case scenario, we would estimate Theravectys could be entitled to a low single digit royalty.

Capital Structure

After its IPO on June 2014, in which IMDZ raised gross proceeds of \$60.0 million from a public equity offering of 5 million shares of common stock at \$12/share, the company has cash of \$69.9M, which is sufficient to fund operations to 2016. Lock-up expiration from the IPO occur 180 days post-IPO. IMDZ plans to use the net proceeds to fund clinical trials of LV305, G100, G305 and CMB305, pipeline development, and remainder for working capital and other general corporate purposes. Jefferies was joint book-running manager for the transaction.

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Management Team

Carlos Paya, M.D., Ph.D. - Chairman, President & Chief Executive Officer

Carlos Paya joined Immune Design in May 2011 as President, Chief Executive Officer and director. He previously served as President of Elan Corporation, November 2008 to April 2011, which was acquired by Perrigo Company. Before joining Elan Corporation, Dr. Paya was at Eli Lilly & Company, from September 2001 to November 2008, as Vice President, Lilly Research Laboratories. Dr. Paya was Professor of Medicine, Immunology, and Pathology, and Vice Dean of the Clinical Investigation Program at the Mayo Clinic in Rochester, Minnesota, from 1991 to 2001. He received his M.D. and Ph.D. degrees from the University of Madrid and underwent postdoctoral training at the Institute Pasteur, Paris, France.

Stephen R. Brady, JD. LLM - Chief Business Officer

Stephen R. Brady joined Immune Design in September 2013 as Chief Business Officer. Previously he was at 3-V Biosciences, Inc., from April 2010 to August 2013, first as Vice President, Corporate Development, Strategy and Operations, and then As Chief Business Officer. From April 2007 to March 2010, he was at Proteolox, Inc., most recently serving as Vice President, Corporate Development. Prior to Proteolix, Inc., Mr. Brady served as Senior Corporate Counsel at Lexicon Pharmaceuticals, Inc. Mr. Brady was also a Vice President with Lazard Venture Advisors, a division of Lazard Freres & Co. LLC, and an associate at Morrison & Foerster LLP. Mr. Brady received a B.A. in English from the University of Oregon, a J.D. from the University of the Pacific and an LL.M. from New York University School of Law.

Wayne Gombotz, Ph.D. - Chief Development Officer

Wayne Gombotz joined Immune Design in December 2011 as Chief Development Officer. He previously served as Vice President Pharmaceutical Operations at Omeros Corporation, from May 2005 to October 2011. Before joining Omeros Corporation, Dr. Gombotz held several executive management positions including Vice President, Process Science & Pharmaceutical Development at Corixa Corporation (2002 to 2005), and Sr. Director, Analytical Chemistry and Formulation at Immunex Corporation (1993 to 2002). He was a staff scientist at Bristol-Myers Squibb and Enzytech Inc. Dr. Gombotz also currently serves as an Advisory Board Chair of the Center for Intracellular Delivery of Biologics and an Advisory Board Member for the University of Washington's Department of Bioengineering. Dr. Gombotz received an M.S. and Ph.D. degree in Bioengineering from the University of Washington where he is an Affiliate Assistant Professor.

Frank J. Hsu, M.D., - Vice President, Head of Oncology

Frank Hsu joined Immune Design in October 2013 as Vice President, Head of Oncology. Dr. Hsu recently served as Chief Medical Officer for Zyngenia, Inc., where he was responsible for strategic planning and clinical development of multi-specific, multi-valent agents for the company's lead programs in immune-mediated diseases and oncology. Prior to that, Dr. Hsu was Senior Medical Director at Genzyme in the Transplant and Oncology Division, where for more than nine years he was responsible for the clinical development of products in the areas of hematology, oncology, and stem cell transplant, and supported medical affairs and corporate development. From 1996-2003 he served as an Assistant Professor of Medicine in the Section of Oncology and as co-Director of the Immunology Research Program of the Yale Cancer Center. In addition to his current role, he serves on the Board of Trustees of the Grunebaum Cancer Research Foundation. Dr. Hsu received his B.S. from Stanford University, his M.D. from Harvard Medical School, and his residency training in Internal Medicine at UCSF. He was a clinical and research fellow in Oncology at Stanford University from 1990-1996.

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Richard T. Kenney, M.D., FACP - Chief Medical Officer

Richard T. Kenney joined Immune Design in September 2013 as Chief Medical Officer. He previously served as Chief Medical Officer for Crucell Holland BV, from July 2012 to August 2013. From December 2009 to June 2012 he was at Vical Incorporated, a publicly traded biopharmaceutical company, in various key positions, and last served as Senior Vice President, Clinical Development. Dr. Kenney held key positions in vaccine development at GSK Biologicals, from December 2005 to November 2009, and he most recently served as Senior Director of Global Clinical R&D. From April 2005 to December 2005 he served as Vice President, Clinical Development of ID Biomedical until it was acquired by GSK Biologicals. Dr. Kenney had various positions at Iomai Corporation from March 2001 to April 2005, where he most recently served as Vice President, Medical and Regulatory Affairs. Dr. Kenney was a Lead Research Investigator at the FDA, Center for Biologics Evaluation and Research, Office of Vaccine Research and Review from July 1995 to February 2001. Dr. Kenney completed his residency in internal medicine at Duke University Medical Center, and received his postdoctoral training at the National Institutes of Health, National Institute of Allergy and Infectious Diseases, completing a fellowship in infectious diseases, and then post-doctoral training in molecular parasitology and tropical medicine. He received board certifications in Internal Medicine and Infectious Diseases. He earned his M.D. degree at Harvard Medical School and graduated with special honors from George Washington University.

Jan Henrik ter Meulen, M.D., Dr. habil. DTM&H, - Chief Scientific Officer

Jan Henrik ter Meulen joined Immune Design in October 2013 as Chief Scientific Officer. He previously served as Executive Director of Vaccine Research and Head of Department of Vaccine Basic Research at Merck Research Laboratories, from April 2008 to September 2013. Prior to Merck, March 2003 to April 2008, Dr. ter Meulen served as Executive Director Infectious Diseases at Crucell Holland and, from September 2006 to April 2008, as Chief Scientific Officer at Etna Biotech S.r.l., a subsidiary of Crucell. Dr. ter Meulen has an M.D. from Albert Ludwigs University Freiburg im Breisgau, a medical doctorate from Julius Maximilians University Wuerzburg, a higher doctorate from Philipps University Marburg, a Diploma in Tropical Medicine and Hygiene from the London School, and is a board certified in Clinical Microbiology by the Chambers of Physicians, Hamburg Germany.

Paul Rickey - Vice President, Finance & Administration

Paul Rickey joined Immune Design in July 2009 and serves as Vice President, Finance and Administration, Secretary and Treasurer. Prior to joining Immune Design, he served in various positions, most recently Corporate Controller of Northstar Neuroscience, a publicly traded medical device company, from July 2006 to June 2009. He was the Accounting Manager at Mobliss Inc., a wireless software company, from 2004 to 2006 and was an employee of Ernst & Young LLP, an international professional services firm, from 2001 to 2004. Mr. Rickey has a B.A. in Business Administration, Accounting, and Masters in Professional Accounting from the University of Washington. He currently serves on the board of the Northwest Association of Bioscience Financial Officers and received his Certified Public Accountant Certification from the state of Washington and currently holds an active license.

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Exhibit 16: IMDZ income statement

Immune Design

Quarterly Income Statement

	2012 A	2013A			2014E			2015E	2016E	2017E	2018E	2019 E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032
	FY	FY	1QA	2QA	3QE	4QE	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY
Revenue:																									
CMB305 - synovial sarcoma	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	10.3	21.1	32.6	33.6	37.0	39.7	40.9	42.1	43.4	44.7	4
CM B 305 - NSCLC	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	13.5	85.1	119.3	156.6	1810	207.4	236.1	255.7	276.6	294.8	3
G100 - M CC	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	12.1	14.8	17.7	19.9	212	22.6	24.0	25.6	26.6	27.7	28.8	
License and collaboration revenues	3.0	16	0.0	0.3	0.3	0.3	0.8	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	2.0	2.0	
Total revenue, net	3.0	1.6	0.0	0.3	0.3	0.3	0.8	2.0	2.0	2.0	2.0	2.0	2.0	14.1	40.5	126.0	173.8	213.4	242.5	273.1	304.5	326.4	349.7	370.3	392
Costs and expenses:																									
Cost of goods sold	15	0.7	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	18	5.8	18.6	25.8	317	36.1	40.7	45.4	48.7	52.2	55.2	5
Research & development	8.6	11.6	4.1	4.0	4.2	3.9	16.2	26.8	25.0	25.0	19.3	20.3	20.9	215	22.1	22.8	23.5	24.0	24.4	24.9	25.4	25.9	26.2	26.5	
Selling, general & administrative	3.7	4.4	14	1.4	1.4	1.4	5.6	2.6	2.8	3.0	3.3	3.4	3.6	13.7	14.1	14.6	15.0	15.4	15.9	16.4	16.9	17.4	17.9	18.4	
otal operating expenses	13.8	16.7	5.5	5.4	5.6	5.3	21.8	29.4	27.8	28.0	22.6	23.7	24.5	37.0	42.1	56.0	64.3	71.1	76.4	82.0	87.7	92.0	96.2	100.1	104
ncome (loss) from operations	(10.9)	(15.1)	(5.5)	(5.2)	(5.4)	(5.1)	(21.0)	(27.4)	(25.8)	(26.0)	(20.6)	(21.7)	(22.5)	(23.0)	(1.5)	70.0	109.5	142.3	166.1	191.1	216.8	234.4	253.4	270.2	287
ther income (expense):																									
Miscellaneous (expense) income	0.0	(10)	(2.7)	0.0	0.0	0.0	(2.7)	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	0.0	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	0.0	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	0.0	0.0	0.0	0.0	0.0	0.0	
ilitelest 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	0.0	0.0	0.0	0.0	0.0	0.0	
et profit (loss) before income taxes	(10.8)	(16.0)	(8.2)	(5.2)	(5.4)	(5.1)	(21.0)	(27.4)	(25.8)	(26.0)	(20.6)	(21.7)	(22.5)	(23.0)	(1.5)	70.0	109.5	142.3	166.1	191.1	216.8	234.4	253.4	270.2	28
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	7.0	11.0	14.2	16.6	66.9	75.9	82.0	88.7	94.6	1
Income tax (%)										0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	10.0%	10.0%	10.0%	10.0%	35.0%	35.0%	35.0%	35.0%	35.0%	35
et Income (GAAP)	(10.8)	(16.0)	(8.2)	(5.2)	(5.4)	(5.1)	(21.0)	(27.4)	(25.8)	(26.0)	(20.6)	(21.7)	(22.5)	(23.0)	(1.5)	63.0	98.6	128.1	149.5	124.2	140.9	152.4	164.7	175.6	18
Adjusted Items (Non-GAAP)																									
Stock options	0.3	0.8	0.2	0.2	0.2	0.2	0.8	12	15	2.0	2.0	2.0	2.0	2.5	2.5	3.0	3.0	3.0	4.0	5.0	5.0	5.0	5.0	6.0	
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	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	
et Income (Non-GAAP)	(10.5)	(15.2)	(8.0)	(5.0)	(5.2)	(4.9)	(20.2)	(26.2)	(24.3)	(24.0)	(18.6)	(19.7)	(20.5)	(20.5)	1.0	66.0	101.6	131.1	153.5	129.2	145.9	157.4	169.7	181.6	19
PS, GAAP																									
Basic	(30.43)	(2.28)	(0.81)	(0.30)	(0.31)	(0.29)	(1.71)	(158)	(122)	(1.19)	(0.83)	(0.86)	(0.82)	(0.83)	(0.05)	2.22	3.44	4.42	5.11	4.21	4.72	5.06	5.41	5.71	6
iluted	\$ (30.43)	\$ (2.28)	\$ (0.81)	\$ (0.30)	\$ (0.31)	\$ (0.29) \$	(1.71)	\$ (1.58)	\$ (1.22)	\$ (1.19)	\$ (0.83)	\$ (0.86)	\$ (0.82)	\$ (0.82)	\$ (0.05)	\$ 2.16	\$ 3.31	\$ 4.21	\$ 4.82	\$ 3.93	\$ 4.37	\$ 4.63	\$ 4.90	\$ 5.13	\$ 5.
leighted average share- Basic	0.4	7.0	10.1	17.4	17.4	17.4	15.6	17.4	212	219	24.8	25.3	27.6	27.8	28.1	28.4	28.7	29.0	29.2	29.5	29.8	30.1	30.4	30.7	
Veighted average share- Diluted	0.4	7.0	10.1	17.4	17.4	17.4	15.6	17.4	212	219	24.8	25.3	27.6	28.1	28.7	29.2	29.8	30.4	310		32.3	32.9	33.6	34.3	

Initiating Coverage

August 18, 2014

Exhibit 17: IMDZ balance sheet

Immune Design

Balance Sheet

(All values in \$MM)														
	2012A	2013 A	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY
Current assets:														
Cash and cash equivalents	12.8	30.4	69.9	45.0	73.0	48.5	83.0	64.8	96.2	77.7	80.6	148.6	251.0	382.9
Cash and investments	12.8	30.4	69.9	45.0	73.0	48.5	83.0	64.8	96.2	77.7	80.6	148.6	251.0	382.9
Accts receivable	0.5	0.1	0.1	0.1	0.2	0.2	0.3	0.5	0.5	0.5	0.5	0.5	0.5	0.5
Inventory	0.3	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
Prepaid expenses	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	13.7	30.7	70.2	45.3	73.4	48.9	83.5	65.4	96.9	78.4	81.3	149.3	251.7	383.6
Property and equipment, net	0.5	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
Total assets	14.3	31.0	70.5	45.6	73.7	49.2	83.8	65.7	97.2	78.7	81.6	149.6	252.0	383.9
Current liabilities:														
Accounts payable	11	0.9	1.7	2.1	3.6	2.3	4.5	5.1	6.2	7.3	8.4	9.5	9.5	9.5
Accrued expenses	15	1.1	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0
Deferred rent	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	2.6	2.0	3.7	4.1	5.6	4.3	6.5	7.1	8.2	9.3	10.4	11.5	11.5	11.5
Deferred rent, excluding current portion	0.0	0.1	1.4	1.4	1.4	14	14	1.4	1.4	14	14	1.4	14	14
Total Liabilitiy	2.6	2.1	5.1	5.5	7.0	5.7	7.9	8.5	9.6	10.7	11.8	12.9	12.9	12.9
Total stockholders' equity	11.6	28.9	65.4	40.1	66.7	43.5	75.9	57.2	87.6	68.0	69.8	136.7	239.1	371.0
Total liabilities and stockholders' equity	14.2	31.0	70.5	45.6	73.7	49.2	83.8	65.7	97.2	78.7	81.6	149.6	252.0	383.9

Source: Company data and Jefferies estimate

August 18, 2014

Exhibit 18: IMDZ cash flow statement

Immune Design

Cash Flow Statement

(All values in \$MM)														
	2012A	2013 A	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
	FY	FY	FY	FY	FY									
Cash flows from operating activities:	(10.8)	(16.0)	(210)	(27.4)	(25.8)	(26.0)	(20.6)	(217)	(22.5)	(23.0)	(15)	63.0	98.6	128.
Net income														
Adjustments to reconcile cash by operating activities:														
Depreciation and amortization expense	0.5	0.4	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2
Compensation expense	0.2	0.3	8.0	1.2	1.5	2.0	2.0	2.0	2.0	2.5	2.5	3.0	3.0	3.0
Revaluation of convert preferred stock liability/other	0.0	1.0	2.7	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:														
Acct receivable	(0.5)	0.4	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Inventory	(0.3)	0.1	0.1	0.4	1.5	(13)	2.2	0.6	11	11	11	11	0.0	0.0
Prepaid expenses	0.2	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
Accounts payable	0.5	(0.2)	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	(0.4)	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	(9.6)	(14.3)	(16.0)	(24.8)	(21.8)	(24.3)	(15.4)	(18.1)	(18.4)	(18.3)	3.1	68.1	102.6	132.1
Cash flows from investing activities: Purchase of fixed assets	(0.3)	(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
Net cash (used in) provided by investing activities	(0.3)	(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)
Net cash (asea in) provided by investing activities	(0.3)	(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)
Cash flows from financing activities:														
Issuance of common stock, net of offering costs	0.0	0.0	55.8	0.0	50.0	0.0	50.0	0.0	50.0	0.0	0.0	0.0	0.0	0.0
Issuance of common stock from exercise of stock options	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
Proceeds from preferred stock	10.6	32.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Proceeds from notes payable	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
Principal payments on debt	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	10.6	32.1	55.8	0.0	50.0	0.0	50.0	0.0	50.0	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	0.8	17.6	39.6	(25.0)	28.0	(24.5)	34.5	(18.3)	31.5	(18.5)	2.9	68.0	102.4	131.9
Cash and cash equivalents at beginning of period	12.0	12.8	30.3	69.9	45.0	73.0	48.5	83.0	64.8	96.2	77.7	80.6	148.6	2510
Cash and cash equivalents at end of period	12.8	30.3	69.9	45.0	73.0	48.5	83.0	64.8	96.2	77.7	80.6	148.6	251.0	382.9

Initiating Coverage

August 18, 2014

Exhibit 19: IMDZ DCF analysis

Immune Design

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	2029E	2030E	2031E	2032E
•	-																				
Sales	3.0	1.6	8.0	2.0	2.0	2.0	2.0	2.0	2.0	14.1	40.5	126.0	173.8	213.4	242.5	273.1	304.5	326.4	349.7	370.3	392.2
Operating Expenses	13.8	16.7	21.8	29.4	27.8	28.0	22.6	23.7	24.5	37.0	42.1	56.0	64.3	71.1	76.4	82.0	87.7	92.0	96.2	100.1	104.2
EBIT	(10.9)	(15.1)	(21.0)	(27.4)	(25.8)	(26.0)	(20.6)	(21.7)	(22.5)	(23.0)	(1.5)	70.0	109.5	142.3	166.1	191.1	216.8	234.4	253.4	270.2	287.9
(-): Taxes	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	7.0	11.0	14.2	16.6	66.9	75.9	82.0	88.7	94.6	100.8
EBIAT	(10.9)	(15.1)	(21.0)	(27.4)	(25.8)	(26.0)	(20.6)	(21.7)	(22.5)	(23.0)	(1.5)	63.0	98.6	128.1	149.5	124.2	140.9	152.4	164.7	175.6	187.1
(+):Depreciation	0.5	0.4	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0
(+):FAS-123 Options	0.3	0.8	0.8	1.2	1.5	2.0	2.0	2.0	2.0	2.5	2.5	3.0	3.0	3.0	4.0	5.0	5.0	5.0	5.0	6.0	6.0
(-): Capital expenditures	0.3	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	0.0	0.0	0.0	0.0	0.0	0.0	0.0
(-): Changes in working capital	1.9	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	0.0	8.0	0.0	0.0	0.0
Unlevered free cash flow	(12.2)	(14.2)	(20.1)	(26.1)	(24.2)	(24.0)	(18.5)	(19.6)	(20.4)	(20.4)	1.1	66.1	101.6	131.2	153.5	129.2	145.9	156.6	169.7	181.6	193.1

Source: Jefferies estimates, company data

\$79.6 129 73% \$19.9 \$19.9 129 2024 2,988 29 926 31%	75% \$21.2 \$ \$21.2	7%	75% \$22.6 \$22.6	7%	75% \$24.0 \$24.0	696	\$25.6 \$25.6 \$25.6	6%	\$106.4 75% \$26.6 \$26.6 2029	496	2030	4%	2031	4%	\$119.9 75% \$30.0 \$30.0 2032
75% \$19.9 \$19.9 12% 2024 2,988 29	75% \$21.2 \$21.2 2025 3,047 945	7%	75% \$22.6 \$22.6 \$22.6 2026	7%	75% \$24.0 \$24.0 2027	6%	75% \$25.6 \$25.6 \$25.6	6%	75% \$26.6 \$26.6 2029	4%	75% \$27.7 \$27.7 2030	4%	75% \$28.8 \$28.8 2031	4%	75% \$30.0 \$30.0
\$19.9 \$19.9 129 2024 2,988 29 926	\$21.2 \$21.2 2025 3,047 945		\$22.6 \$22.6 2026 3,108		\$24.0 \$24.0 2027		\$25.6		\$26.6 \$26.6 2029		\$27.7		\$28.8 \$28.8 2031		\$30.0 \$30.0 2032
\$19.9 F 129 2024 2,988 29 926	2025 3,047 945		\$22.6 2026 3,108		\$24.0 ° 6		\$25.6		2029		\$27.7 2030		\$28.8		\$30.0
2024 2,988 29 926	2025 3,047 945		2026 3,108		2027		2028		2029		2030		2031		2032
2,988 ²⁹	3,047 945	2%	3,108	2%		2%		2%		2%		296		2%	
926	945	2%	,	2%	3,171	2%	3,234	2%	3,299	2%	3,365	2%	3,432	2%	3,501
926	945	2%	,	2%	3,171	296	3,234	2%	3,299	2%	3,365	2%	3,432	2%	3,501
			964												
31%	31%				983		1,003		1,023		1,043		1,064		1,085
			31%		31%		31%		31%		31%		31%		31%
269	274		280		285		291		297		303		309		315
9%	9%		9%		9%		9%		9%		9%		9%		9%
490 109	5 512	4%	535	4%	558 4	196	582	4%	594	2%	606	2%	618	2%	630
41.0%	42.0%		43.0%		44.0%		45.0%		45.0%		45.0%		45.0%		45.0%
\$79.6 129	\$84.8	7%	\$90.3	7%	\$96.1	6%	\$102.3	6%	\$106.4	4%	\$110.7	496	\$115.2	4%	\$119.9
0%	096		0%		0%		096		096		0%		096		096
\$79.6	\$84.8		\$90.3		\$96.1		\$102.3		\$106.4		\$110.7		\$115.2		\$119.9
	\$79.6 12% 0%	41.0% 42.0% \$79.6 12% \$84.8 0% 0%	41.0% 42.0% \$79.6 12% \$84.8 7% 0% 0%	41.0% 42.0% 43.0% \$79.6 12% \$84.8 7% \$90.3 0% 0% 0%	41.0% 42.0% 43.0% \$79.6 12% \$84.8 2% \$90.3 2% 0% 0% 0%	41.0% 42.0% 43.0% 44.0% 579.6 12% \$84.8 7% \$90.3 7% \$96.1 0% 0% 0%	41.0% 42.0% 43.0% 44.0% \$79.6 12% \$84.8 7% \$90.3 7% \$96.1 6% 0% 0% 0% 0% 0%	41.0% 42.0% 43.0% 44.0% 45.0% 579.6 12% \$84.8 7% \$90.3 7% \$96.1 6% \$102.3 0% 0% 0% 0%	41.0% 42.0% 43.0% 44.0% 45.0% 45.0% 579.6 12% 584.8 7% 590.3 7% 596.1 6% 5102.3 6% 0% 0% 0% 0% 0%	41.0% 42.0% 43.0% 44.0% 45.0% 45.0% 45.0% 579.6 12% 584.8 7% 590.3 7% 596.1 6% 5102.3 6% 5106.4 0% 0% 0% 0% 0% 0% 0%	41.0% 42.0% 43.0% 44.0% 45.0% 45.0% 579.6 12% \$84.8 7% \$90.3 7% \$96.1 6% \$102.3 6% \$106.4 4% 0% 0% 0% 0% 0% 0%	41.0% 42.0% 43.0% 44.0% 45.0% 45.0% 45.0% 45.0% 45.0% 579.6 12% 584.8 7% 590.3 7% 596.1 6% 5102.3 6% 5106.4 4% 5110.7 0% 0% 0% 0% 0% 0% 0% 0% 0%	41.0% 42.0% 43.0% 44.0% 45.0% 45.0% 45.0% 45.0% 579.6 12% 584.8 7% 590.3 7% 596.1 6% 5102.3 6% 5106.4 4% 5110.7 4% 6% 6% 6% 6% 6% 6% 6% 6% 6% 6% 6% 6% 6%	41.0% 42.0% 43.0% 44.0% 45.0% 45.0% 45.0% 45.0% 45.0% 45.0% 45.0% 45.0% 45.0% 45.0% 45.0% 45.0% 45.0% 45.0% 45.0% 45.0% 45.0% 65.0%	41.0% 42.0% 43.0% 44.0% 45.0% 45.0% 45.0% 45.0% 45.0% 45.0% 579.6 12% 584.8 7% 590.3 7% 596.1 6% 5102.3 6% 5106.4 4% 5110.7 4% 5115.2 4% 0% 0% 0% 0% 0% 0% 0% 0% 0% 0% 0%

Initiating Coverage

August 18, 2014

Exhibit 21: CMB305 revenue build for NSCLC

CMB305 U.S. NSCLC revenue build		2022		2023		2024		2025		2026		2027		2028		2029		2030		2031		2032	
Total no. of lung cancer pts	3%	564,936	3%	581,884	3%	599,340	3%	617,320	3%	635,840	3%	654,915	3%	674,563	3%	694,800	3%	715,644	3%	737,113	3%	759,226	3%
No. of NSCLC pts % of total lung cancer pts	3%	480,195 85%	3%	494,601 85%	3%	509,439 85%	3%	524,722 85%	3%	540,464 85%	3%	556,678 85%	3%	573,378 85%	3%	590,580 85%	3%	608,297 85%	3%	626,546 85%	3%	645,342 85%	3%
No. of St. IIIb/St. IV NSCLC pts % of NSCLC pts	3%	360,146 75%	3%	370,951 75%	3%	382,079 75%	3%	393,542 75%	3%	405,348 75%	3%	417,508 75%	3%	430,034 75%	3%	442,935 75%	3%	456,223 75%	3%	469,909 75%	3%	484,007 75%	3%
No. of 1st-line NSCLC pts % of St. IIIb/St. IV NSCLC pts	3%	180,073 50%	3%	185,475 50%	3%	191,040 50%	3%	196,771 50%	3%	202,674 50%	3%	208,754 50%	3%	215,017 50%	3%	221,467 50%	3%	228,111 50%	3%	234,955 50%	3%	242,003 50%	3%
No. of 2nd-line NSCLC pts % of St. IIIb/St. IV NSCLC pts	3%	108,044 30%	3%	111,285 30%	3%	114,624 30%	3%	118,063 30%	3%	121,604 30%	3%	125,253 30%	3%	129,010 30%	3%	132,880 30%	3%	136,867 30%	3%	140,973 30%	3%	145,202 30%	3%
No. of 3rd/4th-line NSCLC pts % of St. IIIb/St. IV NSCLC pts	3%	72,029 20%	3%	74,190 20%	3%	76,416 20%	3%	78,708 20%	3%	81,070 20%	3%	83,502 20%	3%	86,007 20%	3%	88,587 20%	3%	91,245 20%	3%	93,982 20%	3%	96,801 20%	3%
No. of CMB305-treated pts % of CMB305-eligible pts		360 0.5%		2,226 3.0%	518%	3,057 4.0%	37%	3,935 5.0%	29%	4,459 5.5%	13%	5,010 6.0%	12%	5,590 6.5%	12%	5,935 6.7%	6%	6,296 6.9%	6%	6,579 7.0%	4%	6,873 7.1%	4%
CMB305 sales in 3rd/4th-line NSCLC Net price - CMB305		\$54.0 \$150,000		\$340.5 \$153,000	530% 2%	\$477.0 \$156,060	40% 2%	\$626.4 \$159,181	31% 2%	\$724.0 \$162,365	16% 2%	\$829.7 \$165,612	15% 2%	\$944.4 \$168,924	14% 2%	\$1,022.7 \$172,303	8% 2%	\$1,106.5 \$175,749	8% 2%	\$1,179.3 \$179,264	7% 2%	\$1,256.7 \$182,849	7% 2%
Risk discount		75%		75%		75%		75%		75%		75%		75%		75%		75%		75%		75%	
Total Net Sales of CMB305 in NSCLC - risk adj. (\$M	1)	\$13.5		\$85.1		\$119.3		\$156.6		\$181.0		\$207.4		\$236.1		\$255.7		\$276.6		\$294.8		\$314.2	

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Exhibit 22: CMB305 revenue build for synovial sarcoma

CMB-305	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032
<u>u.s.</u>											
Total Net Sales of CMB-305 in synovial sarcoma (SM)	\$41.0	\$84.5 106%	\$130.6 55%	\$134.5 3%	\$147.8 10%	\$158.6 7%	\$163.4 ° 3%	\$168.3 ^{3%}	\$173.4 ^{3%}	\$178.7 ⁷ 3%	\$184.1 3%
Risk discount	75%	75%	75%	75%	75%	75%	75%	75%	75%	75%	75%
Total Net Sales of CMB-305 in synovial sarcoma - risk adj. (\$M)	\$10.3	\$21.1	\$32.6	\$33.6	\$37.0	\$39.7	\$40.9	\$42.1	\$43.4	\$44.7	\$46.0
Total Net Sales of CMB-305 in synovial sarcoma - risk adj. (\$M)	\$10.3	\$21.1 106%	\$32.6 55%	\$33.6 3%	\$37.0 10%	\$39.7 7%	\$40.9 3%	\$42.1 3%	\$43.4 3%	\$44.7 3%	\$46.0 3%
	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032
U.S. Synovial Sarcoma Market Opportunity											
Total no. of synovial sarcoma patients (000's)	1,823 1%	1,841 1%	1,859 1%	1,878 1%	1,897 1%	1,916 1%	1,935 1%	1,954 1%	1,974 1%	1,993 1%	2,013 1%
Patients on CMB-305 (000's)	273	552	837 52%	845 1%	910 8%	958 5%	967 7 1%	977 1%	987 7 1%	997 7 1%	1,007 1%
% market share	15.0%	30.0%	45.0%	45.0%	48.0%	50.0%	50.0%	50.0%	50.0%	50.0%	50.0%
U.S. Net Sales of CMB-305 in synovial sarcoma (SM)	\$41.0	\$84.5 106%	\$130.6 55%	\$134.5 3%	\$147.8 10%	\$158.6 7%	\$163.4 ⁷ 3%	\$168.3 3%	\$173.4 3%	\$178.7 3%	\$184.1 3%
Risk discount	75%	75%	75%	75%	75%	75%	75%	75%	75%	75%	75%
U.S. Net Sales of CMB-305 in synovial sarcoma - risk adj. (SM)	\$10.3	\$21.1	\$32.6	\$33.6	\$37.0	\$39.7	\$40.9	\$42.1	\$43.4	\$44.7	\$46.0
Assumptions											
Total annual price (net)	\$150,000 0%	\$153,000 2%	\$156,060 2%	\$159,181 2%	\$162,365 2%	\$165,612 2%	\$168,924 2%	\$172,303 2%	\$175,749 2%	\$179,264 2%	\$182,849 2%

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Company Description

Immune Design Corp., a clinical-stage immunotherapy company, focuses on the development of novel immune-based therapies based on its DCVex and GLAAS discovery platforms for cancer and other chronic conditions. Its product candidates in Phase I clinical trials comprise LV305, CMB305, and G305 for the treatment of solid tumor types, such as breast cancer, melanoma, non-small cell lung cancer, ovarian cancer, or sarcoma; and G100 for the treatment of patients with merkel cell carcinoma. The company was founded in 2008 and is headquartered in Seattle, Washington.

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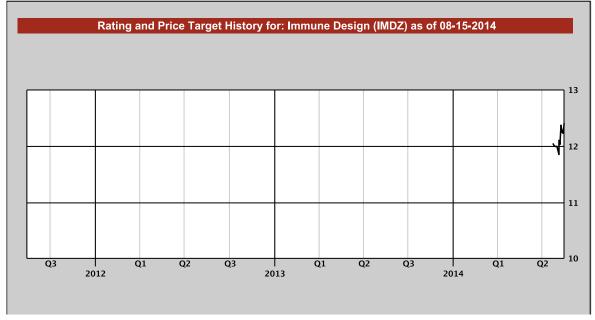
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			IB Serv./Past 12 Mos.			
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