

Equity Research

Immune Design Corp.

IMDZ: We Initiated Coverage With An Outperform Rating

- Summary:** We have initiated coverage of Immune Design with an Outperform rating and a \$17-18 valuation range. We believe IMDZ's differentiated platform has strong scientific merit for the treatment of cancers and believe shares will appreciate over time as the company's early-stage programs progress and potentially show activity. We see IMDZ as an attractive company that can become an important player longer term in the emerging immuno-oncology space. Our valuation range is based on a blend of discounted out-year EPS and sales multiples adjusted for probabilities of success in various solid tumor types.

- We believe IMDZ's immune-targeted platforms have strong scientific rationale and differentiated features.** The company's "ZVex" technology utilizes gene therapy specifically targeted to dendritic cells, in order to generate cytotoxic T-lymphocytes against specific tumor antigens. We believe both preclinical models testing ZVex, as well as early clinical data from other approaches such as CAR-T, demonstrate the clear promise of using activated T-cells to fight cancer. IMDZ's platform may also have some differentiating advantages, including more physiologic immune up-regulation, potential generation of immune memory capable of keeping cancers at bay, and utilization of GLA (through IMDZ's GLAAS platform) to further enhance immune responses.

- Based on this scientific foundation, we see a good probability that IMDZ's clinical candidates will show immune activation and antitumor effects in humans.** IMDZ is currently exploring LV305 (lentivirus) and G305 (TLR4 agonist) to generate immune responses against the tumor antigen NY-ESO-1, with initial ph. I safety and immunogenicity data in Q1 2015; management plans to combine the two in a "prime boost" strategy (CMB305) later this year. Though exact measures of immune activity may be difficult to benchmark, we expect, based on the science and preclinical evidence, that the study will likely show signals of antigen-specific immune up-regulation that would speak to the technology's potential in treating cancer. We also believe the choice of NY-ESO-1 as the initial tumor antigen to study makes sense given its likely safety and good validation.

- We see broad applicability for IMDZ's technology across multiple cancer types, with a large future market opportunity.** We agree with IMDZ's strategy of moving its programs forward in rare cancers, soft tissue sarcomas, which have high NY-ESO-1 expression and could enable a more rapid approval path, as well as 1-2 more common cancers like NSCLC and/or ovarian cancer, which could provide greater long-term revenue opportunity. We believe \$1.3B in worldwide sales of CMB305 could be achievable upon success in those solid tumors by 2023 and note that demonstration of efficacy in other cancer types, or utilization of the ZVex technology against other tumor antigens, could further increase the long-term revenue potential for IMDZ.

Valuation Range: \$17.00 to \$18.00 from NA to NA

Our valuation range is based on applying a 30x multiple to our probability-adjusted 2023E EPS and discounting at 15%, blended with a 5x multiple of 2023E probability-adjusted sales, discounting at 12%. Risks include ZVex's failure to show clinical efficacy, a safety signal, immunotherapy competition, and manufacturing.

Investment Thesis:

We believe IMDZ is undervalued based on the promise of ZVex/GLAAS as novel immunotherapy platforms for cancers.

Please see page 22 for rating definitions, important disclosures and required analyst certifications
All estimates/forecasts are as of 08/18/14 unless otherwise stated.

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Outperform / V

Sector: Biotechnology

Market Weight

Initiation of Coverage

EPS	2013A		2014E		2015E	
	Curr.	Prior	Curr.	Prior	Curr.	Prior
Q1 (Mar.)	NE	(\$0.81) A	NC	NE	NC	
Q2 (June)	NE	(0.60)	NE	NE	NC	
Q3 (Sep.)	NE	(0.52)	NE	NE	NC	
Q4 (Dec.)	NE	(0.40)	NE	NE	NC	
FY	(\$2.28)	(\$2.24)	NE	(\$1.61)	NE	
CY	(\$2.28)	(\$2.24)		(\$1.61)		
FYP/EPS	NM	NM		NM		
Rev.(MM)	\$2	\$0		\$0		

Note: 2014 quarterly EPS do not equal annual EPS due to calculation of EPS based on diluted vs. basic shares

Source: Company Data, Wells Fargo Securities, LLC estimates, and Reuters

NA = Not Available, NC = No Change, NE = No Estimate, NM = Not Meaningful

V = Volatile, ♦ = Company is on the Priority Stock List

Ticker	IMDZ
Price (08/15/2014)	\$12.16
52-Week Range:	\$11-13
Shares Outstanding: (MM)	15.8
Market Cap.: (MM)	\$192.1
S&P 500:	1,953.49
Avg. Daily Vol.:	0
Dividend/Yield:	\$0.00/0.0%
LT Debt: (MM)	\$0.0
LT Debt/Total Cap.:	0.0%
ROE:	NM
3-5 Yr. Est. Growth Rate:	NM
CY 2014 Est. P/EPS-to-Growth:	NM
Last Reporting Date:	06/23/2014

Source: Company Data, Wells Fargo Securities, LLC estimates, and Reuters

Brian Abrahams, M.D., Senior Analyst

(212) 214-8060

brian.abrahams@wellsfargo.com

Matthew J. Andrews, Senior Analyst

(617) 603-4218

matthew.j.andrews@wellsfargo.com

Shin Kang, Ph.D., Associate Analyst

(212) 214-5036

shin.kang@wellsfargo.com

Ronald Hsu, M.D., Associate Analyst

(212) 214-5064

ronald.hsu@wellsfargo.com

Together we'll go far



Company Description:

Immune Design Corporation (IMDZ) is a clinical stage biopharmaceutical company, headquartered in San Francisco, California and Seattle, Washington, developing novel immunotherapies for cancer and infectious diseases. IMDZ is developing an in vivo targeting approach to specifically target key regulatory immune cells called dendritic cells to enable the body's own immune system to fight cancer and other diseases. IMDZ's technology platform consists of two main components, IMDZVex ("ZVex") and GLAAS. ZVex is a virus-based cell targeting vector that specifically delivers tumor (or other immunogen) antigen of interest to DCs such that robust induction of cytotoxic T-cells in effect fights the disease. There are several pipeline products under clinical development: LV305 (ZVex-NY-ESO-1) and G305 (GLAAS-NY-ESO-1) in phase I study for solid tumors, CMB305 (ZVex-NY-ESO-1 plus GLAAS) expected to enter the clinic end-2014, and G100 (GLAAS) in phase I study in Merkel cell carcinoma.

Investment Thesis

We have initiated coverage of IMDZ with an Outperform rating and a \$17-18 valuation range. We believe Immune Design's differentiated immunotherapy platform has strong scientific merit for the treatment of cancers, and we expect shares to appreciate over time as the early-stage programs progress in the clinic and potentially demonstrate activity. We see Immune Design as an attractive idea in the emerging immuno-oncology space.

We believe IMDZ's immune-targeted ZVex and GLAAS platforms have strong scientific rationale and differentiated features. ZVex utilizes gene therapy specifically targeted to dendritic cells in order to generate cytotoxic T-lymphocytes (CTLs), which are designed to then attack tumor cells expressing the antigen of interest. Tumor antigens are any antigens that are expressed in greater amounts in tumors compared to normal tissues, and which can be targets for immune recognition of cancer. Though Immune Design's technology has not yet been proven effective in humans, we believe other companies' CAR-T platforms, which have shown good initial clinical responses in cancer patients, provide good validation of the concept of activating T cells against specific tumor antigens. Additionally, ZVex may even offer some potential advantages by virtue of its differentiated dendritic cell targeting, including more physiologic immune up-regulation, greater versatility for use against intracellular targets, less cumbersome preparation and administration, and induction of immune memory that could lead to persistent antitumor effects. In animal models, ZVex substantially increased the numbers of activated T cells, supporting the mechanistic approach, and showed tumor inhibitory effects in several animal models, providing some proof of principle, though with the caveat that animal models in cancer do not always translate well to humans. CTL generation by ZVex was boosted by IMDZ's second platform, GLAAS, a TLR4 agonist that less specifically stimulates the immune system. While TLR agonists have been used as adjuvants by others in the past, we believe its use in combination with ZVex – as it is planned to be tested in the future – could provide a novel "prime boost" strategy to maximize generation of a tumor-specific immune response and optimize potential anticancer benefits.

Based on this strong scientific foundation, we believe IMDZ's clinical candidates, while early stage, have a good likelihood of showing immune activation and ultimately, anticancer activity, in humans. IMDZ is testing LV305, a ZVex with a NY-ESO-1 tumor antigen payload, in phase I for various types of advanced solid tumors, with initial immunogenicity data expected in Q1 2015. The company subsequently plans to combine it with TLR4 agonist G305 (GLAAS + NY-ESO-1), also currently in phase I, in a combination termed CMB305. We believe the choice of NY-ESO-1 as a tumor antigen to study first makes sense: the antigen has minimal expression in normal tissues compared to tumors, minimizing safety risk of an autoimmune response, and it has been validated as an immunotherapy target in studies showing antitumor activity of NY-ESO-1 specific T-cells generated by other means. While exact measures of immune activity observed in the phase I may be difficult to benchmark, due to small patient numbers, a heterogeneous population, and limited historical comparators, we believe, based on the science and preclinical evidence, that the study will show signals of antigen-specific immune up-regulation (if not signals of clinical activity), which would speak to the promise of the technology in treating cancer.

We see broad potential applicability for IMDZ's technology across multiple cancer types, with a market opportunity of about \$1.3 billion by 2023. Following the phase I, IMDZ will likely study CMB305 in a common tumor type such as non-small cell lung cancer (NSCLC) or ovarian cancer, as well as in soft-tissue sarcomas. We believe this strategy makes sense. While larger tumors could provide a greater long-term revenue opportunity, soft tissue sarcomas, given their rarity and high rate of NY-ESO-1 expression, could potentially provide a more rapid path to approval, possibly enabling CMB305 to reach the market as early as 2018 if data are compelling. The development landscape in solid tumors is very crowded, and CMB305 would be viable only in the portion of patients whose cancers express NY-ESO-1. Still, we believe there remain significant unmet needs, particularly among refractory patients, and the prevalence of NY-ESO-1 expressing

tumors is not insubstantial – up to 30% in NSCLC and 60% in ovarian cancer – meaning the revenue opportunity could still be robust. Additionally, we believe there is good scientific rationale, as well as some preclinical validation, for combining ZVex-based agents with checkpoint inhibitors such as anti-PD-1 agents, an emerging class of cancer therapies, something that could help IMDZ both developmentally and commercially as the immune checkpoint therapies mature and potentially get incorporated into broader treatment paradigms. We see \$1.3B in potential worldwide sales of CMB305 by 2023 in soft-tissue sarcomas, NSCLC, and ovarian cancer, but note that CMB305 could have efficacy in other tumor types, as well, and ZVex could also utilize other tumor antigens, which could all expand the future market opportunity for IMDZ's technology.

Valuation

We have established a \$17-18 valuation range for Immune Design. We based our valuation analysis on our probability-adjusted revenue projections for CMB305 in soft-tissue sarcomas, where we assume a probability of success of 25%, NSCLC, where we assume a probability of success of 20%, and ovarian cancer, where we assume a probability of success of 20%. Assuming about 29 million shares diluted shares outstanding, which accounts for several potentially dilutive capital raises, as well as a \$100 million up-front payment for an ex-U.S. CMB305 partnership, we arrived at a probability-adjusted 2023 EPS of \$1.86. Applying a 30x multiple, which we believe is appropriate for a biotechnology company of Immune Design's stage potential, and discounting at 15% for slightly over eight years, would yield a valuation of \$17. Using a valuation analysis based on sales multiples, applying a 5x multiple on our estimated probability-adjusted worldwide product sales of \$266 million, and discounting back slightly over eight years at 12% yields a potential valuation of \$18. Blending these two methodologies would yield a valuation range of \$17-18.

Exhibit 1. Immune Design valuation analysis

Year:	2023E	Discount (yrs)	8.4	EPS:	\$1.86	Shares out:	29,189
Product:	Indication:	Region	Probability of success:	Prob-weighted sales	Net to company:	Probability-weighted EPS contribution:	
CMB305	Sarcomas	Worldwide	25%	\$37M	20%	\$0.26	
CMB305	NSCLC	Worldwide	20%	\$157M	20%	\$1.10	
CMB305	Ovarian cancer	Worldwide	20%	\$71M	20%	\$0.50	
Total				\$266M		\$1.86	
Discount Rate:							
EPS Multiple:	5%	10%	15%	20%	25%		
15	\$19	\$13	\$9	\$6	\$4		
20	\$25	\$17	\$12	\$8	\$6		
25	\$31	\$21	\$14	\$10	\$7		
30	\$37	\$25	\$17	\$12	\$9		
35	\$43	\$29	\$20	\$14	\$10		
40	\$49	\$33	\$23	\$16	\$11		
Discount Rate:							
Sales Multiple:	10%	11%	12%	13%	14%		
4	\$16	\$15	\$14	\$13	\$12		
5	\$20	\$19	\$18	\$16	\$15		
6	\$25	\$23	\$21	\$20	\$18		

Source: Wells Fargo Securities, LLC estimate

Upcoming Milestones and Product Pipeline

Exhibit 2. Upcoming milestones

Product	Event	Timeline
LV305	Ph.I safety and immunogenicity data	1Q15
G305	Ph.I safety and immunogenicity data	1Q15
CMB305	IND filing Initiate ph.I study Ph.I safety and immunogenicity data Initiate ph.II study	2H14 end-2014 2H15 2H15
G100	Complete Merkel cell carcinoma study	1Q15

Source: Company reports and Wells Fargo Securities, LLC estimates

Exhibit 3. Product pipeline

Product (partner)	Indication/mechanism	Status
LV305	Soft tissue sarcomas, melanoma, ovarian cancer, NSCLC, breast cancer	Phase I
G305	Soft tissue sarcomas, melanoma, ovarian cancer, NSCLC, breast cancer	Phase I
CMB305	Soft tissue sarcomas, melanoma, ovarian cancer, NSCLC	Preclinical
G100	Merkel cell carcinoma	Phase I
GLA (AZN/MedImmune)	Three infectious disease indications (MEDI7510 - RSV)	Phase I
GLA (Medicago)	Pandemic influenza	Phase I
GLS (Sanofi)	Allergy	Preclinical

Source: Company reports and Wells Fargo Securities, LLC

Key Risks

- **Clinical risk.** While there is a significant amount of preclinical data supporting ZVex's potential as a differentiated platform in immunotherapy, IMDZ's products remain very early stage, and there is no clinical data yet to corroborate that the platform will translate to humans. ZVex's ability to target DC cells has been well demonstrated in animal models and in human DCs in vitro, but we believe it will be key to show that ZVex targeting of human DC is sufficiently high enough and able to stimulate CTL response in humans, and that this translates to a clinical antitumor response. If ZVex-mediated antigen expression in DC is not achievable in humans in vivo and/or it is unable to induce sufficient CTL response, IMDZ's platform would likely be deemed less differentiated compared with other immunotherapy platforms, and may not ultimately succeed clinically. Although NY-ESO-1 is a well-characterized tumor antigen, it has never been tested in the ZVex system. As such, it is difficult to determine whether a robust CTL response could be generated to induce tumor killing. Additionally, it is possible that immune tolerance could be broken by ZVex such that an anti-ESO-1 immune response could lead to unforeseen autoimmune side effect.
- **Regulatory risk.** ZVex platform utilizes lentiviral vectors to stimulate the immune system, similar to gene therapy approaches. The food and Drug Administration (FDA) has never reviewed or approved a drug using viral vectors, which we believe adds regulatory uncertainty. We believe there could be a higher bar for safety as this novel therapeutic modality has a relatively limited safety record. Additionally, given that there are several immunotherapies with some overlapping features, any setbacks associated with other modalities might negatively affect the regulatory standards for the whole class.
- **Commercial risk.** ZVex-NY-ESO-1-based therapy is to be targeted to patients with tumors that express the target antigen. While a significant number of tumors that IMDZ is targeting express a meaningful level of NY-ESO-1, it is possible that even smaller subset of these patients might respond to treatment, which could further limit the addressable market. Additionally, given that the company is still in the early development phase, it is difficult to predict the competitive landscape when a ZVex-based therapy reaches the market. As there are several other competing immunotherapies in the same class (e.g., monoclonal TCRs and CAR-Ts) and separate classes, the target market could be significantly more crowded, leading to a potential lower end-market share.
- **Manufacturing risk.** Because the gene therapy field is still in a relatively nascent stage of development and there are no FDA-approved commercial products yet available, industrial-scale manufacturing technology and capacity remain limited to only a few specialized manufacturers. Due to the limited number of available suppliers, any disruption in the supply chain could significantly delay clinical development, regulatory approval, and/or commercial supply. Cost of goods is also likely to be more expensive than more traditional therapies such as small molecules.

Technology Platform

Overview

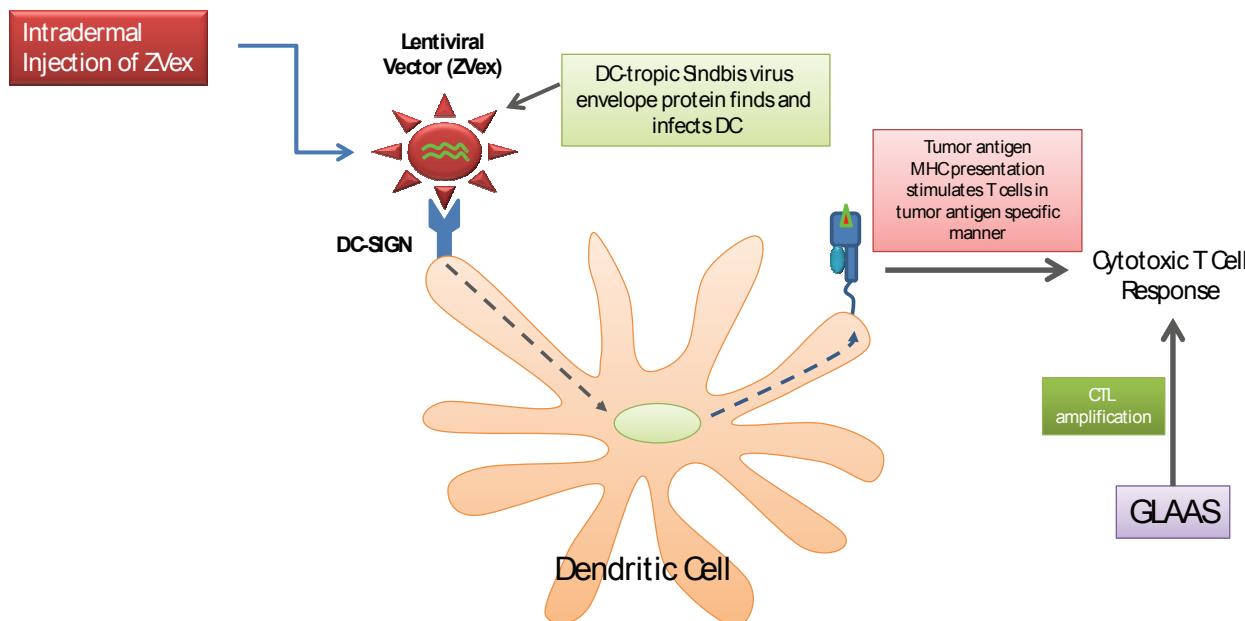
Immune Design's platform is based on a dual, "prime boost" approach to generating tumor-specific immune responses – ZVex and GLAAS. There are two main components to IMDZ's proprietary platform that differentiate it from other competing technologies: (1) cancer-fighting immune cells (cytotoxic T lymphocytes, or CTLs) are "primed" through the ZVex-treated dendritic cells, i.e., "professional" immune cells that instruct cytotoxic T cells to recognize foreign pathogens or tumor cells, and (2) once CTLs are primed (or educated to recognize and kill tumor antigen bearing cells), this population of primed, tumor-specific CTLs can be amplified by GLAAS, a well-validated immune-boosting agent through TLR4. The prime boost system utilizes *in vivo* delivery and stimulation of relevant immune cells to activate and boost cancer immunity.

ZVex – Educating Immune Cells to Recognize Tumors

ZVex utilizes gene therapy to generate cytotoxic T-lymphocytes (CTLs) "primed" to kill tumor cells. In contrast to traditional immunotherapy/cancer vaccine approaches, which require extracting relevant immune cell populations and manipulating these cells *ex vivo*—which makes preparation and administration of therapy cumbersome and inconvenient—ZVex (formerly called DCVex) targets this same subset of immune cells, namely DCs, specifically and efficiently through a lentiviral gene delivery system. Lentiviral vectors are well characterized and have gained growing use in drug development. An advantage over other viral vector therapy platforms is that lentivirus is known for efficient infectivity and protein expression, and greater safety, due to minimal immunogenicity. ZVex utilizes a non-integrating vector, which removes some of the theoretical safety concerns, and is a third-generation lentivirus that is recombination/replication incompetent.

As the central regulator of immune response, dendritic cells have long been the focus of cancer immunotherapies/vaccines. Dendritic cells are key players in the immune system that provide constant surveillance of the body's diverse antigens to distinguish self/normal, aberrant/transformed, and pathogen-derived antigens in order to instruct the effector cells, namely T-cells, as to what to mount a response against. Not only are dendritic cells important for the priming of T cells to initiate their activation and expansion in an antigen-specific manner, but they are also thought to be important in maximizing the memory response of T cells, which would provide, at least in theory, a long-lasting immune surveillance and response against relapsing cancer (or infection). While it is still unclear whether other similar/competitive technologies, such as monoclonal TCR or CAR-T, would also be able to induce a similar level of memory T cell response, based on its more physiologic DC-mediated priming and re-stimulation upon re-challenge, we believe the memory T cell response could be more robust with ZVex.

We view ZVex's ability to specifically target such dendritic cells *in vivo* as the most differentiated aspect of IMDZ's technology platform. Unlike most first-generation cancer immunotherapies, where "priming" takes place *ex vivo*, IMDZ's platform utilizes an engineered lentiviral construct that expresses a targeting molecule that specifically binds to DC-SIGN protein, a surface marker that is specifically expressed in dendritic cells. The basis of the "homing" mechanism is derived from envelope proteins of alphavirus, such Sindbis virus, which has natural tropism for dendritic cells. Sindbis virus is not generally found in the United States and Europe (other than Finland), so the proportion of patients with pre-existing antibodies that could theoretically neutralize the vector would likely be very low. Sindbis virus is transmitted through mosquito bites, but given that ZVex takes only the envelop proteins, it is unlikely that there is any risk for Sindbis infection or related symptoms. When the target dendritic cell is "infected" with the virus, it expresses the viral genes, particularly the corresponding tumor antigen, which are presented on the dendritic cell surface in the context of MHC presentation. Finally, these dendritic cells, now loaded with tumor antigen via MHC Class I, interact with CTLs, which then leads to a rapid activation and expansion of CTLs that are specifically targeted against the tumor antigen of interest.

Exhibit 4. Mechanism of action of ZVex technology

Source: Wells Fargo Securities, LLC

The dendritic cell targeting technology was developed at the laboratory of Dr. David Baltimore, a renowned scientist. Dr. Baltimore, the former president of Caltech, is one of the most renowned scientists in the world, given his contribution to science, including much of the pioneering work in virology, immunology, cancer biology, and genetic engineering. He became one of the youngest Nobel laureates at the age of 37. We believe that having core technology developed by such an important scientist's team gives additional credibility and validation to the foundation of Immune Design's platform, and we note that Dr. Baltimore remains involved in the company as a member of its board of directors and scientific advisory board. The original work and the preclinical proof-of-concept studies have been published in many reputable scientific journals.

Preclinical studies have indeed confirmed this highly specific targeting of dendritic cells, *in vitro* and *in vivo*. A proof-of-concept study for the DC-specific targeting lentivirus vector, which served as the basis for ZVex platform, was published in Nature Biotechnology (Vol. 26, no. 3, 3/2008). In the study, DC-specific virus was able to specifically transduce the gene of interest (ovalbumin for immune response assays and GFP for tracing studies) *in vitro*—in mouse and human cells expressing DC-SIGN—and *in vivo*, where approximately 3% of DCs were shown to express the transgene delivered by the viral expression system; moreover, DC-targeted expression of the antigen-induced robust antigen-specific T-cell expansion (CTL response). Overall, we believe these preclinical, proof-of-concept studies are well conducted, particularly being from the laboratory of Dr. David Baltimore at CalTech), and they give us confidence that the scientific basis of IMDZ ZVex platform technology is strong.

GLAAS – Amplifying The Immune Response

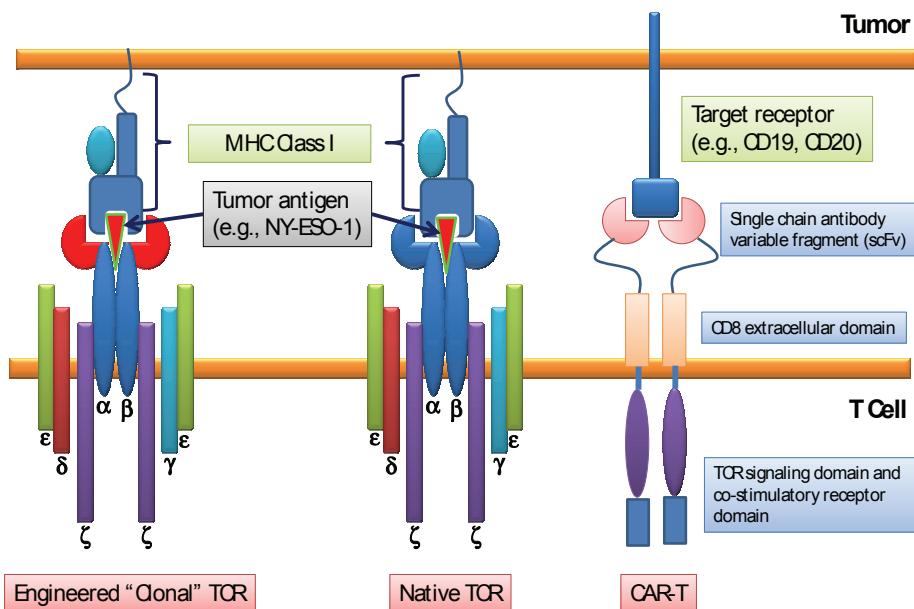
The role of the second component of Immune Design's technology platform, GLAAS, is to boost the immune response fostered by ZVex. GLAAS, short for Glucopyranosy Lipid A (GLA) Adjuvant Systems, is aimed at using GLA, a synthetic mimic of an immune-stimulating bacterial cell wall substance, to bind to TLR4 receptors on the surface of, and activate, dendritic cells. Although the GLAAS system is also being tested on its own to boost the immune response against endogenous tumor antigens (discussed further on), in the context of the ZVex platform, it is planned to be studied in combination with a tumor-specific antigen and ZVex to generate CD4 T-cells, further expand cytotoxic T-lymphocytes, and up-regulate the anti-tumor immune response primed by ZVex.

Comparing and Contrasting IMDZ's Platform versus CAR-T

Though Immune Design's technology has not yet been proven effective in humans, we believe that work in platforms such as CAR-T has validated the concept of activating T cells against specific cancer antigens. ZVex is one of several approaches being used in cancer cellular immunotherapy. One approach that has garnered much excitement of late is chimeric antigen receptor T cells (CAR-T) technology. While there are important differences between CAR-T and ZVex, one commonality is that in both systems, maximal CTL activity against the tumor antigen is desired. Thus far, in both pediatric and adult studies of B-cell malignancies, CAR-T resulted in very high rates of complete responses, with long durations of response. For instance, in an NCI-conducted, relapsed/refractory DLBCL study using a CAR-T cell therapy against CD19, a protein on B-cell malignancies, the response rate was 86%, with 16 of 24 patients achieving remission. While both the technology and the tumor antigen/target differ from Immune Design's approach, we believe this provides some indirect validation for the potential effectiveness of specific cytotoxic T-lymphocyte generation against cancers.

Beyond these conceptual similarities, we see notable differences between Immune Design's ZVex and CAR-T technology, some of which could potentially confer theoretical advantages for ZVex. Whereas ZVex achieves generation of CTLs specific to tumor antigens through gene therapy-mediated targeting of DC cells and physiological antigen presentation, CAR-T bypasses the need for DC-mediated priming by directly genetically engineering *ex vivo* such CD8+ CTLs versus the target of interest (see Exhibit 5).

Exhibit 5. Schema of different T cell immunotherapies



Source: Wells Fargo Securities, LLC

One key difference is that CAR-T is only limited to protein targets expressed on the cell surface (e.g., CD19, CD20), and because the target is a cell surface protein that may be expressed in both normal and diseased tissues, it could have more theoretical safety issues. ZVex may also be more versatile in that it is able to target both intra- and extracellular targets, as well as being able to use tumor antigen targets that are most well characterized and validated, such as NY-ESO-1, an “off the shelf” tumor antigen applicable to many different cancer types and patients. An important limitation of the CAR-T platform is that the chimeric receptor is permanently transduced into T cells such that it persists throughout the life of the T cells (i.e., no off switch), something that may have contributed to observed serious toxicities in some patients. In contrast, ZVex targets the more physiologic mechanisms to stimulate the immune system (and the tumor antigen expression is only transient), which could theoretically reduce safety concerns, though this would need to be borne out clinically. On the other hand, CAR-T technology has been more directly validated with clinical data, and the high numbers of activated T cells that can be infused using that system – which contain domains that themselves “supercharge” the potential response to antigens – could produce more rapid onset of activity, particularly

against hematologic cancers. A comparison of ZVex with other modern T-cell targeting technologies follows (see Exhibit 6):

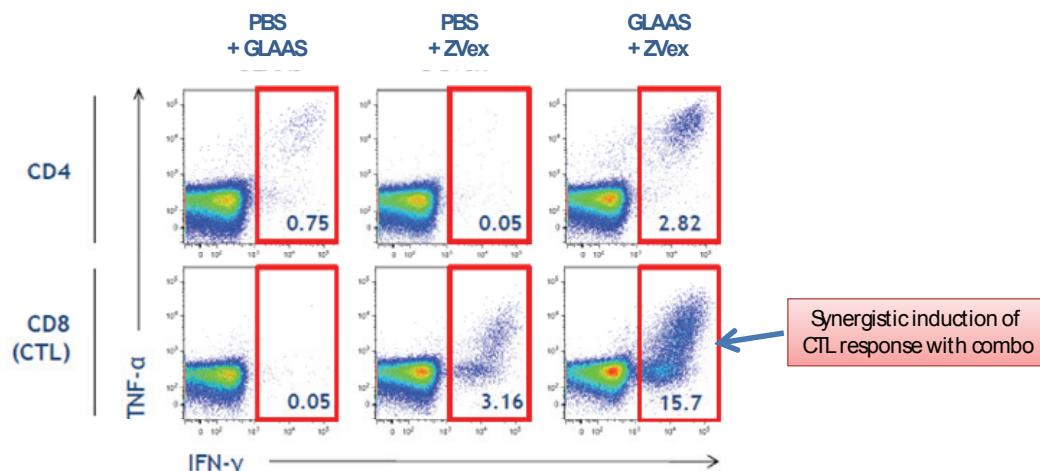
Exhibit 6. Comparison of different anti-tumor CTL-inducing platform technologies

	ZVex	CAR-T	Engineered T cells
Description	In vivo transduced dendritic cell-mediated activation of native T cells (CTLs); uses "off-the-shelf" tumor antigens (e.g., NY-ESO-1)	Personalized, adoptive transfer of monoclonal T cells expressing chimeric T cell receptor against cell surface receptor target (e.g., CD19, CD20); does not require DC-mediated priming	Personalized, adoptive transfer of monoclonal T cells expressing recombinant T cell receptor against MHC Class I-associated tumor antigen (e.g., NY-ESO-1); does not require DC-mediated priming
Delivery	In vivo transduction of DC-targeted lentivirus using antigen of choice	Ex-vivo transduction of T cells with high affinity antibody fragment chimeric receptor against target of interest	Ex-vivo transduction of T cells with monoclonal recombinant TCR against target of interest
Targets	Solid tumors, possibly hematologic cancers	Hematologic malignancies; toxicities observed in solid tumors	Hematologic and solid tumors
Efficacy	Strong preclinical data with strong CTL response and anti-tumor activity; activity not yet validated in humans	Impressive activity in B cell malignancies and neuroblastoma	Positive response in melanoma and synovial cell carcinoma
Safety	Clean safety profile in preclinical settings; safety not yet validated in humans	Potential for severe immune related toxicities, including death	Potential for severe immune related toxicities

Source: Company reports and Wells Fargo Securities, LLC

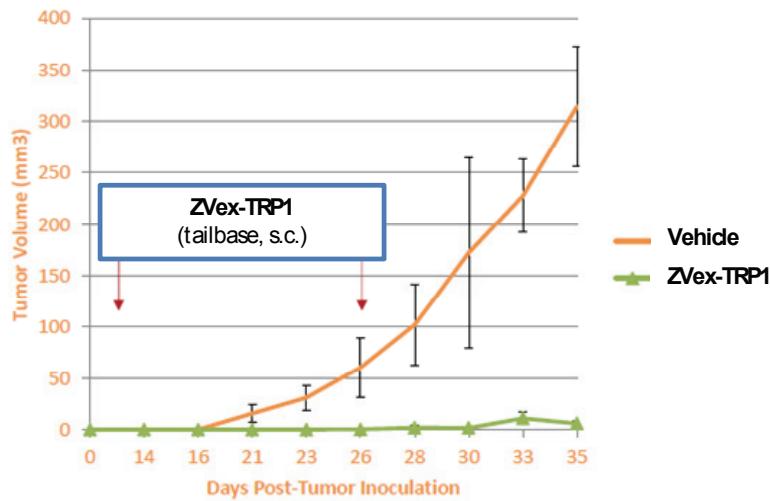
Preclinical Data

In an animal system for assessing immune response, ZVex was shown to generate cytotoxic T-lymphocytes against tumors expressing the targeted antigen, supporting the concept. The preclinical mouse model is a well-established immune challenge/response model wherein the animals were treated subcutaneously with ZVex containing the tumor antigen of interest, and subsequent immune response was characterized by surveying different immune cells and cytokines induced by the treatment. As the central goal of immunotherapy has been to induce robust CTL response—which closely mirrors the cancer-killing potential—the magnitude of CTL response has been used as a surrogate for potential efficacy of such immunotherapy. CTL response is characterized by CD8+ T cells actively expressing IFNgamma, which is a cytokine associated with activated CD8 T cells that are capable of recognizing and killing tumors (or any other cells) that express the specific antigen that they were “instructed” by DC to kill, in this case through ZVex mediated priming of tumor-antigen specific CD8+ T cells. As shown in Exhibit 7, animals treated with ZVex induced a 50-fold increase in activated CD8+ T cells compared to GLAAS-treated animals (which are not expected to induce CTL response alone), and a 300-fold increase when treated with both agents, suggesting that GLAAS boosts a ZVex-induced CTL response.

Exhibit 7. Substantial antigen-specific T-cell responses observed in animal model

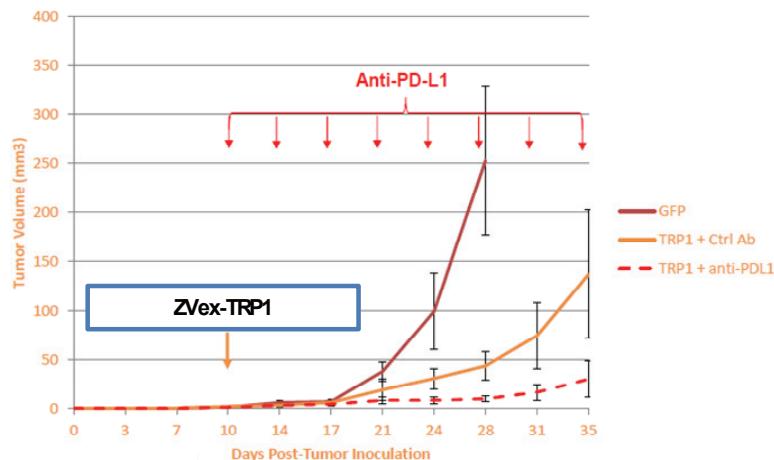
Source: Company reports

Though animal models have their limitations, the ZVex approach has shown tumor inhibition in several systems, which we believe provides some proof of principle. With the caveat that preclinical cancer models often do not translate to human clinical experience, the ZVex system has shown activity in several tumor treatment models with different tumor antigens, which we believe strengthens the validity of the approach, short of having human clinical data. The main mouse tumor model utilized was the B16 melanoma model, one of the most widely used tumor/metastasis rodent models and one that models a particularly aggressive tumor form. Upon treatment with ZVex containing TRP1, a well-characterized melanocyte specific antigen (self-antigen), the treatment arm showed complete suppression of tumor growth compared with rapidly growing tumor volume in the control animals (see Exhibit 8).

Exhibit 8. Tumor growth inhibition observed in B16 mouse melanoma model lends support for activity of vector

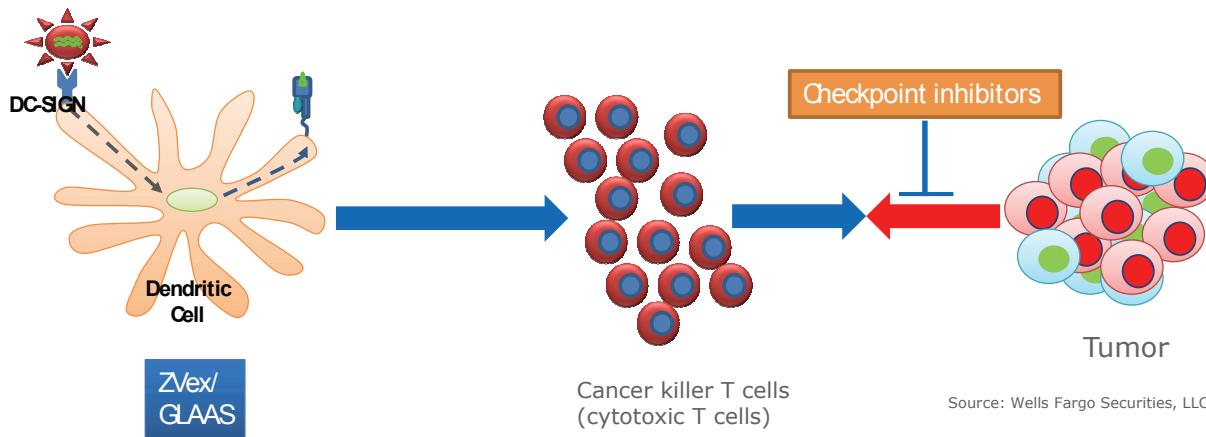
Source: Company reports

Additionally, when a similar experiment was conducted to evaluate the combination effect with a well-known agent with strong activity in melanoma, anti-PD-L1, significant synergy was observed (see Exhibit 9).

Exhibit 9. Synergy observed with anti-PD-L1 agent in animal model

Source: Company reports

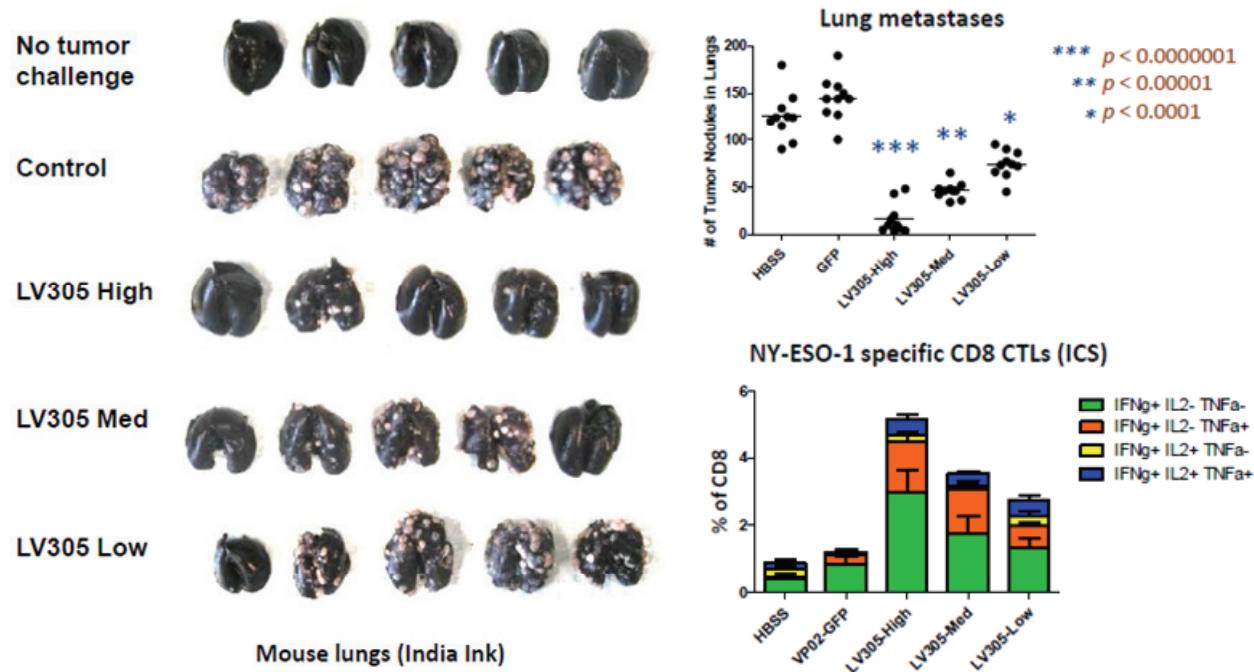
There is a strong scientific rationale, in our view, for potentially combining ZVex and checkpoint inhibitors (e.g., anti-PD-1, anti-PD-L-1). The concept of immunotherapy is premised on the idea that the host immune system mounts a challenge against the tumor (offense) and/or inhibiting the tumor's ability to prevent/neutralize host's immune response (defense). It is likely that optimizing the CTL response through ZVex (or monoclonal TCR or CAR-T) and preventing the tumor's defense against the host immune system (checkpoint inhibitors) will likely result in a synergistic effect on anti-tumor effect (see Exhibit 10).

Exhibit 10. Illustration of theoretical synergy between ZVex/GLAAS and checkpoint inhibitors

Source: Wells Fargo Securities, LLC

In another rodent model (CT-26 mouse colon cancer), using NY-ESO-1/ZVex, Immune Design further demonstrated that treatment with ZVex resulted in inhibition of tumor metastasis to the lung, in a dose-dependent manner. The anti-tumor/metastasis was correlated with NY-ESO-1-specific CTL responses, demonstrating that anticancer effects can occur using the NY-ESO-1 tumor antigen (see Exhibit 11):

Exhibit 11. Anti-metastasis activity in animal model supports potential for activity with NY-ESO-1 tumor antigen



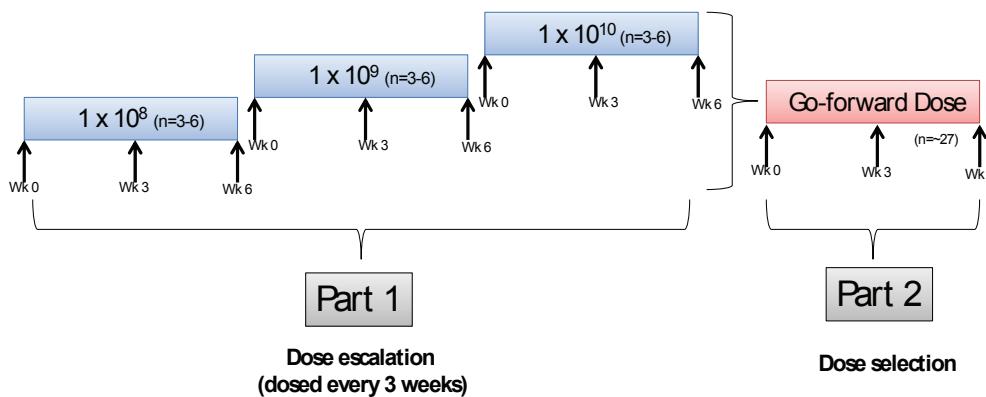
Source: Company reports and Wells Fargo Securities, LLC

The company has also performed other rodent studies using different cancer xenograft models targeting different antigens (e.g., ovalbumin in the E.G7 tumor model), which showed consistent anti-tumor activity associated with ZVex treatment. Together, we believe there is solid preclinical evidence for ZVex-mediated CTL response and anti-tumor potential.

Clinical Program

LV305

IMDZ is currently assessing its lead ZVex (LV305) in five solid tumors in ph.I. LV305 (for lentivirus 305) is a ZVex with a NY-ESO-1 payload, without a GLAAS boost. The phase I trial of LV305, which began in April 2014, is an open-label, multi-center, U.S.-based study designed to evaluate safety and immunogenicity of the drug in approximately 36 patients with advanced melanoma, NSCLC, ovarian cancer, sarcoma, or breast cancer expressing NY-ESO-1. The study is designed for dose-finding with a traditional 3+3 dose escalation design, starting with 1×10^8 viral particles administered intradermally every three weeks and then two higher cohorts, followed by a second part consisting of dose expansion at the highest, safest dose in advanced melanoma, NSCLC, ovarian cancer, or sarcoma (see Exhibit 12).

Exhibit 12. Design of ongoing phase I ZVex dose escalation study in advanced solid tumors

Source: Company reports, www.clinicaltrials.gov, and Wells Fargo Securities, LLC

We believe the tumor antigen being used in trial, NY-ESO-1, is validated as an immunotherapy target. In addition to the animal data described above in which NY-ESO-1 dendritic cell transfection reduced tumor metastases in the CT-26 mouse cancer model, NY-ESO-1-based immunotherapies have also shown activity in humans using other systems. In a study in which patients' T-cells were genetically engineered to express a T-cell receptor specific for MHC I presenting the NY-ESO-1 peptide, then transferred back to the patients, it was shown that objective clinical response was observed in 67% of synovial cell carcinoma patients (4/6) and 45% of melanoma patients (5/11), whose tumors expressed high levels of NY-ESO-1. We believe these results provide clinical rationale for several of the cancer indications that IMDZ is currently pursuing, and the concept that generating NY-ESO-1 specific T-cells against high NY-ESO-1-expressing tumors can be efficacious. Additional supporting evidence using the “traditional” vaccine approach—pulsed with tumor peptide—also showed hints of efficacy in ovarian cancer when treated with chemotherapy. Separately, melanoma patients treated with ipilimumab had a greater clinical response when there was a concomitant immune response against NY-ESO-1. More than half of patients who responded to ipilimumab showed immune responses against NY-ESO-1, suggesting that the CTL response mediated by NY-ESO-1 could synergize with ipilimumab’s anti-immunosuppressive effects.

Though not all patients’ tumors will express NY-ESO-1 such as to be eligible for treatment with this ZVex, LV305, we believe the approach should be applicable to a reasonably broad set of cancers. NY-ESO-1 is reported to be expressed in many tumor types: 10-70% of metastatic melanomas, breast, prostate, thyroid, and ovarian cancers, and most notably, very highly in synovial sarcomas and myxoid round cell liposarcoma (80%) (see Exhibit 13):

Exhibit 13. Prevalence of NY-ESO-1 expression in tumors under exploration ranges from 12% to 30%, up to 80+%

Tumor type	NY-ESO-1 expression
Synovial sarcoma / myxoid round cell liposarcoma	~80%+
Melanoma	50-70%
Ovarian cancer	40-60%
NSCLC	12-30%

Source: Company reports, Journal of Clinical Oncology, vol. 29 no. 7, 2011

On the safety side, while theoretical risk exists, we believe there is sufficient scientific data to support that NY-ESO-1-mediated immunotherapy will unlikely cause a significant autoimmune response. NY-ESO-1 belongs to a type of tumor antigens called “cancer-testis antigens” wherein the antigen expression is limited to the testis in a normal individual, but expressed frequently and at high levels in tumors (the physiological relevance is unclear). While there might be theoretical safety concerns related to LV305 potentially eliciting an immune response against testis tissues that express the target antigen, the likelihood of such an adverse event is low given that testis does not express MHC I, a required component for T cells to mount a CTL attack, as well as the fact that testis is an “immune privileged” organ (like the brain, eyes, and

certain other vital organs) that can avoid inflammatory immune responses. The level of NY-ESO-1 expression is also substantially lower than in tumors. Another theoretical risk is immunogenicity to the viral envelope upon repeat administration, something worth watching, in our view. Reassuringly, though, Immune Design has tested up to four doses in animals, and while antibodies to the envelope do appear to develop after the third dose, these have not appeared to be functionally neutralizing.

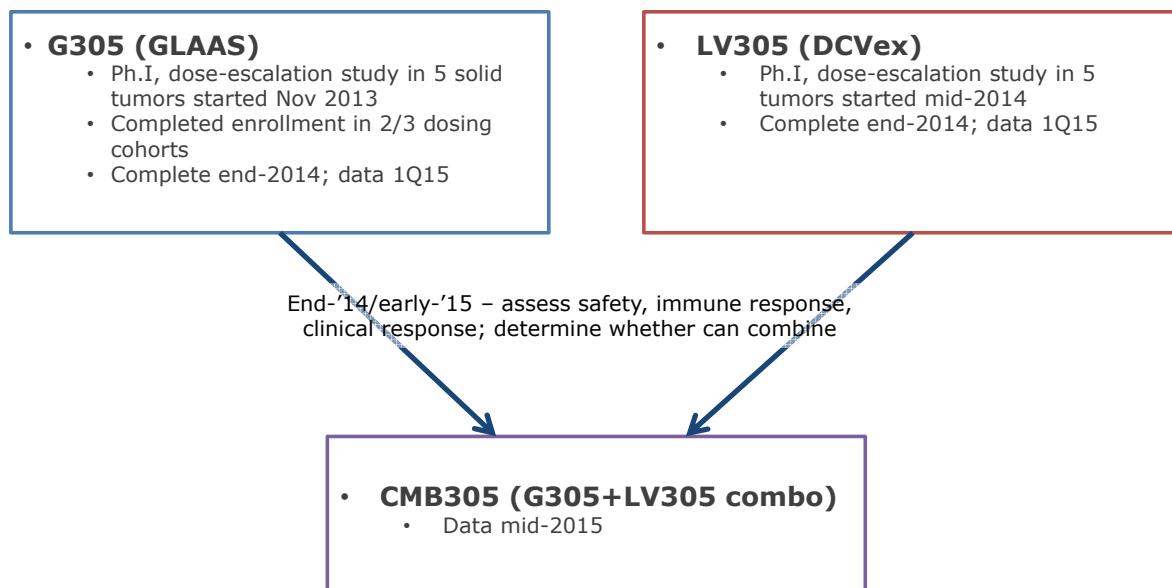
Thus far, the trial appears to be proceeding as planned, and initial data could be reported in Q1 2015; we would look for immunogenicity data to signal potential activity. We believe the first cohort of the LV304 phase I has already been completed and enrollment is continuing. Top-line data on safety and immunogenicity are expected by Q1 2015. While the exact measures may be difficult to interpret, both because of the small study size and patient heterogeneity, as well as the evolving understanding of what immune endpoints would be predictive of benefits, we believe the data should provide a general sense of whether desired antigen-specific immune upregulation is taking place. Immune Design plans to use T-cell proliferation assays with the NY-ESO-1 antigen, wherein a two-fold induction from baseline might indicate activity of ZVex. This also includes phenotyping of T cells (Tregs, subsets of memory T cells that could be reactive to antigen), with any significant increase in the proportion of CD8 T-cells that are antigen specific (for instance, from 0.1% to 1.0-3.0%), also indicative of a specific immune-stimulating effect. Immune design also plans to examine levels of functionality of such T-cells using genetic and cytotoxicity analyses, though there are no clear benchmarks for results at this point.

G305

In parallel, IMDZ is conducting a phase I study of TLR4 agonist G305 (GLA + NY-ESO-1 antigen), the idea being to combine it with LV305 in the future. G305 consists of TLR4 agonist and immune stimulator GLA, administered in combination with recombinant full-length NY-ESO-1 protein as an intramuscular injection, the idea being to up-regulate the immune system and generate an immune response to the tumor antigen. NY-ESO-1-based cancer vaccines, consisting of the tumor antigen plus an adjuvant (ISCOMATRIX, MPLA), have been tested by others in the past, with varying degrees of effectiveness. We believe the goal of this study would be to show safety and perhaps some immunogenicity, but the differentiating feature would be G305's potential to be combined with LV305; we do not expect Immune Design to develop G305 on its own. The phase I study, which began in November 2013, is testing three escalating doses, beginning at 2.5mcg, in cohorts of 3-6 patients with the same tumor types as in the phase I LV305 study – advanced breast cancer, melanoma, NSCLC, ovarian cancer, or sarcoma expressing NY-ESO-1. Initial data are expected in Q1 2015.

CMB305

If both LV305 and G305 are safe, IMDZ plans to combine them in a “prime boost” approach study starting next year termed “CMB305.” Given the complementary mechanisms and substantial synergy observed preclinically between the ZVex and GLAAS approaches, Immune Design plans to combine the two drugs for additional phase I work, the idea being to optimally boost the population of tumor antigen-specific cytotoxic T-lymphocytes. The company has completed a toxicology package for the combination and is submitting it to the FDA for IND filing by the end of September 2014. We would expect the phase I trial to start sometime by the end of 2014, and to enroll patients with NY-ESO-1 expressing advanced cancers, with the specific tumor types likely depending on activity observed in the LV305 phase I (and possibly the G305 phase I, as well). We believe Immune Design may choose to focus on synovial sarcoma, given the high rate of NY-ESO-1 positivity among patients with this tumor type and the small population, which may facilitate a more rapid orphan-like development plan, as well as on a more broad indication, such as NSCLC or ovarian cancer. Though exact dosing would await completion of the phase I work for each component, our sense is that Immune Design may alternate doses of LV305 with G305, to try and initially trigger the tumor-specific CTLs, then boost them, though one risk is that the optimal administration patterns to maximize and enhance the specific immune responses are not fully known.

Exhibit 14. Summary of time lines and potential trial designs for LV305, G305, and CMB305

Source: Company reports and Wells Fargo Securities, LLC

Market opportunity

We believe the market opportunity for CMB305, if it is successful in at least three solid tumor indications, could be **\$1.3B several years into launch**. Our assumption is that in soft-tissue sarcomas (synovial sarcoma and myxoid round cell liposarcoma), Immune Design could potentially have a more rapid path to approval, given that these are orphan indications and bring CMB305 (or just LV305) to market in these diseases – which have a high (80%+) NY-ESO-1 antigen prevalence – by 2018 in a favorable scenario. However, these indications are relatively small; we estimate approximately \$150M in worldwide sales by 2023. In larger cancers like NSCLC and ovarian cancer, we believe a more traditional full development path would be required, with the agent potentially reaching the market in 2020. We assume pricing across all indications of \$13,000 per month in today's dollars, increasing marginally each year for a launch price of \$15,200/cycle and with a lower price in Europe. We assume approximately 10-11 cycles of therapy in soft-tissue sarcomas, with fewer average months of therapy for NSCLC and ovarian cancer patients given the more rapid progression of refractory patients. We assume the drug is initially used in later-line patients with NY-ESO-1 positivity, with what we believe are reasonable penetrations in eligible refractory patients in the larger cancers, where there are more options, 15-20% by 2023, and slightly higher in soft-tissue sarcomas. Based on our discussions with Immune Design, we believe the company's strategy will be to retain U.S. rights; as such, we estimate that the company assumes the costs for launching the drug in the U.S. sales and recognized U.S. revenue, and that the company receives royalties of about 20% on European sales of CMB305, for out-year 2023 revenue of \$860MM. Success in additional tumor types could potentially lead to upside to our revenue estimates.

Exhibit 15. Revenue build for CMB305 in several solid tumor indications

Soft tissue sarcomas (synovial sarcoma / myxoid round cell liposarcoma)

		2018E	2019E	2020E	2021E	2022E	2023E
United States	U.S. population	322,292,786	325,193,421	328,120,162	331,073,244	334,052,903	337,059,379
	Number of pts with SS/MRCL	1,289	1,301	1,312	1,324	1,336	1,348
	Patients with NY-ESO-1 antigen (80%)	1,031	1,041	1,050	1,059	1,069	1,079
	CMB305 penetration	5%	15%	20%	24.0%	28%	31.00%
	Price per month (cycle)	\$15,208	\$15,816	\$16,449	\$17,107	\$17,791	\$18,503
	# of cycles	10.4	10.5	10.6	10.7	10.8	10.9
Revenues		\$8,156,064	\$25,922,826	\$36,615,262	\$46,542,082	\$57,511,816	\$67,435,408
Europe	E.U. population (Major EU countries)	537,154,644	541,989,036	546,866,937	551,788,739	556,754,838	561,765,632
	Number of pts with SS/MRCL	2,149	2,168	2,187	2,207	2,227	2,247
	Patients with NY-ESO-1 antigen (80%)	1,719	1,734	1,750	1,766	1,782	1,798
	CMB305 penetration	0%	5%	15%	20%	24%	28%
	Price per month (cycle)	\$12,167	\$12,653	\$13,159	\$13,686	\$14,233	\$14,802
	# of cycles	10.4	10.5	10.6	10.7	10.8	10.9
Revenues		\$0	\$11,521,256	\$36,615,262	\$51,713,425	\$65,727,790	\$81,212,535
Total Revenues		\$8,156,064	\$37,444,081	\$73,230,524	\$98,255,507	\$123,239,606	\$148,647,943
Probability Adjusted Revenues (25%)		\$2,039,016	\$9,361,020	\$18,307,631	\$24,563,877	\$30,809,901	\$37,161,986
NSCLC							
United States	U.S. population	322,292,786	325,193,421	328,120,162	331,073,244	334,052,903	337,059,379
	Number of pts with NSCLC (incidence)	203,044	204,872	206,716	208,576	210,453	212,347
	Patients with NY-ESO-1 antigen (20%)	40,609	40,974	41,343	41,715	42,091	42,469
	NSCLC 2L with NY-ESO-1 antigen	24,365	24,585	24,806	25,029	25,254	25,482
	CMB305 penetration in 2L	0%	0%	2%	5%	9%	15%
	Price per month (cycle)	\$15,208	\$15,816	\$16,449	\$17,107	\$17,791	\$18,503
# of cycles		6.5	6.6	6.7	6.8	6.9	7
Revenues		\$0	\$0	\$54,676,776	\$145,579,936	\$279,022,170	\$478,561,393
Europe	E.U. population (Major EU countries)	537,154,644	541,989,036	546,866,937	551,788,739	556,754,838	561,765,632
	Number of pts with NSCLC (incidence)	338,407	341,453	344,526	347,627	350,756	353,912
	Patients with NY-ESO-1 antigen (20%)	67,681	68,291	68,905	69,525	70,151	70,782
	NSCLC 2L with NY-ESO-1 antigen	40,609	40,974	41,343	41,715	42,091	42,469
	CMB305 penetration in 2L	0%	0%	0%	1.00%	4.00%	7.00%
	Price per month (cycle)	\$12,167	\$12,653	\$13,159	\$13,686	\$14,233	\$14,802
# of cycles		6.5	6.6	6.7	6.8	6.9	7
Revenues		\$0	\$0	\$0	\$38,821,316	\$165,346,471	\$308,039,517
Total Revenues		\$0	\$0	\$54,676,776	\$184,401,252	\$444,368,642	\$786,600,911
Probability Adjusted Revenues (20%)		\$0	\$0	\$10,935,355	\$36,880,250	\$88,873,728	\$157,320,182
Ovarian cancer							
United States	U.S. population	322,292,786	325,193,421	328,120,162	331,073,244	334,052,903	337,059,379
	Number of pts with ovarian cancer (incident)	22,560	22,764	22,968	23,175	23,384	23,594
	Patients with NY-ESO-1 antigen (45%)	10,152	10,244	10,336	10,429	10,523	10,617
	2nd+ line platinum refractory	7,614	7,683	7,752	7,822	7,892	7,963
	CMB305 penetration in 2L+	0%	0%	5%	10%	15%	20%
	Price per month (cycle)	\$15,208	\$15,816	\$16,449	\$17,107	\$17,791	\$18,503
# of cycles		6.5	6.6	6.7	6.8	6.9	7
Revenues		\$0	\$0	\$42,716,231	\$90,987,460	\$145,324,047	\$206,276,463
Europe	E.U. population (Major EU countries)	537,154,644	541,989,036	546,866,937	551,788,739	556,754,838	561,765,632
	Number of pts with ovarian cancer (incident)	45,658	46,069	46,484	46,902	47,324	47,750
	Patients with NY-ESO-1 antigen (45%)	20,546	20,731	20,918	21,106	21,296	21,488
	2nd+ line platinum refractory	15,410	15,548	15,688	15,829	15,972	16,116
	CMB305 penetration in 2L+	0%	0%	0%	1.00%	4.00%	9.00%
	Price per month (cycle)	\$12,167	\$12,653	\$13,159	\$13,686	\$14,233	\$14,802
# of cycles		6.5	6.6	6.7	6.8	6.9	7
Revenues		\$0	\$0	\$0	\$14,731,303	\$62,743,081	\$150,287,137
Total Revenues		\$0	\$0	\$42,716,231	\$105,718,763	\$208,067,128	\$356,563,600
Probability Adjusted Revenues (20%)		\$0	\$0	\$8,543,246	\$21,143,753	\$41,613,426	\$71,312,720
U.S. sales of CMB305		\$8,156,064	\$25,922,826	\$134,008,269	\$283,109,478	\$481,858,033	\$752,273,264
EU sales of CMB305		\$0	\$11,521,256	\$36,615,262	\$105,266,044	\$293,817,342	\$539,539,189
Total CMB305 revenues		\$8,156,064	\$37,444,081	\$170,623,531	\$388,375,522	\$775,675,375	\$1,291,812,453
Total prob-adjusted CMB305 revenues		\$2,039,016	\$9,361,020	\$37,786,232	\$82,587,880	\$161,297,055	\$265,794,888

Source: Wells Fargo Securities, LLC estimates

G100

Early anecdotal clinical response provides rationale for the “endogenous antigen” approach consisting of an intra-tumoral injection of GLAAS to induce an immune response against Merkel cell carcinoma. G100 is the simplest approach among IMDZ’s immunotherapies, based on the concept that GLAAS, as a potent immune stimulator, could serve as an “adjuvant” to tumor-derived antigens, thereby inducing the immune system to kill the tumor. There is no tumor antigen co-administered in this regimen. Instead, it is the tumor itself that provides the antigens, and in the presence of GLAAS, it could become immunogenic and more susceptible to the patient’s cancer immunity. While the incidence of Merkel cell carcinoma is low, at around 1,500 cases in the United States per year, as a locally accessible cancer associated with immunosuppression, it provides a very rational testing ground for the concept of GLAAS-induced immunostimulation against tumor antigens. Encouragingly, the first patient achieved a complete response following two doses of G100, though three additional treated patients have not had a response. The study is ongoing and is expected to finish by Q1 2015. An additional study in combination with local radiation in the second cycle is being considered, as are other malignancies, such as NHL. At this stage, we do not ascribe significant value to the program, relative to the ZVex-based programs.

Partnerships

IMDZ has several partnerships with a number of biopharmaceutical companies from outlicensing of GLAAS technology in the infectious disease space. In 2010, the company entered into a license agreement with MedImmune/AstraZeneca, granting AZN the rights to develop and commercialize GLAAS as adjuvant in vaccines for three different infectious disease indications. IMDZ received \$4.5MM in up-front payment and is eligible for a mid-single-digit royalty and milestone payments (\$62.9–76.0MM). MEDI7510, which we believe is the first of the potential candidates stemming from this agreement to enter the clinic, is an RSV vaccine that moved into phase I in April 2014. In 2011, Immune Design licensed GLAAS to Sanofi for allergy and recently expanded the scope of the agreement (i.e., on August 7, 2014) to cover certain food allergies. IMDZ has received an undisclosed up-front payment and is eligible for up to \$168MM in milestones, as well as tiered royalties. In 2013, IMDZ granted Medicago a non-exclusive license to develop pandemic flu vaccines based on GLAAS technology, in exchange for \$0.5 million up-front potential developmental milestones of \$9.5MM, and mid-single-digit royalties for a certain time period. Medicago’s pandemic flu vaccine is currently in phase I. G103, for HSV-2 infection, remains unpartnered.

Intellectual Property / Legal

Intellectual property. ZVex technology was licensed from the California Institute of Technology. Patents on the platform include methods of generating lentiviral vector to target dendritic cells. They also have the composition of matter patents on the lentiviral vector and packaging cells for manufacturing the virus. The original “CalTech” family of ZVex patents and application encompass the method of targeting DC via lentiviral vectors containing specific cell tropic envelop proteins. These patents and application have expiration dates of approximately 2027-2031. Additional patents on improvements and modifications of ZVex have been filed/issued to IMDZ. We note that expiration dates are estimated based on the filing date and not the priority date.

Exhibit 16. CalTech ZVex IP portfolio exclusively licensed to IMDZ

Publication Date	Publication Number	Title	Est. exp.
9/25/2012	US8273345	Targeted gene delivery to dendritic cells	2031
12/11/2012	US8329162	Targeted gene delivery for dendritic cell vaccination	2027
1/24/2008	US20080019998A1	TARGETED GENE DELIVERY FOR DENDRITIC CELL VACCINATION	2027
5/13/2010	US20100120122A1	TARGETED GENE DELIVERY FOR DENDRITIC CELL VACCINATION	2030
5/13/2010	US20100120140A1	TARGETED GENE DELIVERY FOR DENDRITIC CELL VACCINATION	2030
9/1/2011	US20110212530A1	METHOD OF TARGETED GENE DELIVERY USING VIRAL VECTORS	2031
3/22/2012	US20120070462A1	Targeted Gene Delivery to Dendritic Cells	2031

Source: USPTO, company reports, and Wells Fargo Securities estimates, LLC

There are additional patents in-licensed from the University of North Carolina on various modified retroviral vectors and GLAAS technology was in-licensed from the Infectious Disease Research Institute (IDRI).

Exhibit 17. IMDZ IP on ZVex, GLAAS, and other pipeline products

Publication Date	Publication Number	Title	Abstract	Est. exp.	Comments
5/29/2012	US8187872	Lentiviral vectors pseudotyped with a sindbis virus envelope glycoprotein	Lentiviral vector particles comprising a Sindbis virus E2 glycoprotein variant and a lentiviral vector genome comprising a sequence of interest are provided. A lentiviral vector particle comprising: (a) an envelope comprising a Sindbis virus E2 glycoprotein variant; and (b) a lentiviral vector genome comprising a sequence of interest; wherein the E2 glycoprotein variant facilitates infection of dendritic cells by the lentiviral vector particle, and wherein the E2 glycoprotein variant has reduced binding to heparan sulfate compared to a reference sequence (HR strain).	2031	Method and composition of viral vectors comprising of alphavirus envelop protein that has a natural tropism for DC; method of delivering tumor antigens to DC
12/4/2012	US8323662	Methods useful for generating highly mannosylated pseudotyped lentiviral vector particles comprising a Vpx protein	Materials and methods useful for generating highly mannosylated pseudotyped lentiviral vector particles comprising a Vpx protein are provided. More specifically, methods for generating such materials include culturing in a culture medium including kifunensine a virus packaging cell with a lentiviral vector genome including a polynucleotide encoding an exogenous antigen, a polynucleotide encoding a Sindbis E2 glycoprotein that preferentially binds dendritic cells expressing DC-SIGN, and a polynucleotide encoding a Vpx protein or a Vpr protein that retains SAM HD1-inhibiting activity, followed by isolating a pseudotyped lentiviral vector particle that preferentially binds dendritic cells expressing DC-SIGN.	2032	Method of manufacturing lentivirus using cell culture system
3/17/2011	US20110064763A1	LENTIVIRAL VECTORS PSEUDOTYPED WITH A SINDBIS VIRUS ENVELOPE GLYCOPROTEIN	Lentiviral vector particles comprising a Sindbis virus E2 glycoprotein variant and a lentiviral vector genome comprising a sequence of interest are provided. A lentiviral vector particle comprising: (a) an envelope comprising a Sindbis virus E2 glycoprotein variant; and (b) a lentiviral vector genome comprising a sequence of interest; wherein the E2 glycoprotein variant facilitates infection of dendritic cells by the lentiviral vector particle, and wherein the E2 glycoprotein variant has reduced binding to heparan sulfate compared to a reference sequence (HR strain).	2030	Method and composition of viral vectors that target DC and packaging cell lines for manufacturing virus
12/15/2011	US20110305748A1	Vaccines for Pandemic Influenza	Pharmaceutical and vaccine compositions comprise recombinant hemagglutinin from a pre-pandemic or pandemic influenza virus and an adjuvant comprising GLA. A particularly relevant pre-pandemic influenza virus is H5N1. Kits and methods of using the compositions are also provided.	2031	Composition and method of making flu vaccine using GLA as adjuvant; likely related to technology licensed to Mitsubishi Tanabe for developing vaccine for pandemic influenza
2/16/2012	US20120039932A1	Lentiviral Vectors Pseudotyped with a Sindbis Virus Envelope Glycoprotein	Lentiviral vector particles comprising a Sindbis virus E2 glycoprotein variant and a lentiviral vector genome comprising a sequence of interest are provided. A lentiviral vector particle comprising: (a) an envelope comprising a Sindbis virus E2 glycoprotein variant; and (b) a lentiviral vector genome comprising a sequence of interest; wherein the E2 glycoprotein variant facilitates infection of dendritic cells by the lentiviral vector particle, and wherein the E2 glycoprotein variant has reduced binding to heparan sulfate compared to a reference sequence (HR strain).	2031	Method and composition of viral vectors comprising of alphavirus envelop protein that has a natural tropism for DC; method of delivering tumor antigens to DC
10/18/2012	US20120263754A1	Methods for Enhancing Immunogen Specific Immune Responses by Vectored Vaccines	Provided herein are methods for inducing a specific immune response in a subject by administering to the subject an immunogenic composition comprising a recombinant expression vector, or a vector particle comprising the recombinant expression vector, which vector comprises a polynucleotide sequence that encodes an immunogen of interest. The methods further comprise administering an adjuvant composition either concurrently or sequentially with the immunogenic composition.	2032	Method of boosting immune response of ZVex, likely related to prime-boost method
11/15/2012	US20120288515A1	SYNTHETIC LONG PEPTIDE (SLP)-BASED VACCINES	Synthetic long peptides and an adjuvant are formulated together and administered to a subject in order to raise an immune response. In certain embodiments, the adjuvant is GLA.	2032	Vaccine composition and method of using the vaccine in conjunction with GLA
12/27/2012	US20120328655A1	IMMUNOGENIC COMPOSITIONS AND METHODS OF USING THE COMPOSITIONS FOR INDUCING HUMORAL AND CELLULAR IMMUNE RESPONSES	Compositions and methods are provided herein for improved dual immunization strategies that induce in a subject an immune response that includes a humoral immune response and cellular immune response, both CD4 and CD8 T lymphocyte immune responses, thereby providing a complete adaptive immune response to one or more antigens. The methods described are therefore useful for treating and/or preventing (i.e., reducing the likelihood or risk of occurrence) different diseases, disorders, and conditions such as cancers and infectious diseases for which induction of both a humoral immune response and cellular immune response is desired and beneficial.	2032	Method of boosting immune response of ZVex, likely related to prime-boost method
3/27/2014	US20140086947A1	VACCINES FOR HSV-2	Compositions of recombinant HSV-2 proteins and an agonist of the innate immune system, such as an adjuvant, are provided as a vaccine. Proteins include an envelope glycoprotein and a structural protein other than an envelope glycoprotein, e.g., a capsid or tegument protein. The vaccine is for use in either HSV-2 sero positive or sero negative subjects.	2033	HSV-2 vaccine using GLA (G103)
5/8/2014	US20140127247A1	VACCINES FOR HSV-2	Compositions of recombinant HSV-2 proteins and an agonist of the innate immune system, such as an adjuvant, are provided as a vaccine. Proteins include an envelope glycoprotein and a structural protein other than an envelope glycoprotein, e.g., a capsid or tegument protein. The vaccine is for use in either HSV-2 sero positive or sero negative subjects.	2033	HSV-2 vaccine using GLA (G103)

Source: USPTO, company reports, and Wells Fargo Securities estimates, LLC

Legal Proceedings: On July 25, 2014, IMDZ announced that a French company, Theravectys, has filed a lawsuit against IMDZ related to an alleged interference with the contractual relationship with a Belgian company Henogen, with which Theravectys apparently had an exclusive relationship to manufacture Theravectys' lentivirus-based therapeutic vaccine against HIV. The lawsuit was filed in the Delaware Court of Chancery. This recent lawsuit is related to the previous lawsuit filed by Theravectys against IMDZ in 2013, filed in the Federal District Court of Delaware, which Theravectys voluntarily dismissed in April 2014 without further pursuing the case (refer to IMDZ's S-1 filing). The complaints filed for both cases are virtually identical—interference with contractual relations, misappropriation of trade secrets, unfair competition, and unjust enrichment claim was added to the newly filed case—likely in reference to IMDZ's IPO. Though we are not lawyers, after speaking with Immune Design, we are reassured that there was no intentional interference with Theravectys' and Henogen's exclusivity agreement. The viral vector being used by Theravectys is different than that of IMDZ, and the manufacturing protocol was devised by IMDZ -- Henogen was hired as only a contract manufacturer. It is unclear whether Theravectys actually will prosecute this case, but IMDZ indicated that it will vigorously defend against the lawsuit. IMDZ no longer contracts with Henogen for manufacturing and has established a U.S.-based manufacturer.

Financials

Exhibit 18. Simplified Balance Sheet (Pro Forma, as of March 31, 2014, estimated)

Assets	
Cash and cash equivalents	\$78,262
Other assets	\$814
Total assets	\$79,076
Liabilities and Stockholders' Equity	
Current Liabilities	\$2,172
Deferred Revenue	\$0
Other liabilities	\$87
Stockholders' equity	\$76,817
Total liabilities and stockholders' equity	\$79,076

(in thousands of dollars)

Source: Company Reports and Wells Fargo Securities, LLC

Exhibit 19. Income Statement

	2012A	2013A	1Q A	2QE	3QE	4QE	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E
<i>Revenues</i>																
Licensing revenues (1)	\$876	\$729	\$0	\$0	\$0	\$0	\$0	\$0	\$0	\$0	\$0	\$0	\$0	\$0	\$0	\$0
Product sales / royalties (2)	1,877	870	25	100	100	325	358	393	433	476	523	576	633	697	766	10,000
Other, net (3)	-	-	-	-	-	-	-	-	-	10,000	10,000	35,000	35,000	10,000	10,000	10,000
U.S. sales of CMB305 (prob-adjusted)	-	-	-	-	-	-	-	-	-	2,039	6,481	28,632	58,949	99,247	153,826	22,394
Total revenues	\$2,960	1,599	25	100	100	325	358	393	433	12,515	17,580	66,039	99,310	122,354	186,986	
<i>Expenses</i>																
Cost of products sold	1,518	669	14	75	75	75	239	268	295	324	683	1,429	5,013	9,317	15,410	23,649
Research and development	8,604	11,554	4,078	4,160	4,243	4,328	16,808	17,648	26,472	29,120	32,032	32,993	33,982	35,002	36,052	37,134
Selling, general and administrative	3,713	4,433	1,446	1,952	2,030	2,111	7,540	8,671	9,017	12,624	25,249	40,398	60,597	66,657	69,323	72,096
Total operating expenses	13,835	16,656	5,538	6,187	6,348	6,514	24,587	26,785	32,069	57,964	74,820	99,593	110,976	120,785	132,878	
<i>Operating income/(loss)</i>																
Interest and other income	(10,875)	(15,057)	(5,513)	(6,087)	(6,248)	(6,414)	(24,262)	(28,230)	(35,392)	(41,636)	(45,449)	(57,240)	(33,554)	(11,686)	1,569	54,108
Change in fair value of convertible preferred stock warrant	35	37	1	3	6	9	20	46	40	52	78	67	49	54	39	57
(Loss) income before benefit from income taxes	(10,840)	(15,976)	(8,223)	(6,083)	(6,242)	(6,405)	(26,953)	(26,183)	(35,351)	(41,584)	(45,370)	(57,173)	(33,504)	(11,612)	1,608	54,165
Benefit (expense) from income taxes																
Net (loss) income	(10,840)	(15,975)	(8,223)	(6,083)	(6,242)	(6,405)	(26,953)	(26,183)	(35,351)	(41,584)	(45,370)	(57,173)	(33,504)	(11,612)	1,608	54,165
<i>Earnings Per Share (GAAP)</i>																
Shares Outstanding (Basic)	356	7,008	10,139	10,139	11,906	15,839	12,006	16,239	19,139	22,039	22,439	26,239	26,639	27,039	27,439	
Shares Outstanding (Diluted)	356	7,008	-	-	-	-	-	-	-	-	-	-	-	-	-	

Source: Company reports and Wells Fargo Securities, LLC estimates

Note: 2014 quarterly EPS do not equal annual EPS due to calculation of EPS based on diluted vs. basic shares

(1) related to Medimmune collaboration

(2) Reflects sales of GLAAS to collaborators

(3) Includes amortization of potential upfront for ex-U.S. CME305 partnership, potential milestones

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IMDZ: Risks include ZVex's failure to show clinical efficacy, a safety signal, immunotherapy competition, and manufacturing.

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SECURITIES: NOT FDIC-INSURED/NOT BANK-GUARANTEED/MAY LOSE VALUE

Sam J. Pearlstein
Co-Head of Equity Research (212) 214-5054
sam.pearlstein@wellsfargo.com

Diane Schumaker-Krieg
Global Head of Research, Economics & Strategy
(212) 214-5070 / (704) 410-1801
diane.schumaker@wellsfargo.com

Todd M. Wickwire
Co-Head of Equity Research (410) 625-6393
todd.wickwire@wellsfargo.com

Paul Jeanne, CFA, CPA
Associate Director of Research
(443) 263-6534 / (212) 214-8054
paul.jeanne@wellsfargo.com

Lisa Hausner
Global Head of Publishing
(443) 263-6522
lisa.hausner@wellsfargo.com

CONSUMER

Beverage/Convenience Stores/Tobacco

Bonnie Herzog (212) 214-5051
Jessica Gerberi, CFA (212) 214-5029
Adam Scott (212) 214-8064

Cosmetics, Household & Personal Care

Chris Ferrara, CFA, CPA (212) 214-8050
Joe Lachky, CFA (314) 875-2042
Zachary Fadem, CPA (212) 214-8018

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Trace A. Urdan (415) 947-5470
Jeffrey Lee (415) 396-4328

Food

John Baumgartner, CFA (212) 214-5015

Homebuilding/Building Products

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Joey Matthews, CPA (415) 396-3873

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Timothy Conder, CPA (314) 875-2041
Karen Wang (314) 875-2556
Marc J. Torrente (314) 875-2557

Restaurants & Foodservice

Jeff Farmer, CFA (617) 603-4314
Imran Ali (617) 603-4315

Retail

Paul Lejuez, CPA, CFA (212) 214-5072
Tracy Kogan (212) 214-8065
Justin C. Matthews (212) 214-8059
Matt Nemer (415) 396-3938
Omair Asif (415) 222-1159
Maren Kasper (415) 396-3194
Kate Wendt (415) 396-3977
Evren Kopelman, CFA (212) 214-8024
Connie Wang (212) 214-5024

ENERGY

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David R. Tameron (303) 863-6891
Gordon Douthat, CFA (303) 863-6920
Brad Carpenter, CFA (303) 863-6894
Jamil Bhatti, CFA (303) 863-6880

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Michael J. Blum (212) 214-5037
Sharon Lui, CPA (212) 214-5035
Praneeth Satish (212) 214-8056
Eric Shiu (212) 214-5038
Ned Baramov (212) 214-8021
David Freeland (212) 214-5050
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Neil Kalton, CFA (314) 875-2051
Sarah Akers, CFA (314) 875-2040
Jonathan Reeder (314) 875-2052
Glen F. Pruitt (314) 875-2047
Peter Flynn (314) 875-2049

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Lauren Hendrix (713) 577-2543

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Jonathan Bock, CFA (443) 263-6410
Ronald Jewsikow (443) 263-6449
Gregory Nelson (704) 410-2197

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Andrew Bond (443) 263-6526

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Rashmi H. Patel, CFA (212) 214-8034

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Vivek Agrawal (443) 263-6563
Charles Nabhan (443) 263-6578

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Jason Harbes, CFA (212) 214-8068

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Brian C. Abrahams, M.D. (212) 214-8060
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Shin Kang, PhD (212) 214-5036
Ronald Hsu, M.D. (212) 214-5064

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Gary Lieberman, CFA (212) 214-8013
Ryan Halsted (212) 214-8022

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Stephen Lynch (901) 271-5552
Nathan Weissman (901) 271-5553

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Luke E. Sergott (212) 214-8027

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Polly Sung, CFA (617) 603-4324
Brian Fitzgerald (617) 603-4277

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Lei Huang (212) 214-8039
Craig W. Bijou (212) 214-8038
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Michael Faerm (212) 214-8026

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Gary S. Liebowitz, CFA (212) 214-5055

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David H. Lim (443) 263-6565
Deepa Raghavan, CFA (443) 263-6517

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Sabina Chatterjee	(212) 214-8049
Maggie Cheung	(212) 214-8011

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Gabe S. Hajde	(216) 643-2967
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Allison Poliniak-Cusic, CFA	(212) 214-5062
Michael L. McGinn	(212) 214-5052

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Justin Ward	(617) 603-4268
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Amir Chaudhri	(212) 214-5045

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Ignatius Njoku	(415) 396-4064
Steve Cho	(415) 396-6056

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Stephan Bisson	(212) 214-8033
John Huh	(212) 2148044

Satellite Communications

Andrew Spinola	(212) 214-5012
----------------	----------------

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Rich Cummings	(212) 214-8030

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Jason S. Belcher	(443) 462-7354

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Jeffrey J. Donnelly, CFA	(617) 603-4262
Dori Kesten	(617) 603-4233
Robert LaQuaglia, CFA, CMT	(617) 603-4263
Tamara Fique	(443) 263-6568

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Young Ku, CFA	(443) 263-6564
Blaine Heck, CFA	(443) 263-6529

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Andrew Spinola (212) 214-5012

Communication Technology

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Michael Kerlan (212) 214-8052

Gray Powell, CFA (212) 214-8048

Priya Parasuraman (617) 603-4269

E-commerce

Matt Nemer (415) 396-3938

Trisha Dill, CFA (312) 920-3594

Information & Business Services

William A. Warmington, Jr. (617) 603-4283

Bill DiJohnson (617) 603-4271

Internet

Peter Stabler (415) 396-4478

Ignatius Njoku (415) 396-4064

Steve Cho (415) 396-6056

IT & BPO Services

Ed Caso, CFA (443) 263-6524

Richard Eskelsen, CFA (410) 625-6381

Tyler Scott (443) 263-6540

IT Hardware

Maynard Um (212) 214-8008

Munjal Shah (212) 214-8061

Santosh Sankar (212) 214-8007

Semiconductors

David Wong, CFA, PhD (212) 214-5007

Amit Chanda (314) 875-2045

Parker Paulin (212) 214-5066

Software/Internet, Technology

Jason Maynard (415) 947-5472

Karen Russillo (415) 396-3505

Vilma Chuy (415) 396-3345

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Timothy W. Willi (314) 875-2044

Robert Hammel (314) 875-2053

Alan Donatiello, CFA (314) 875-2054

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Gina Martin Adams, CFA, CMT (212) 214-8043

Peter Chung (212) 214-8063

Strategic Indexing

Daniel A. Forth (704) 410-3233

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John E. Silvia, PhD (704) 410-3275

Mark Vitner (704) 410-3277

Jay H. Bryson, PhD (704) 410-3274

Eugenio J. Alemán, PhD (704) 410-3273

Sam Bullard (704) 410-3280

Anika Khan (704) 410-3271

RETAIL RESEARCH MARKETING**Retail Research Marketing**

Colleen Hansen (410) 625-6378

Wells Fargo Securities, LLC Institutional Sales Offices

Wells Fargo Securities, LLC
One Boston Place
Suite 2700
Boston, MA 02108
(877) 238-4491

Wells Fargo Securities, LLC
550 California Street
SAC Tower, 6th Floor, Suite 625
San Francisco, CA 94104-1004

Wells Fargo Securities, LLC
10 S. Wacker Drive
18th Floor
Chicago, IL 60606
(312) 345-1187

Wells Fargo Securities, LLC
375 Park Avenue
New York, NY 10152-0005
(800) 876-5670

Wells Fargo Securities International Limited
1 Plantation Place
30 Fenchurch Street
London, EC3M 3BD
44-207-962-2879

