William Blair

Chimerix, Inc.

An Antiviral Specialist Coming of Age: Initiating Coverage With an Outperform Rating and $28 Price Target

We are initiating coverage of Chimerix with an Outperform rating and a price target of $28. Chimerix has produced two clinical antiviral candidates employing its proprietary lipid-conjugation technology. The novel antivirals have much enhanced efficacy and safety profiles compared with the original unconjugated drugs, enabling expanded applications.

Our Outperform rating is centered on our belief that Chimerix's lead asset, CMX001, which is entering Phase III studies, will be a best-in-class and dominating leader in the prevention of double-stranded DNA (dsDNA) viral infections in the transplant setting and beyond, with multibillion-dollar revenue potential. Compared with the currently available therapies and agents in clinical development, CMX001 has the best antiviral potency, the broadest antiviral coverage, a more convenient dosing schedule, and most importantly, a much more favorable tolerability profile, which could enable longer-term use in the prevention setting. The Phase III SUPPRESS study in hematopoietic stem cell transplantation (HSCT) patients for cytomegalovirus (CMV) prevention will begin dosing in mid-2013, and top-line data is expected in the first half of 2015. We expect CMX001 to reach the market by early 2016 and become the market leader in the prevention setting.

We value CMX001 at $22 per share, based on our probability-adjusted net present value (NPV) model. We estimate CMX001 worldwide sales will reach $940 million in the United States and Europe in the HSCT setting in 2030, the year before patents are set to expire. Assuming an 80% probability of success and no terminal value beyond 2031, our probability-adjusted NPV model suggests a fair value for CMX001 of $22 at mid-2014. Potential sources of upside to our revenue estimates include longer duration of prevention therapy (we project 14 weeks); application in treating and preventing viral infections other than CMV; utility of CMX001 in the solid-organ transplant (SOT) setting, which represents a similarly sized market as HSCT; and sales in territories outside the United States and Europe.

Chimerix's second asset, CMX157, has been licensed to Merck for development of novel HIV combo therapies that could have certain advantages over Gilead's industry-leading regimens. We assign $4 per share to the program, which is in Phase I development. We estimate a 35% probability for the CMX157-containing combo to reach the market in 2019, $1.1 billion in peak worldwide sales, and 15% royalties to Chimerix.

We believe a number of catalysts will drive value in the next 12-24 months, including: 1) initiation of SUPPRESS around mid-2013, 2) Phase II data of CMX001 in preemptive treatment of adenovirus (AdV) in HSCT patients during the second half of 2013, and 3) top-line data release of SUPPRESS during the first half of 2015.

Chimerix, Inc., a biopharmaceutical company based in Durham, North Carolina, focuses its research-and-development efforts on antiviral therapies.

May 6, 2013
Basic Report (13-047)

Stock Rating:	**Outperform**
Company Profile:	**Aggressive Growth**
Price Target:	$28.00
Symbol:	CMRX (Nasdaq)
Price:	$20.95 (52-Wk.: $15–$22)
Market Value (mil.):	$500
Fiscal Year End:	December

Estimates	**2012A**	**2013E**	**2014E**
EPS FY	($5.71)	($2.20)	($2.03)
Sales (mil.)	$33.7	$3.5	Nil
Valuation			
P/E	NM	NM	NM

Trading Data	
Shares Outstanding (mil.)	25.5
Float (mil.)	13.1
Average Daily Volume (thous.)	340
Financial Data	
Long-Term Debt/Total Capital	8.2%
Enterprise Value (mil.)	$404
Price/Book	NM

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Please consult pages 39-40 of this report for all disclosures.

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Investment Summary

Technology Platform: Lipid-Conjugation Technology to Enhance Drug Properties
Based in Durham, North Carolina, Chimerix is a biopharmaceutical company focused on developing antiviral therapies. Founded in 1996, the company licensed lipid-conjugation technology from the University of California in 2002 and has to date pushed two drug candidates into the clinic.

- The first drug candidate is CMX001, a lipid-conjugate of cidofovir (brand name Vistide), manufactured by Gilead Sciences. Cidofovir is approved for CMV retinitis; although it is active against a broad spectrum of double-stranded DNA (dsDNA) viruses, its utility is restricted due to safety issues including kidney toxicity and neutropenia. CMX001 can achieve much higher intracellular concentrations than cidofovir, has much higher potency, is dosed orally twice a week as opposed to intravenously like cidofovir, and has eliminated cidofovir's key adverse effects of kidney toxicity and neutropenia.

- The second drug candidate is CMX157, a lipid-conjugate of tenofovir (brand name Viread), developed and marketed by Gilead Sciences for the treatment of HIV. Compared with Viread, CMX157 has much-improved antiviral potency, can overcome Viread resistance mutations, has a much longer half-life, can be dosed orally once weekly, and should be devoid of the renal and bone toxicities that long-term treatment with Viread typically leads to.

Viral Infections in the Transplant Setting Represent an Important Market
More than 20,000 HSCTs and 28,000 SOTs are performed each year in the United States, and a similar number of transplants are performed in Europe; these numbers have been growing at about a 4% annual rate. Effectively managing opportunistic infections in transplant patients is important for transplant success and decreasing morbidity and mortality afterward. The most common viral infection in transplant recipients is cytomegalovirus (CMV); adenovirus (AdV) and BK virus (BKV) are also common and can cause serious consequences. These viruses are present in a majority of the adult population but are suppressed by the intact immune system. In a transplant patient whose immune system is severely compromised, these viruses could be reactivated if they are already in the patient or infect the patient for the first time, leading to increased morbidity and mortality.

Existing Antiviral Therapies in HSCT Are Used for Treatment or Preemption, but Are Not Suitable for Prevention
There are three major paradigms to manage viral infections in transplant: treating the infections when symptoms are present, preemptively treating the patient when the virus appears in the blood (viriemia) but before symptoms develop, and treating the patients prophylactically to prevent infection.

Among these three approaches, prevention is the most desirable. In fact, in SOT, administering ganciclovir (brand name Cytovene) or its prodrug valganciclovir (brand name Valcyte) for three or six months to prevent CMV infection is standard of care. In HSCT, however, longer-term use of ganciclovir for CMV prevention was compromised by its toxicity. At present, there are no therapies approved in HSCT for CMV prevention.

Chimerix's Lead Asset, CMX001, Is Best-in-Class and Uniquely Suitable for Prevention
CMX001 has the following properties that make it suitable for the prevention setting in HSCT.

- It is the most potent among all agents, including those that have been approved and those currently in clinical development.

- It has the broadest spectrum against all five families of dsDNA viruses, including the most prevalent CMV, as well as other viruses such as AdV and BKV. Other drug candidates in development target only CMV, or CMV and one other virus.

- It has a high barrier to resistance. No resistant mutations have been detected in humans to date. Most other agents have documented resistance.

- Its tolerability profile is suitable for the prevention indication. The major side effect is diarrhea, which could be managed to minimize the adverse impact.

- It has a long half-life, enabling a convenient twice-a-week oral dosing.

Registrational Strategy: Top-Line Data From SUPPRESS by Early 2015 and on the Market by Early 2016

Chimerix has devised an efficient registrational strategy to push CMX001 on the market as quickly as is practical, in our opinion.

- The first indication to pursue for CMX001 is prevention of CMV infection in the HSCT setting, with potential approval and launch in late 2015 or early 2016. Chimerix wishes to use the single pivotal SUPPRESS study to obtain conditional approval in this indication first, then supplement the new drug application with a pediatric HSCT study to obtain full approval.

- Chimerix might also pursue a preemption approval for adenovirus infections in the HSCT setting. As AdV infection is much less frequent than CMV, a prevention study design is not practical because it would require an excessive number of patients. As a result, Chimerix is conducting a Phase II preemption study in AdV-infected HSCT patients, and data is expected during the second half of 2013. Chimerix may be able to file for approval in this setting based on the Phase II data if a benefit in reduction in mortality or other significant benefits can be demonstrated.

- To fully exploit CMX001's broad spectrum coverage of dsDNA families of viruses and further differentiate CMX001 from other agents, Chimerix designed the secondary endpoints in SUPPRESS to examine CMX001's activity in preventing infections by other dsDNA viruses, such as AdV and BKV. Significant activities against these viruses could lead to additional claims on the label and expand CMX001's utility into other viral infections.

- Chimerix is negotiating with European regulatory authorities to elucidate a clear pathway to approval on the continent.

- Besides pursuing indications in the HSCT setting, Chimerix might expand clinical studies of CMX001 into the SOT setting. Although ganciclovir and valganciclovir are already used for CMV prevention in SOT, CMX001 might provide important incremental advantages in higher potency as well as activity against ganciclovir and valganciclovir mutations, which could translate into better efficacy, a broader spectrum covering not only CMV (for example, BKV is particularly detrimental in kidney-transplant recipients), a more convenient dosing schedule (twice a week versus once daily), and, most importantly, a much better tolerability profile devoid of myelosuppression.

Chance of Success of SUPPRESS Based on Phase II Data: We Estimate 80%

In the Phase II study (Study 201) for CMV prevention in HSCT patients, 10% of CMX001-treated patients experienced prevention failure, versus 37% from placebo, which demonstrated a relative 73% reduction in CMV disease and risk for infection (p=0.002). When the inclusion criteria and protocol of the Phase III SUPPRESS study were applied to the Study 201 patients, the analysis yielded a relative reduction of 52% in prevention failure for the CMX001-treated group versus placebo, in the most conservative scenario.

Further, in the SUPPRESS study, the initiation of prevention therapy is much earlier (within the first two weeks following transplant) than that in Study 201 (on day 24 post-transplant), which could lead to more favorable outcomes, as earlier initiation of prevention therapy might result in more effective suppression of CMV reactivation. Lastly, the duration of therapy in SUPPRESS is also longer (14 weeks) than that in Study 201 (9-13 weeks), which is also in favor of SUPPRESS.

The Phase III SUPPRESS study is powered at 85% to detect 50% reduction in prevention failure for CMX001-treated patients versus placebo. Based on the overall profile of CMX001, the Phase II data, and the SUPPRESS design, we are highly confident that SUPPRESS will be a positive study. We assign an 80% probability of success to SUPPRESS.

CMX001 Commercialization Strategy: Taking on North America
Chimerix plans to commercialize CMX001 on its own in the United States and Canada. The market is highly concentrated; it is estimated that 200 institutions in the United States perform HSCT and/or SOT transplants. As a result, a commercial team of 50 people would be sufficient to market CMX001 in North America.

Chimerix is contemplating strategies in Europe and may decide to commercialize CMX001 on the continent by itself. In our model, we project that Chimerix will license out the commercial rights to CMX001 to a partner and receive 30% royalties on sales on the continent.

We expect Chimerix licensing rights to CMX001 in territories outside the United States and Europe to maximize the global commercial potential of CMX001.

Competitive Landscape in CMV Prevention
There are two major competitive agents in development for CMV prevention: letermovir from partners Merck and AiCuris, and TransVax from partners Astellas and Vical. Both letermovir and TransVax are entering Phase III studies, perhaps a few months behind CMX001 in timing. We note that both letermovir and TransVax are specific for CMV, while CMX001 is much broader in spectrum coverage. Further, Phase II studies for both agents excluded high-risk patients, while Study 201 of CMX001 included high-risk patients up to 35% of the study population. We expect to continue to monitor the progress of the competitive agents; the Phase III designs and data will determine relative strengths of the profiles of these agents, which will consequently affect their respective market shares once commercialized.

William Blair Revenue Model for CMX001 Sales in the United States
We illustrate our projections for CMX001 U.S. sales in exhibit 1, on the following page. As discussed above, 20,000 HSCT procedures are performed annually in the United States; the number of procedures grew at a 4% annual compound rate from 2001 to 2011. We conservatively project 2% growth from 2016 and beyond.

- ***High risk versus low risk.*** The prevention strategy is likely most useful in higher-risk patients, although physicians we spoke with predicted that prevention is likely to be used universally if a medicine with a suitable profile is available. There are a few factors that determine whether the HSCT patient is at high risk for CMV infections; these include whether the patient already harbors the CMV virus and whether the transplant is allogeneic (patients receive the transplanted cells from other donors) or autologous (patients receive their own transplanted cells or from an identical twin); allogeneic recipients are at higher risk. About 40%-50% of allogeneic patients receive cells from mismatched donors or cord blood (banked stem cells from newborns' placentas) as opposed to well-matched donors; these patients are considered to be at even higher risk.

- ***Competitive market share.*** As illustrated in exhibit 1, we peg peak penetration of CMX001 at 75% in high-risk allogeneic transplant patients and 50% in low-risk allogeneic transplant patients. For autologous patients, we conservatively estimate 30% of patients receive prevention and for CMX001 to have 35% peak penetration. These assumptions are based on profiles of CMX001 and competitive agents letermovir and TransVax. We note that while CMX001 studies included high-risk patients up to 35% of the study population, both Phase II studies of letermovir and TransVax excluded high-risk patients. It is unclear whether these two agents are to be studied in high-risk agents in Phase III studies. As a result, in our current model, we assume that CMX001 will have less competition in high-risk patients and more competition in lower-risk patients.

Exhibit 1
Chimerix, Inc.
William Blair U.S. Revenue Model for CMX001

		2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030
U.S. Allogeneic HSCT Adults (CMV/BKV)		8,784	8,960	9,139	9,322	9,508	9,698	9,892	10,090	10,292	10,498	10,708	10,922	11,140	11,363	11,590
Annual growth	2%															
Allogeneic unrelated donors (high risk)		3,514	3,584	3,656	3,729	3,803	3,879	3,957	4,036	4,117	4,199	4,283	4,369	4,456	4,545	4,636
proportion eligible	40%															
Market share: CMX001		20%	40%	50%	70%	75%	75%	75%	75%	75%	75%	75%	75%	75%	75%	75%
Allogeneic High Risk Adults treated with CMX001		703	1,434	1,828	2,610	2,852	2,909	2,968	3,027	3,088	3,149	3,212	3,277	3,342	3,409	3,477
Allogeneic related donors (low risk)		5,270	5,376	5,483	5,593	5,705	5,819	5,935	6,054	6,175	6,299	6,425	6,553	6,684	6,818	6,954
proportion eligible	60%															
Market share: CMX001		20%	30%	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%
Allogeneic Low Risk Adults treated with CMX001		1,054	1,613	2,193	2,237	2,282	2,328	2,374	2,422	2,470	2,519	2,570	2,621	2,674	2,727	2,782
U.S. Allogeneic HSCT Peds (CMV/AdV)		1,577	1,608	1,640	1,673	1,707	1,741	1,776	1,811	1,847	1,884	1,922	1,960	2,000	2,040	2,080
Annual growth	2%															
Market share: CMX001		5%	20%	50%	50%	50%	50%	50%	50%	50%	50%	50%	50%	50%	50%	50%
Allogeneic Pediatrics treated with CMX001		79	322	820	837	853	870	888	906	924	942	961	980	1,000	1,020	1,040
U.S. Autologous Adults + Peds (BKV/CMV/AdV)		14,640	14,933	15,232	15,536	15,847	16,164	16,487	16,817	17,153	17,496	17,846	18,203	18,567	18,939	19,317
Annual growth	2%															
High-risk autologous aduls + peds eligible	30%	4,392	4,480	4,569	4,661	4,754	4,849	4,946	5,045	5,146	5,249	5,354	5,461	5,570	5,682	5,795
Market share: CMX001		0%	2%	6%	10%	20%	35%	35%	35%	35%	35%	35%	35%	35%	35%	35%
Autologous patients treated with CMX001		-	90	274	466	951	1,697	1,731	1,766	1,801	1,837	1,874	1,911	1,950	1,989	2,028
# total CMX001 treated patients in U.S.		1,836	3,458	5,115	6,150	6,939	7,805	7,961	8,120	8,282	8,448	8,617	8,789	8,965	9,144	9,327
# patients treated with CMX001 in US, adjusted		1,569	2,956	4,374	5,258	5,932	6,673	6,806	6,943	7,081	7,223	7,368	7,515	7,665	7,818	7,975
compliance	95%															
discontinuation	10%															
Pricing per course (in dollars)		56,806	57,942	59,101	60,283	61,488	62,718	63,973	65,252	66,557	67,888	69,246	70,631	72,044	73,484	74,954
duration of treatment (weeks)	14															
price increase	2%															
gross-to-net discount	12%															
Net pricing per course (in dollars)		49,989	50,989	52,009	53,049	54,110	55,192	56,296	57,422	58,570	59,742	60,936	62,155	63,398	64,666	65,960
Net CMX001 sales in U.S. (in dollars)		78,457	150,733	227,472	278,942	321,003	368,296	383,175	398,655	414,761	431,517	448,950	467,088	485,958	505,591	526,017

Source: William Blair & Company, L.L.C. estimates

- ***Pricing.*** We price CMX001 at $56,000 per 14-week course of therapy in the United States at launch in 2016, and we apply a number of discount factors, including 95% compliance rate, 10% discontinuation rate, and a gross-to-net discount rate of 12%. As a result, net price is at $50,000 per course for CMX001 in 2016; we project an annual 2% price increase beyond 2016. As illustrated in exhibit 11, on pages 18 and 19, the current U.S. pricing of valganciclovir in SOT is roughly $12,000 for 12 weeks of CMV prevention therapy, and salvage therapies, such as Foscavir, cost $30,000 to $44,000 per course. We believe the pricing of CMX001 in our model is reasonable, as there are no approved prevention therapies in HSCT, CMX001 can salvage valganciclovir failures as well, and CMX001 might provide greater benefit to patients because it could prevent infections from a number of important viruses in addition to CMV.

- ***Our revenue projection.*** We therefore derive U.S. peak sales of $526 million in 2030, the year before the patents are set to expire. We projected EU sales of CMX001 with similar rationale and reached peak sales for CMX001 in the United States and Europe at $940 million.

Outperform Rating Based on Market Potential of CMX001 in the HSCT Setting, With Substantial Upside

Our Outperform rating is centered on our belief that CMX001 will dominate as a prevention therapy in the future transplant setting. As discussed above, we believe that CMX001 sales will reach $940 million at peak in the United States and Europe in the HSCT setting only, before patent expiration in 2031.

Potential sources of major upside to our revenue estimate include the following:

- Longer duration of prevention treatment in HSCT. We assume 14 weeks in our model, which is the duration being studied in the Phase III SUPPRESS study. In SOT, the prevention regimen in practice is as long as 24 weeks, and in some cases one year.

- The SOT market, which is a slightly larger market than HSCT. Ganciclovir and valganciclovir are entrenched in the market, but CMX001 could provide important incremental advantages.

- Application of CMX001 in treating and preventing viral infections other than CMV.

- Sales in geographical areas outside the United States and Europe.

Chimerix's Second Asset, CMX157, Is a Once-Weekly Nuke Targeting HIV

CMX157 is a lipid-modified prodrug of tenofovir (brand name Viread); CMX157 is 200 times more potent than tenofovir and can overcome tenofovir resistance in vitro. Further, the long half-life might enable once-weekly dosing. Lastly, the safety profile is much improved as well, and CMX157 could be devoid of the renal and bone toxicities observed with Viread.

Chimerix completed a Phase I study of CMX157 before licensing it to Merck in July 2012. Merck intends to put CMX157 together with other compounds in the portfolio to develop a once-weekly oral combo therapy for HIV.

Intellectual Property for CMX001 and CMX157; License From University of California

Composition-of-matter (CoM) patents for CMX001 and CMX157 are set to expire in December 2020; adding the five-year Hatch-Waxman extension, both will eventually expire in December 2025. In addition, Chimerix believes the method of use and polymorph CoM applications could extend protection of both assets until 2031. Therefore, we project sales for CMX001 and CMX157 until 2031, and zero beyond 2031. We are comfortable with these assumptions in our model based on the fact that polymorph patents are a commonly used strategy for CoM extension and also because Merck performed extensive due diligence on Chimerix's patent portfolio before its in-licensing of CMX157.

Chimerix licensed exclusive worldwide rights to the lipid-conjugation technology to modify antiviral compounds from The Regents (governing board) of the University of California (UC) in 2002. Chimerix issued 64,788 shares of common stock to UC for the license and is obligated to pay UC up to $3.4 million in milestones and low-single-digit royalties on sales.

Valuation: 12-Month Price Target of $28

In building a probability-adjusted NPV model, we estimate the peak sales of a given drug candidate, its probability of advancing to the next stage of development and eventually reaching the market, and a company's share of revenue and expenses depending on the commercialization plan and/or structure of partnerships, if any. We then calculate the cash flows after adjusting all revenues and expenses with respective cumulative probabilities for each stage. The cash flows are then discounted back using an industry-specific weighted average cost of capital (WACC) of 12% to arrive at a probability-adjusted NPV for each drug candidate. Once we determine the NPV for each candidate, we then add net cash and other costs, which include expenses not directly associated with the development of the clinical candidates, to arrive at a fair value estimate for a stock.

In exhibit 2, we summarize our sum-of-the-parts valuation for Chimerix. CMX001 in the HSCT setting, pegged at $22 per share, constitutes the majority of Chimerix valuation; we assign an 80% probability of success to CMX001 in the HSCT setting.

We assume peak sales of CMX001 to be roughly $530 million in the United States and $410 million in Europe. In the United States, we factor in a 50-person commercial organization by Chimerix to market CMX001; for Europe, we project that Chimerix will license out the commercial rights to CMX001 to a partner and receive 30% royalties on EU sales.

CMX157 is valued at about $4 per share; we assume a 35% probability of success, peak sales of $1.1 billion in HIV, and 15% royalties on sales to Chimerix.

Exhibit 2
Chimerix, Inc.
Sum-of-the-Parts Fair Value
(dollars in thousands)

Drug Candidate	Peak Sales	Stage of Development	Estimated Launch Date	Probability of Commercialization	Percentage of Sales to Company	Probability-Adjusted NPV	Value Per Share	Percentage of Fair Value
CMX001— United States	$526,017	Phase III start mid-2013	H1:2016	80%	100%	$445,114	$17.42	62.2%
CMX001— European Union	$413,065	Phase III start mid-2013	H1:2017	80%	30%	$114,102	$4.47	15.9%
CMX157— HIV	$1,074,060	Phase I	H1:2019	35%	15%	$112,361	$4.40	15.7%
Subtotal						**$671,576**	**$26.29**	**93.8%**
Net Cash at Mid-2014						$55,829	$2.19	7.8%
Net Present Value of Additional Gain (Loss)*						($11,364)	($0.44)	(1.6%)
Sum-of-the-Parts Fair Value						**$716,042**	**$28.03**	**100.0%**

*Includes costs not directly related to programs above
Sources: Company reports and William Blair & Company, L.L.C. estimates

Key Catalysts Driving Value in the Next 12-24 Months

In exhibit 3, we summarize key upcoming events for Chimerix; highlighted events are potential stock-moving catalysts.

Over the next 12-24 months, Chimerix will dose the first patient in the SUPPRESS study around mid-2013, report Phase II data of CMX001 in preemptive treatment of AdV in HSCT patients during the second half of 2013, and report top-line data of SUPPRESS during the first half of 2015.

Exhibit 3
Chimerix, Inc.
Clinical Development Timeline and Milestones

Drug	CMX001			CMX157
Indication	Prevention of CMV Infection in HSCT	Preemption of AdV Infection in Adult and Pediatric HSCT	Smallpox Under Animal Efficacy Rule	HIV
Class	Lipid-conjugated cidofovir (Vistide)			Lipid-conjugated tenofovir (Viread)
Partner				*Merck*
Q3:12				Licensed worldwide rights to Merck (July)
Q4:12				
Q1:13		Type C meeting with the FDA		
Q2:13	Finalized Phase III SUPRESS adult study design in April; dosing mid-year (Study 301, n=450)		BARDA contract renewal (May)	
Q3:13				
Q4:13	Initiate Phase III pediatric study (Study 311)	Phase II pediatric study data release (Study 202)		
Q1:14		Potential NDA submission		
Q2:14				
Q3:14	Completion of enrollment of SUPPRESS			
Q4:14		Potential approval and launch		
H1:15	Data release from SUPPRESS			Merck starts Phase II study; Chimerix collects milestone
H2:15	Submit NDA and MAA			
H1:16	Approval and launch; data from pediatric study			
H2:16				

Highlight: events likely to affect stock price
Sources: Company reports and William Blair & Company, L.L.C. estimates

Experienced Management Team

Chimerix is led by an experienced management team whose executives have successful track records. In exhibit 4, we summarize key experience of the top executives.

Exhibit 4
Chimerix, Inc.
Management Team

Management	Position	Previous Experience
Kenneth I. Moch	President and Chief Executive Officer	Chief operating officer, Chimerix Founder, Euclidean Life Science Advisors President and CEO, Biomedical Enterprises, Inc. Managing director, Healthcare Investment Banking, Think Equity President and CEO, Alteon President and CEO, Biocyte Corporation Co-founder and vice president, The Liposomal Company, Inc. Consultant, McKinsey & Company, Inc.
Timothy W. Trost	Senior Vice President and Chief Financial Officer	Vice president and CFO, Argos Therapeutics, Inc. Senior vice president and CFO, InteCardia, Inc. Executive vice president and CFO, Coastal Physician Group, Inc. Vice president of finance, Morganite North America, Inc. Senior manager, Price Waterhouse
M. Michelle Berrey, M.D., M.P.H.	Chief Medical Officer	Chief medical officer, Pharmasset, Inc. Vice president, viral diseases, clinical pharmacology and discovery medicine, GlaxoSmithKline
Michael D. Rogers, Ph.D.	Chief Development Officer	Chief development officer, Pharmasset, Inc. Vice president, viral diseases, discovery medicine, HIV clinical research, GlaxoSmithKline
Jung E. Choi	Senior Vice President–Corporate Development	Head of corporate development, Gilead Sciences Vice president, Bay City Capital Engagement manager, McKinsey & Company, Inc.
Hervé Mommeja-Marin, M.D.	Vice President–Clinical Research	Senior medical director, infectious diseases, i3 Research Director, clinical research, Gilead Sciences Clinical research physician, Triangle Pharmaceuticals

Sources: Chimerix, Inc. and William Blair & Company, L.L.C.

Key Risks to Our Outperform Rating and Price Target

Key risks to our Outperform rating and price target include: 1) failure of CMX001 to meet primary or second endpoints in the SUPPRESS study, 2) a worse-than-expected tolerability profile for CMX001, 3) failure of CMX157 to advance in Merck's HIV pipeline, 4) other clinical and business-development setbacks, and 5) financing risks.

Opportunistic Infections in the Transplant Setting

Major Types of Transplant

In general, transplant can be classified into two major types: HSCT and SOT. In exhibit 5, we summarize the types and definitions of transplants.

Hematopoietic stem cells (HSCs) are blood-forming cells. Transplanting healthy donor HSCs into a recipient who has malignant or damaged bone marrow could lead to disease remission; HSCT is curative for a number of hematological cancers and blood disorders. During an HSCT, the recipient first undergoes a conditioning regimen to suppress the immune system, and then the donor cells are infused. The donor cells will begin to grow in the recipient and produce differentiated blood cells, including red blood cells, white blood cells, and platelets; the establishment of successful survival and growth of the donor cells is called engraftment. Engraftment of the transplanted cells usually occurs within 40 days after transplant; the goal is for the absolute neutrophil count (ANC) to reach 500. T cells are usually reconstituted 100 days after transplant. As a result, the first 100 days is important for the patient for engraftment; it is optimal to stay free of infections and complications during this period.

HSCT is further defined by the donor of the stem cells (allogeneic or autologous), the source of the stem cells (bone marrow, peripheral blood, or cord blood), and the immunosuppressive regimen the recipient receives before the transplant.

Major SOTs involve heart, kidney, pancreas, liver, and lung, and almost all are allograft. Patients also need to go through a conditioning regimen to suppress the immune system. The patients are exposed to the same risk for infections and complications after transplant.

Exhibit 5
Chimerix, Inc.
Types of Transplants

HSCT (bone marrow, peripheral blood, or cord blood stem cells)		SOT (heart, kidney, pancreas, liver, lung, skin, and other tissues)	
Autologous	Transplanted cells come from the body of the transplant recipient	**Autograft**	Skin graft, other tissue graft
Syngeneic (autologous)	Transplanted cells come from identical twin sibling	Isograft (autograft)	Transplanted tissues or organs come from identical twin sibling
Allogeneic	Transplant cells come from a related or unrelated donor	**Allograft**	Transplant organs come from a related or unrelated human donor; heart, kidney, pancreas, kidney/pancreas double, liver, lung
Cord blood (allogeneic)	Transplant cells come from umbilical cord and placenta from a newborn baby	**Xenograft**	Transplanted organs or tissues come from another species, such as porcine heart valve

Notes: HSCT – hematopoietic stem cell transplantation; SOT – solid organ transplantation
Sources: Wikipedia and William Blair & Company, L.L.C.

Post-Transplant Complications

Post-transplant complications are frequent, including viral infections, bacterial infections, fungal infections, graft-versus-host disease (GvHD), and graft failure.

Most Infections in the Transplant Setting Are Opportunistic Infections

Opportunistic infections are defined as infections caused by organisms—viruses, bacteria, protozoans, and fungi—that are ubiquitous in the environment but rarely cause disease in an immune-competent host. The majority of infections in transplant recipients are opportunistic and are a major cause of death in immune-compromised patients.

The major factor responsible for post-transplant infection is the use of immunosuppression. In general, the more mismatched the donor and recipient, the greater the risk for GvHD and the greater need to continue immunosuppression after transplant. This leads to a higher risk of infections and complications. Most allogeneic HSCTs are considered high risk, and cord blood transplant and transplant from mismatched donors are considered higher risk. Further, HSCT recipients who already have latent viruses are also considered high risk. For SOT, all allografts are considered high risk.

Viral Infections in the Transplant Setting

Five dsDNA viral families. The viruses most responsible for opportunistic infections in transplant recipients are dsDNA viruses. Five families are particularly relevant in human infections: herpes virus, adenovirus, polyomavirus, papillomavirus, and poxvirus. In exhibit 6, we summarize the five families, the representative viruses from each family, and the common diseases they cause.

Exhibit 6
Chimerix, Inc.
Five dsDNA Viral Families

dsDNA Viral Family	Representative Viruses Relevant in Human Infections
Herpesvirus	CMV (cytomegalovirus) HSV-1 and 2 (herpes simplex virus) EBV (Epstein-Barr virus, causes mono) VZV (varicella zoster virus, causes chickenpox and shingles)
Adenoviruses	More than 50 subspecies
Polyomavirus	BKV (BK virus) JCV (JC virus, causes progressive multifocal leukoencephalopahy, or PML)
Papillomavirus	HPV (human papilloma virus, causes cervical cancer)
Poxvirus	VACV (vaccinia) VARV (variola, or smallpox)

Sources: Chimerix Inc. and William Blair & Company, L.L.C.

Viruses causing the most prevalent opportunistic infections in transplant recipients belong to the herpes virus family: CMV, herpes simplex virus (HSV) 1, HSV 2, varicella zoster virus (VZV), and Epstein-Barr virus (EBV). The polyomavirus family, with representatives such as JC virus (JCV) and BK virus (BKV), are also prevalent. AdV is less prevalent, but its infections carry a high mortality rate in pediatric HSCT and SOT recipients.

Risk of CMV reactivation in HSCT. Most people are infected with dsDNA viruses when reaching adulthood; it is estimated that in the United States, 50%-80% are infected with CMV by age 40. Most healthy children and adults have no symptoms with their CMV infections and usually are not aware that they have been infected; CMV is suppressed by the immune system.

However, about 20% to 60% of all transplant recipients develop symptomatic CMV infection as a result of immunosuppression. Risk assessment of CMV infection in HSCT is based on the serological status of the donor and recipient for viruses. An individual who has been infected with CMV develops antibodies against the virus in the serum and the status is termed seropositive. An individual who has never been infected is seronegative.

As summarized in exhibit 7, for an allogeneic HSCT transplant, regardless of the donor's serological status, if the recipient is seropositive for CMV, the risk for reactivation of the virus during transplant is 80%. For an autologous transplant, the risk is decreased to 40% for a seropositive recipient.

Morbidity and Mortality Associated With Viral Infections in the Transplant Setting

In HSCT and SOT recipients, viral infections are associated with significant morbidity, mortality, graft rejection, and co-infection with other pathogens. CMV is the most common and arguably the single-most important viral infection in HSCT as well as SOT, which if uncontrolled could lead to fever, pneumonia, gastrointestinal ulcers, hepatitis, retinitis (which could lead to retinal detachment and blindness), graft injury, and graft dysfunction ranging from mild to life-threatening. CMV infection usually develops during the first 100 days after transplant.

As summarized in exhibit 7, for R+ allogeneic HSCT recipients, death rate in the first year following HSCT that is not due to relapse of the underlying disease (nonrelapse mortality) is 21%; the majority of the deaths result from infections. For R+ autologous HSCT recipients, the nonrelapse mortality rate is 27%.

Exhibit 7
Chimerix, Inc.
Risk Assessment for CMV Reactivation in HSCT

Transplant Type	CMV Serostatus	Risk of CMV Infection	Nonrelapse Mortality
Allogeneic	R+, regardless of donor status	80%	21%
	D- / R-	<5%	17%
	D+ / R-	30%	18%
Autologous	R+	40%	27%

R+: recipient seropositive for CMV; R-: recipient seronegative for CMV
D+: donor seropositive for CMV; D-: donor seronegative for CMV
Risk of CMV infection: likelihood of detectable CMV in blood
Nonrelapse mortality: deaths in the first year following HSCT that are not due to relapse of the underlying disease
Source: Chimerix, Inc.

Three Paradigms Addressing Viral Infections in Transplant: Treatment, Preemption, and Prevention

In the past 30 years, significant improvements have been made in the management of post-transplant infections. The standard of practice evolved from treatment to preemption and to prevention where possible. The development of prevention strategies together with early identification and treatment strategies for various infections has contributed to decreased lengths of hospital stays, lower costs, decreased patient morbidity, and in some cases, increased patient survival.

Treatment: initiating antiviral treatment after detection of disease symptoms. In the 1980s, the standard of care for viral infection in the transplant setting was initiating therapy when the infection signs and symptoms were already present.

Preemptive therapy: initiating antiviral treatment upon detection of virus in blood before onset of disease symptoms; current standard of care in HSCT. The current standard of care is to frequently monitor viral DNA in patients' blood. When a viral presence is detected in the patient and before the infection becomes full-blown and leads to clinical signs and symptoms, treatment is initiated. Ganciclovir was approved for preemptive therapy in HSCT, which demonstrated 93% reduction of CMV disease. The goal of preemptive therapy is to halt progression of the disease.

Prevention: administering antiviral therapy routinely to all at-risk patients to prevent reactivation of the latent virus; this is the most ideal paradigm and current standard of care in SOT. However, currently available therapies are too toxic to be used for prevention in HSCT. Ganciclovir was studied in the HSCT prevention setting in the 1990s. Although it demonstrated a decrease in CMV reactivation and disease, it also led to severe neutropenia in 30% of patients, which resulted in an increased rate of fatal opportunistic infections from other pathogens. To date, no therapies are approved or being used in the prevention setting for HSCT. The goal of prevention is for less frequent monitoring and to eliminate the need for preemptive therapy.

Ganciclovir is the standard of care for prevention in SOT. Prevention of CMV infection is the standard of care in SOT, and ganciclovir and valganciclovir are the most commonly used agent in SOT recipients. Ganciclovir is a 2'-deoxyguanosine nucleoside analogue (nuke) that inhibits the replication of herpes viruses in vitro and in vivo; it is administered either intravenously or orally. Patients at risk for CMV infection are usually given ganciclovir IV within a few days after transplant while at the hospital, and they continue with oral ganciclovir. The duration of the prevention is typically between three and six months, although in some cases prevention therapy could be as long as one year.

Because of the wide use of ganciclovir for CMV prevention in SOT, resistance to ganciclovir has become a problem. Factors associated with increased risk for ganciclovir resistance include high-risk CMV D+/R- serologic status, high viral load, high level of immunosuppression, prolonged antiviral exposure, and suboptimal serum drug concentrations. Valganciclovir, an oral prodrug of ganciclovir with 10 times greater bioavailability, is often used. However, valganciclovir shares the same myelosuppression toxicity as ganciclovir.

Prevention leads to better outcomes than preemptive therapy in SOT, as evidenced by significantly decreased CMV disease incidence and higher long-term graft survival. Further, a number of studies in SOT favor longer duration of prevention therapy for significantly lower incidence and severity of CMV disease.

No drugs approved for prevention in HSCT setting. No drugs have been approved for prevention of CMV or other viruses in the HSCT setting, mostly because of renal toxicity (cidofovir) and myelosuppression (cidofovir, ganciclovir and valganciclovir).

CMX001 Poised to Be First-in-Class Therapy for the Prevention Paradigm in HSCT

A potent, broad-spectrum, safe, and well-tolerated therapy could be suitable for the prevention paradigm in HSCT, and we believe the profile of CMX001 supports such an application.

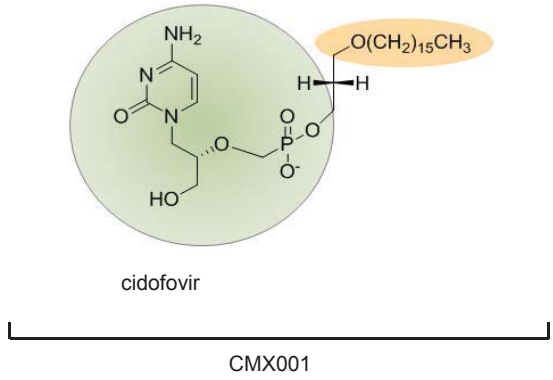
The Technology: Lipid Conjugation of Cidofovir; Improved Potency and Safety

Cidofovir (branded Vistide and manufactured by Gilead) is indicated for CMV retinitis in AIDS patients. Cidofovir has a very broad spectrum against dsDNA viruses; however, it carries a black box warning for renal failure, neutropenia, and carcinogenicity. It is also not orally bioavailable and has to be administered intravenously.

CMX001 is a lipid-modified version of cidofovir, as illustrated in exhibit 8. Following oral dosing, CMX001 is absorbed through the gut, stays intact in the plasma, and is taken up by cells. The lipid tail enables much higher efficiency in delivering CMX001 into cells, minimizing exposure of free cidofovir in the plasma. The lipid tail is cleaved once CMX001 enters the cell, and the resulting cidofovir is converted to the active antiviral species CDV-PP, which blocks replication of dsDNA viruses.

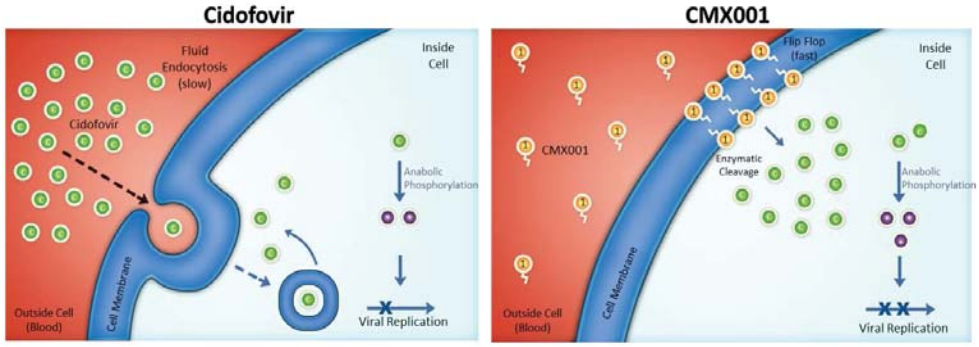
Exhibit 8
Chimerix, Inc.
CMX001: An Enhanced Version of Cidofovir

a) Structure of CMX001: Cidofovir With a Conjugated Lipid Tail



Source: Chimerix, Inc.

b) Increased Intracellular Uptake of CMX001 Versus Cidofovir



Source: Chimerix, Inc.

In exhibit 9, we summarize and contrast CMX001 with cidofovir. CMX001 achieves much lower plasma concentration than cidofovir, but much higher intracellular concentration, thereby increasing potency and greatly enhancing the broad-spectrum coverage against dsDNA viruses. CMX001 is also given orally twice a week (BIW), while cidofovir is given intravenously. Further, cidofovir interacts with the human organic anion transporterv1 (hOAT1) receptor on kidney cells, leading to uptake into the duct systems of the kidney and subsequent renal toxicity. The lipid modification on CMX001 eliminates the interactions with hOAT1, and CMX001 is not taken up in the kidney; hence, there has been no kidney toxicity observed to date.

Exhibit 9
Chimerix, Inc.
Comparison of CMX001 and Cidofovir

	CMX001	Cidofovir
Route of administration	Oral twice weekly	IV weekly
Plasma concentration	Low	High
Intracellular concentration of active antiviral species cidofovir-PP	100x	1x
Antiviral potency	More than 400 times more potent by EC_{50} against CMV; much enhanced potency across spectrum	
Nephrotoxicity	None	Black box warning
Neutropenia	None	Black box warning
Animal carcinogenicity	Yes	Yes

Sources: Chimerix Inc. and William Blair & Company, L.L.C.

The Most Potent and Broadest Spectrum

In exhibit 10, we summarize the EC_{50} (half maximal effective concentration) of various agents on the market and in development against different families of dsDNA viruses. CMX001 is the most potent and has the broadest spectrum.

Cross-Comparison With Agents on the Market and in Development

In exhibit 11, on pages 18 and 19, we compare and contrast key profiles of antiviral agents used in the transplant setting, both on the market and in development. Judging potency, efficacy, and safety in totality, we believe that CMX001 has the potential to become best-in-class for CMV prevention in the HSCT setting.

Exhibit 10
Chimerix, Inc.
CMX001: Broad-Spectrum Activity Against dsDNA Viruses

Viral Family	Virus	CMX001	Cidofovir	Ganciclovir*	Foscarnet	Acyclovir	Maribavir	Letermovir
Herpes	Cytomegalovirus (CMV)	**0.001**	0.4	3.8	50-800	>200	0.31	0.0051
	Epstein-Barr Virus (EBV)	**0.03**	65.6	0.9	<500	6.2	0.63	>10
	Human Herpesvirus 6A (HHV-6A)	**0.003**	2.7	5.8	16	10	Inactive	>10
	Human Herpesvirus 8 (HHV-8)	**0.02**	2.6	8.9	177	>100	Inactive	No data
	Herpes Simplex Virus 1 (HSV-1)	**0.01**	3	0.7	92-95	3.8	Inactive	>10
	Herpes Simplex Virus 2 (HSV-2)	**0.02**	6.5	2.5	91-96	4.4	Inactive	>10
	Varicella Zoster Virus (VZV)	**0.0004**	0.5	1.3	39.8	3.6	Inactive	>10
Adenovirus	Adenovirus 7 (AdV 7)	**0.02**	1.3	4.5-33	Inactive (AdV2)	>100	No data	>10 (AdV2)
Polyoma	BK Virus (BKV)	**0.13**	115	>200	Inactive	>200	No data	No data
	JC Virus (JCV)	**0.045**	>0.1	No data	Inactive	No data	No data	No data
Papilloma	Human Papillomavirus 11 (HPV-11)	**17**	716	Inactive	No data	Inactive	No data	No data
Pox	Variola	**0.1**	27	No data	No data	No data	No data	No data
	Vaccinia	**0.8**	46	>392	Inactive	>144	No data	No data

EC_{50} = concentration in µM required to reduce viral replication by 50% *in vitro*.
Data are compiled from multiple sources and include multiple materials and methodologies.
*Valganciclovir is rapidly converted to ganciclovir *in vivo*. Therefore, ganciclovir is the relevant compound for cell activity studies.
Source: Chimerix, Inc.

Exhibit 11
Competitive Landscape Cross-Comparison

a) Competitive Landscape: Marketed Products

Company	Roche/ Fresenius	Roche	Gilead	AstraZeneca/ Hospira	CSL Behring
Generic Name	ganciclovir	valganciclovir	cidofovir	foscarnet sodium for injection	Pooled CMV hyperimmunoglobulin
Brand Name	Cytovene	Valcyte	Vistide	Foscavir	Cytogam
Stage of Development	Marketed	Marketed	Marketed	Marketed	Marketed
Spectrum/Relative Potency			Broadest spectrum against dsDNA viruses		
Route of Administration/Dosage	IV	Oral once or twice a day with CrCl adjustments	IV	IV	IV
Indication	CMV treatment in HSCT and SOT; standard of care for CMV prevention in SOT; too myelosuppressive for use in HSCT	Standard of care for CMV prevention in SOT; too myelosuppressive for use in HSCT	CMV retinitis in HIV patients; Off-label standard of care for BK/AdV; no label in transplant	CMV retinitis and acyclovir-resistant HSV; no label in transplant but used off-label in ganciclovir resistant SOT recipients	CMV prevention in SOT; used in combination with ganciclovir in high risk SOT recipients
Resistance	Yes	Yes	Activity against ganciclovir-resistant CMV	Activity against ganciclovir-resistant CMV	Not applicable
Safety Profile	Myelosuppressive, but less myelotoxic than Valcyte; nephrotoxicity; resistance				
Nephrotoxicity	Not indicated in low CrCl or hemodialysis	Not indicated in low CrCl or hemodialysis	Black box warning	Black box warning	
Myelosuppression	Black box warning	Black box warning	Black box warning	Anemia and neutropenia	
Seizures	None	None	None	Black box warning	None
Increased Monitoring	Yes	Yes	Yes	Yes	
Dosing Hepatic Impairment	Not studied	Contraindicated in liver transplant; not studied in hepatic impairment	Not studied	Not studied	
Elderly Dosing	Precaution: dose reduction	Precaution: dose reduction	Not studied	Precaution: dose reduction	
Pediatric Label	No label/dose	4 months to 16 years old, in SOT only	No label/dose	No label/dose	
U.S. Pricing	$8,000-$10,500 per 3 or 4 months	$11,500-$23,000 per 3 or 6 months	$1,000-$6,000 per course	$30,000 per course	$30,000 - $44,000 per course
U.S. Sales, 2012	$25 million	$400 million	~ $10 million	~ $10 million	$30 million

Sources: Company reports, IMS Health, Bloomberg, zenRx.org, and William Blair & Company, L.L.C.

Exhibit 11
Competitive Landscape Cross-Comparison

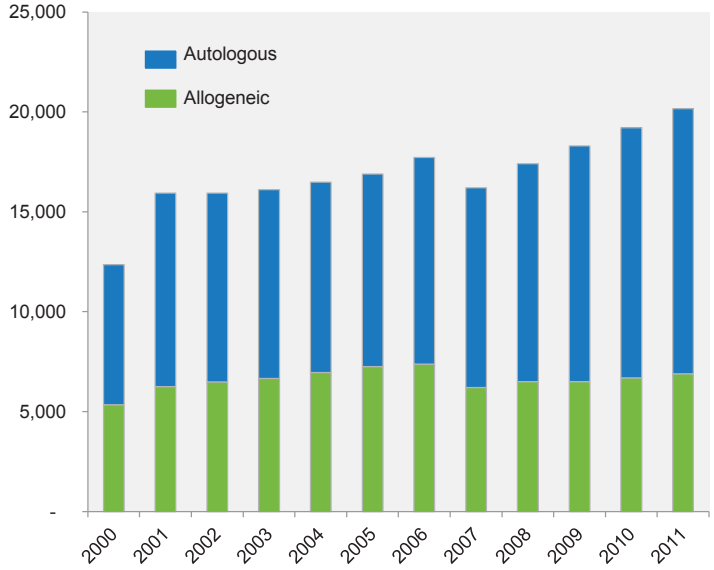
b) Competitive Landscape: Clinical Candidates

Company	Chimerix	Merck/AiCuris	Astellas/Vical	ViroPharma
Generic Name	**CMX001 (lipid conjugate of cidofovir)**	**letermovir**	**ASP0113**	**maribavir**
Brand Name	**--**	**--**	**TransVax**	**--**
Stage of Development	**Pre-Phase III**	**Pre-Phase III**	**Pre-Phase III**	**Phase II**
Spectrum/Relative Potency	Broadest spectrum against dsDNA viruses, with much-improved potency over Vistide			
Route of Administration/Dosage	Oral twice a week	Oral, QD	SubQ once every three months	Oral, BID
Indication	Pursuing CMV prevention indication in HSCT. Then expand to other viruses: AdV, BK, etc.	Pursuing CMV prevention indication in HSCT	Pursuing CMV prevention in HSCT and SOT	Pursuing CMV treatment in patients resistant or refractory to ganciclovir/valganciclovir or Foscarnet
Resistance	No resistance discovered so far; activity against ganciclovir-resistant CMV	?	Not applicable	Activity against ganciclovir-resistant CMV
Safety Profile	Diarrhea leading to 10% discontinuation in Phase II; management plan in Phase III	No notable adverse side effects from Phase II studies		
Nephrotoxicity	None	None	None	None
Myelosuppression	None	None	None	None
Seizures	None	None	None	None
Increased Monitoring				
Dosing Hepatic Impairment	Dose adjustment not needed	Dose adjustment not needed	Dose adjustment not needed	Dose adjustment not needed
Elderly Dosing	Dosed to age 78			
Pediatric Label	Studied in neonates; Phase III study in pediatric patients planned			
U.S. Pricing	We model $50,000 per 14-week course			

Sources: Company reports and William Blair & Company, L.L.C.

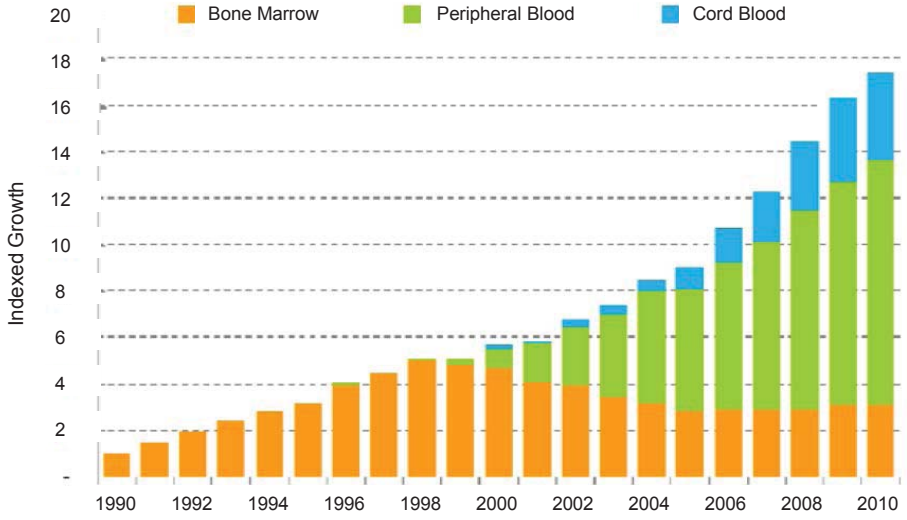
Exhibit 12
Growth of U.S. HSCT Market

a) Growth of U.S. HSCT Market, by Donor Type



Source: Chimerix, Inc.

b) Growth of U.S. HSCT Market, by Stem Cell Source



Source: Fate Therapeutics

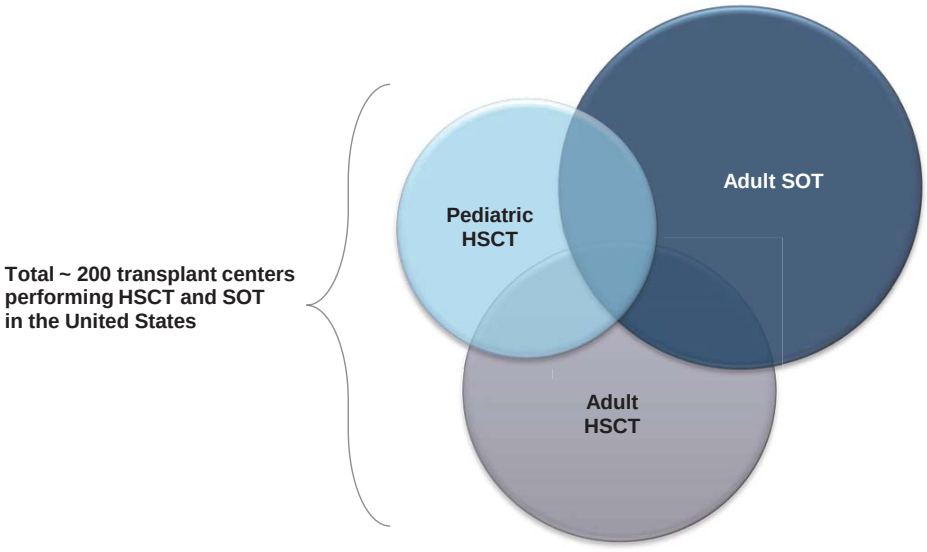
The Viral Prevention Market in HSCT Is Multibillion-Dollar and Highly Concentrated

Consistent Growth of the HSCT Market and Faster Pace for Allogeneic Transplants
As illustrated in exhibit 12a, the annual growth for U.S. HSCT was 4% for the 10-year span between 2001 and 2011. The allogeneic transplants particularly have been growing faster. And as shown in exhibit 12b, which breaks down the transplant cases by stem cell source, peripheral blood and cord blood are growing at a faster pace, whereas bone marrow use has been decreasing over the years. The faster-growing allogeneic transplant in HSCT will heighten the need for minimizing post-transplant complications, including viral infections, an overall trend favorable for CMX001.

Physicians Prefer a Prevention Regimen for HSCT
In the compassionate use program conducted by Chimerix as detailed below, about 30% of patients were infected with two or more dsDNA viruses, although the most prevalent was CMV. Physicians we consulted prefer a broad-spectrum prevention regimen for HSCT to have peace of mind and believe that a suitable agent with the optimal combination of potency, spectrum, and safety for this purpose should become the standard of care.

The Target Market Is Highly Concentrated, Enabling Efficient Marketing and Higher Margin
The market is highly concentrated, as illustrated in exhibit 13; it is estimated that 200 institutions in the United States perform HSCT and SOT transplants. As a result, a commercial team of 50 people would be sufficient to market CMX001 in North America.

Exhibit 13
Chimerix, Inc.
Target Transplant Market for CMX001 Is Highly Concentrated



Source: Chimerix, Inc.

Chimerix plans to commercialize CMX001 on its own in the United States and Canada, and is contemplating strategies in Europe. We project that Chimerix will license out the commercial rights to CMX001 to a partner and receive 30% royalties on sales on the Continent. Further, we expect Chimerix licensing rights to CMX001 in territories outside the United States and Europe to maximize the commercial potential of CMX001.

Upside: Other Viruses; Prolonged Prevention; SOT and Other Territories

In our model, we only assume CMX001 sales in CMV prevention in the HSCT setting. We believe there are a number of scenarios in which CMX001 could be developed for substantial upside to our model.

Other Viruses: AdV, BKV, and More
Usually, one-third of HSCT patients are co-infected with CMV and other dsDNA viruses, such as AdV, BKV, and HSV; therefore, an antiviral agent such as CMX001 that could prevent all dsDNA viral infections and decrease the risk of these opportunistic infections could provide higher clinical and pharmacoeconomic benefits. In clinical practice, prevention with CMX001 could lead to less frequent monitoring and prevention across dsDNA families.

To take advantage of the high potency and broad spectrum of CMX001, Chimerix intends to develop CMX001 against other viral infections as well, most prominently AdV and BKV, as discussed below.

Prolonged Prevention
In SOT, the length of prevention with ganciclovir has been increased from 3 months to 6 months and in some cases 12 months, and studies have shown that longer prevention resulted in better outcomes. We believe it is likely that this trend turns out to be similar in HSCT as well, especially in high-risk patients. We currently assume 14 weeks in our model, which is the duration to be evaluated in Phase III SUPPRESS study.

CMX001 Could Be Best-in-Class for Prevention in SOT
Although valganciclovir is already approved for CMV prevention in SOT, CMX001 might provide important incremental advantages in higher potency, which could translate into better efficacy, a broader spectrum covering not only CMV, a more convenient dosing schedule (twice a week versus once daily), and, most importantly, a much better tolerability profile devoid of myelosuppression. In particular, we note that BKV infection is prevalent in kidney transplant recipients and that there is currently no treatment for it; CMX001 could uniquely fill the void in settings like this. The SOT market is slightly larger than the HSCT market; expansion into the SOT setting could provide meaningful upside to our estimate for CMX001.

Sales Outside the United States and Europe Are Upside as Well
We have only included revenues from the United States and Europe in our model. Sales outside these areas would be upside to our valuation.

CMX001 Clinical Program

In exhibit 14, we list completed, continuing, and proposed clinical studies for CMX001.

Exhibit 14
Chimerix, Inc.
Summary of CMX001 Clinical Program

Study		Patients	Status
Phase I Clinical Pharmacology Studies			
CMX001-102	Phase I: Dose-escalation study in healthy volunteers	84	Completed
CMX001-103	Phase I: Food effect and bioequivalence study	24	Completed
CMX001-106	Phase I: Hepatic impairment study	25	Completed
CMX001-108	Phase I: QTc study	56	Ongoing
CMX001-112	Phase I: Human AME study (mass balance)	6	Completed
CMX001-113	Phase I: DDI study with midazolam	20	Completed
CMX001-114	Phase I: Food effect study	24	Ongoing
Placebo-Controlled Studies			
CMX001-104	Phase Ib: SOT and HSCT recipients with BK virus infection	28	Completed
CMX001-201	Phase II: CMV prevention in adult HSCT recipients	230	Completed
CMX001-202	Phase II: AdV preemption in pediatric and adult HSCT recipient	48	Ongoing; data in second half 2013
CMX001-301	Phase III SUPPRESS: CMV prevention in adult HSCT recipients	450	Initiated April 2013; data first half 2015
CMX001-311	Phase III: CMV prevention in pediatric HSCT recipients	TBD	Likely to be initiated by year-end 2013
Other studies			
EINDs	Emergency INDs for compassionate use, treating CMV, AdV, BKV, EBV, HSV, JCV, HHV, VZV, and HPV	>230	Completed; transitioned to Study 350
CMX001-350	Open-label study, focusing on treating CVM, HSV, and AdV	210	Completed
CMX001-333	Three-year follow-up of patients from the Phase III studies	TBD	

Sources: Chimerix, Inc., ClinicalTrials.gov, and William Blair & Company, L.L.C.

Phase II Study 201: Establishing the Final Dose of 100 mg BIW for Phase III

Design and inclusion criteria. Study 201 was a multicenter, randomized, double-blind, placebo-controlled, dose-escalating study of CMX001 for CMV prevention in high-risk HSCT recipients. The Phase II study enrolled 230 adult HSCT recipients, randomized to various doses of CMX001 and placebo. These patients all received allogeneic HSCT, and some were transplanted with cord blood (10% of study population) or a mismatched donor (25% of study population), rendering them higher risk. The patients were also seropositive for CMV (R+) at baseline; as noted above, R+ allogeneic patients have a risk of CMV infection of 80%.

Dosages studied. CMX001 doses of 40 mg, 100 mg, and 200 mg once weekly (QW) were evaluated. The 100 mg and 200 mg doses were also evaluated on a twice-weekly (BIW) schedule. Patients were treated for 9-13 weeks and followed up for 8 weeks; treatment was initiated within the first four weeks after the transplant.

Endpoints. The primary endpoint was a composite endpoint for prevention failure, defined as 1) incidence of CMV disease at any time during therapy or 2) CMV DNA higher than 200 copies per milliliter (mL) at the time of the last dose of study drug.

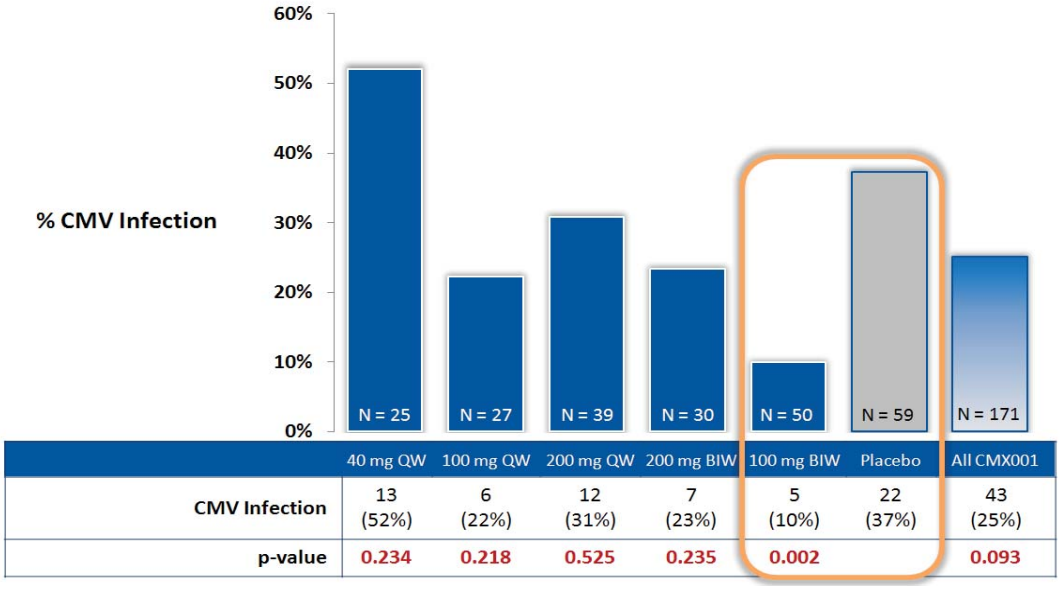
Efficacy: relative 73% reduction in prevention failure and selection of 100 mg BIW dose to advance into Phase III. As illustrated in exhibit 15, all CMX001 doses except for the lowest 40 mg QD dose exhibited antiviral activity compared with placebo. Although both 100 mg BIW and 200 mg BIW were efficacious, the 100 mg BIW dose led to much less diarrhea. Therefore, the 100 mg BIW dose was chosen to advance into Phase III studies.

At the 100 mg BIW dose, 10% (5/50) of patients had prevention failure, versus 37% (22/59) for placebo (p=0.002). Of the five patients who were characterized as prevention failures, one developed CMV disease and four needed preemption therapy because CMV DNA reached more than 200 copies per mL.

For the 41 patients who were CMV PCR negative on the first day of dosing, meaning that the patients were not actively infected with CMV at that time though they harbored the virus, 0% (0/41) of patients on the CMX001 arm versus 32% (15/47) of patients on the placebo arm developed CMV over 1,000 copies/mL (p<0.001).

Exhibit 15
Chimerix, Inc.
Study 201 Results: Efficacy

a) Primary Endpoint: Percent CMV Infection

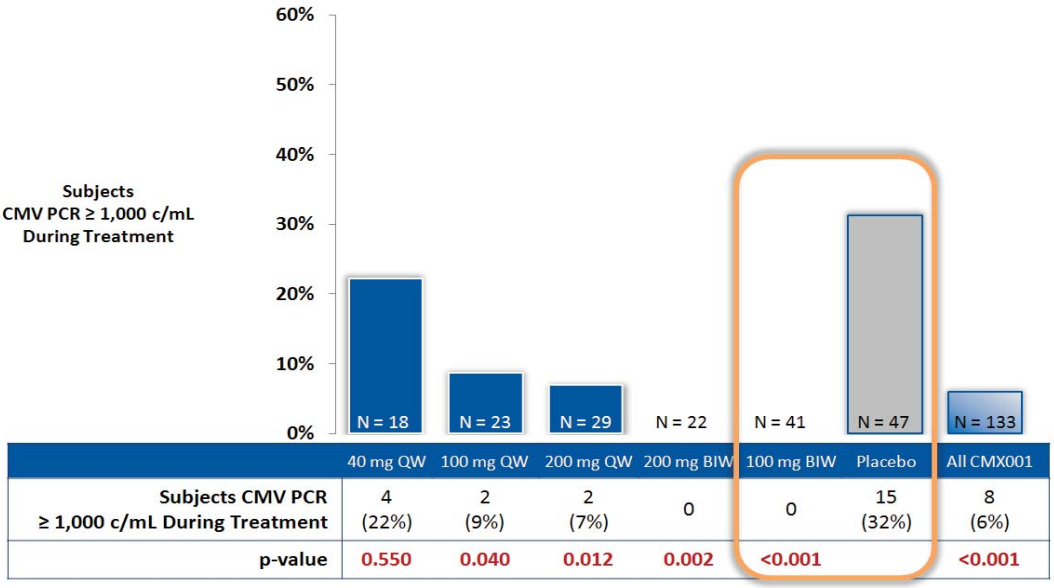


	40 mg QW	100 mg QW	200 mg QW	200 mg BIW	100 mg BIW	Placebo	All CMX001
CMV Infection	13 (52%)	6 (22%)	12 (31%)	7 (23%)	5 (10%)	22 (37%)	43 (25%)
p-value	0.234	0.218	0.525	0.235	0.002		0.093

Sources: Chimerix, Inc., and American Society for Blood and Marrow Transplantation (BMT Tandem) 2012 Meeting

Exhibit 15 (cont.)
Chimerix, Inc.
Study 201 Results: Efficacy

b) Secondary Endpoint: Prevention of CMV PCR > 1000 c/mL

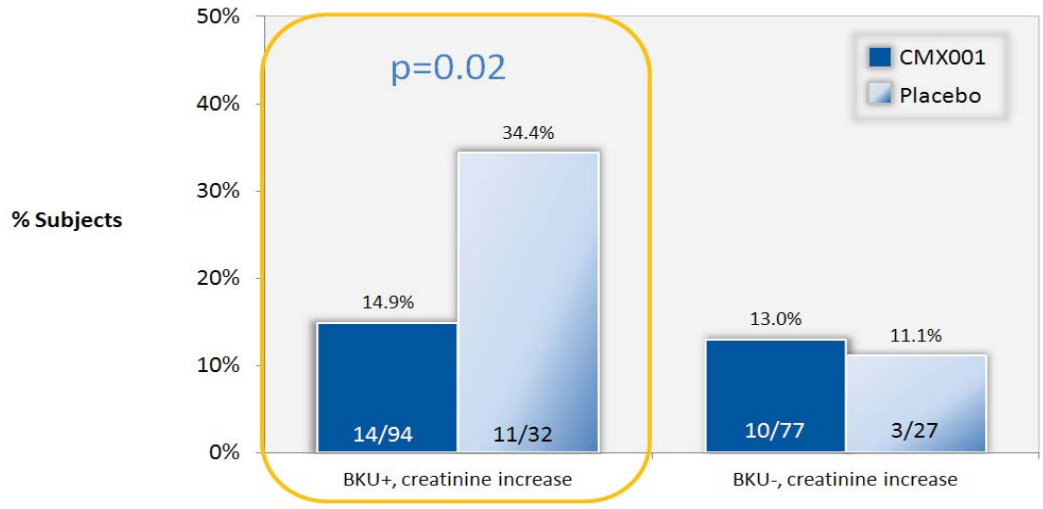


	40 mg QW	100 mg QW	200 mg QW	200 mg BIW	100 mg BIW	Placebo	All CMX001
Subjects CMV PCR ≥ 1,000 c/mL During Treatment	4 (22%)	2 (9%)	2 (7%)	0	0	15 (32%)	8 (6%)
p-value	0.550	0.040	0.012	0.002	<0.001		<0.001

Sources: Chimerix, Inc., and American Society for Blood and Marrow Transplantation (BMT Tandem) 2012 Meeting

Exploratory efficacy against BKV. As illustrated in exhibit 16, CMX001-treated patients who were BKV positive had improved renal function over placebo-treated patients. This exploratory efficacy is encouraging for further studies of CMX001 against BKV, not only in HSCT recipients, but also in SOT recipients, particularly kidney transplant patients.

Exhibit 16
Chimerix, Inc.
Study 201 Results: Percent of Patients With BKV Demonstrating Change in Serum Creatinine



Sources: Chimerix, Inc. and International Immunocompromised Host Society (ICHS) 2012 Meeting

Safety: diarrhea is the key adverse event. As summarized in exhibit 17a, the incidence of adverse events from the CMX001 100 mg BIW arm is comparable to that of placebo. The most prominent adverse event associated with CMX001 treatment is diarrhea; 10% on CMX001 discontinued because of gastrointestinal adverse events, versus 3% for placebo, although overall discontinuation for CMX001 is 36%, lower than the 46% for the placebo arm. For 200 mg BIW, a clear dose effect on diarrhea was observed; 60% dropped out, mostly because of diarrhea.

Exhibit 17
Chimerix, Inc.
Study 201 Results: Safety

a) Frequency and Severity of Adverse Events (AEs)

	CMX001					Placebo
	40 mg QW N = 25	100 mg QW N = 27	200 mg QW N = 39	200 mg BIW N = 30	100 mg BIW N = 50	N = 59
At least one AE	25 (100%)	27 (100%)	39 (100%)	30 (100%)	**50 (100%)**	**58 (98%)**
Grade 3 AE	6 (24%)	10 (37%)	15 (39%)	11 (37%)	**19 (38%)**	**25 (42%)**
Grade 4 AE	4 (16%)	3 (11%)	6 (15%)	8 (27%)	**7 (14%)**	**4 (7%)**
Grade 5 AE	2 (8%)	0	3 (8%)	4 (13%)	**5 (10%)**	**5 (9%)**
AEs leading to withdrawal	15 (60%)	9 (33%)	15 (39%)	18 (60%)	**18 (36%)**	**27 (46%)**

QW: Once-weekly; BIW: Twice-weekly

Sources: Chimerix, Inc. and American Society for Blood and Marrow Transplantation (BMT Tandem) 2012 Meeting

b) Safety and Tolerability Profile

	CMX001 100 mg BIW N = 50	Pooled Placebo N = 59
Total GI AEs Leading to Discontinuation	**5 (10%)**	**2 (3%)**
Abdominal Pain	1 (2%)	0
Diarrhea	2 (4%)	0
Nausea	1 (2%)	2 (3%)
Vomiting	1 (2%)	0
Subjects with Abnormal ALT		
> 3 x – 5 x ULN	10 (20%)	4 (7%)
> 5 x – 10 x ULN	4 (8%)	4 (7%)
> 10 x ULN	2 (4%)	1 (2%)

ULN: Upper Limit of Normal; ALT: Alanine aminotransferase
Sources: Chimerix, Inc. and American Society for Blood and Marrow Transplantation (BMT Tandem) 2012 Meeting

SMMP to decrease incidence of diarrhea. Upon the observation of severe diarrhea, a safety monitoring and management plan (SMMP) was incorporated in the latter cohorts of Study 201, which led to lower dropout rates later in the study. The SMMP will also be implemented in the Phase III study SUPPRESS. With the SMMP, investigators can interrupt study medication when a patient presents GI symptoms and reinitiate after symptoms are relieved.

Reversible ALT elevation also observed. Dose-dependent, transient alanine aminotransferase (ALT) elevation was observed with CMX001 compared with placebo, as summarized in exhibit 17b. For the 100 mg BIW dose, 32% had ALT greater than 3 times the ULN (upper limit of normal), versus 16% for placebo. Such ALT elevations usually returned to baseline levels following completion of

therapy and were not associated with increases in aspartate aminotransferase (AST) or bilirubin; most were mild or moderate in intensity. We note that a concurrent increase of ALT/AST and bilirubin is an indication of drug-induced liver damage.

Importantly, no myelosuppression or nephrotoxicity is seen with CMX001 treatment, a major advantage over cidofovir, ganciclovir, and valganciclovir.

EINDs

A single-patient emergency investigational new drug (EIND) application for an investigational drug candidate can be granted to the treating physician if the drug candidate is urgently needed for the patient's serious or life-threatening condition, no satisfactory alternative therapy is available, and the patient cannot receive the product through any existing clinical trials or expanded access programs.

Aside from the Phase I and II studies sponsored by Chimerix, more than 230 patients have been treated with CMX001 at more than 80 centers under EINDs or foreign equivalent regulations for severe, life-threatening dsDNA infections, including CMV, AdV, BKV, EBV, JCV, HHV-6, HHV-8, HSV-1 and HSV-2, VZV, HPV, moscullum, and vaccinia (smallpox). The average dosing period in the EIND cases is 11 weeks. Most of the patients treated under the EINDs were HSCT and SOT recipients.

Study 350; Data Later in 2013

Study 350 is a multicenter, open-label clinical study of CMX001 in 142 adults and 68 pediatric patients in 36 U.S. transplant centers with severe, life-threatening dsDNA viral infections. Patients must have failed all other available therapies to qualify for this study. The age of patients ranged from 1 to 78, and viral infections included CVM, HSV, EBV, AdV, BKV, JCV, and HHV6. In Study 350, 28% of patients were co-infected with at least two dsDNA viruses. Treatment duration averaged eight weeks. Data from Study 350 is expected to be presented later in 2013.

Study 202: Preemptive Therapy for AdV in HSCT Recipients; Data in Second Half 2013

Study 202 is a multicenter, randomized, placebo-controlled study evaluating CMX001 as a preemptive therapy for AdV infections in 48 adult and pediatric HSCT recipients. Although the incidence of AdV infection is much lower than that of CMV (5%-7%, versus 40%-80% for CMV), AdV carries a much higher mortality rate of 80% (versus 10%-20% for CMV). Chimerix initiated Study 202 in June 2011 in HSCT recipients with AdV infections and finished enrollment of 48 target patients in December 2012. We expect final data to be reported during the second half of 2013.

HSCT patients with asymptomatic AdV viremia were enrolled and randomized to receive CMX001 100 mg BIW, 200 mg QW, or placebo for 12 weeks as preemptive therapy, followed by a four-week follow-up period. For pediatric patients weighing less than 50 kilograms, liquid formation of CMX001 at 2 mg/kg BIW or 4 mg /Kg QW was administered.

The primary endpoint is treatment failure rate, a composite of 1) progression to probable or definitive AdV disease and 2) increasing AdV viremia during randomized therapy that requires discontinuation from randomized therapy, CMX001 versus placebo. Patients who are considered treatment failures during the randomized phase are offered open-label treatment of CMX001.

Secondary endpoints include incidence and time to mortality, the proportion of patients with undetectable plasma AdV PCR, and the proportion of patients who have emergence or progression of CMV, EBV, and BKV viremia or disease during therapy.

Because of the low incidence of AdV infection, a prevention study would have required thousands of patients, which is impractical given the number of HSCT transplants taking place each year. As a result, a preemptive strategy is implemented for AdV infections in Study 202.

Overall Safety to Date Satisfactory: Exposure in More Than 800 Subjects
The current safety database includes about 800 subjects exposed to CMX001 in controlled and uncontrolled settings and supports advancement into Phase III studies.

We note that cidofovir carries a black box warning that includes renal toxicity, neutropenia, and the statement that "in animal studies cidofovir was carcinogenic, teratogenic, and caused hypospermia." This was based on a 26-week intravenous toxicology study in which female rats developed mammary tumors at high doses and included observation of inhibition of spermatogenesis in rats and monkeys. Chimerix observed similar effects in the preclinical studies for CMX001. We expect that CMX001 will carry a black box warning on animal carcinogenicity on its label if eventually approved but that it will not have the human renal toxicity and neutropenia warnings.

Phase III SUPPRESS Design
In exhibit 18, we compare and contrast the design of the SUPPRESS Phase III study with Study 201.

Design; similar patient population as Study 201. SUPPRESS plans to enroll and randomize 450 allogeneic HSCT recipients who are CMV seropositive (R+) 2-to-1 to CMX001 at 100 mg BIW, and placebo. The target population is considered at risk because R+ patients already harbor latent CMV. The double-blind, placebo-controlled study will be conducted at 30-40 sites in the United States and Canada, with significant site overlap with Study 201. Further, higher risk patients, such as cord blood transplant and transplant from mismatched donors, will be included as well.

Primary endpoint. The primary endpoint is also similar to that of Study 201: 1) CMV disease or 2) initiation of preemptive therapy triggered by a positive test for CMV in the blood (viremia). The study is powered at 85%, with one-sided p value of 0.025, to detect 50% reduction in treatment failure for CMX001 versus placebo. The design assumes the prevention failure rate for the placebo arm is 30%. It also assumes that 16% of enrolled patients will drop out, become lost to follow-up, or die from non-CMV-related causes.

The major difference lies in the definition of when to initiate preemptive therapy. There is a clear definition of when to initiate preemption therapy in SUPPRESS. If the patient has CMV DNA over 1,000 copies per mL, the patient should go on preemptive therapy with another antiviral compound. If the CMV DNA copies are less than 1,000 but higher than 150 and the patient meets certain specified criteria in the protocol, such as being higher risk, preemptive therapy should be initiated. As summarized in exhibit 18, the threshold on initiating preemptive therapy in Study 201 was 200 copies per mL.

Timing of initiation of therapy, duration of therapy, and length of follow-up are also different. In Study 201, the initiation of CMX001 therapy was averaged on day 24. In SUPPRESS, the initiation will be earlier; 50% of patients will be initiated with prevention therapy within day seven, and 90% will be initiated therapy within two weeks. We believe earlier initiation of prevention therapy should lead to better suppression of the latent viruses and might lead to better clinical outcomes than those in Study 201.

The duration of therapy in Study 201 was 9-13 weeks, while in SUPPRESS it will be uniform at 14 weeks. Further, in Study 201, patients were followed up for eight weeks following the end of therapy, while in SUPPRESS patients will be followed up for another 10 weeks through week 24. We note that in Study 201 there is one late relapse during the eight-week follow-up period, which is low. As a result, a slightly longer follow-up period should not be disadvantageous to SUPPRESS outcomes, in our view.

Secondary endpoints will contribute to pharmacoeconomic argument. Secondary endpoints include quality of life; use of healthcare resources, including hospital days, and various procedures performed, among others. Further, real-time assessment of symptoms from other viruses will also be performed, which include delirium, pneumonitis, encephalitis, hemorrhagic cystitis, gastroenteritis, retinitis, nephritis, and hepatitis.

Phase III pediatric study (Study 311) design submission to the FDA planned for second half 2013. As discussed above, this study will be used for eventual full approval of CMX001 for CMV prevention in HSCT. Chimerix has not committed to the timing of initiating this study.

Managing diarrhea. In transplant patients, diarrhea could arise from a number of sources, including conditioning regimens, antibacterial infections, other concomitant medicines, or GvHD. A decrease in serum albumin from baseline could prove useful as an additional marker for distinguishing drug-related diarrhea from diarrhea of other sources. The SUPPRESS study will implement the SMMP to manage tolerability and decrease discontinuation as a result of gastrointestinal side effects.

Exhibit 18
Chimerix, Inc.
Comparsion of Study 201 and SUPPRESS

	Study 201 Phase II	SUPPRESS Phase III
Inclusion criteria	R+ HSCT adult, including high-risk patients (10% cord blood transplant, 25% mismatched donors); also included a few PCR+ patients.	R+ HSCT adult, including high-risk patients; but with no PCR+ patients at baseline.
Dosage	Dose escalation to 100 mg BIW and 200 mg QW and placebo; found 100 mg BIW as the optimal dose. About 25-50 patients per dose arm.	n=450, 2:1 to 100 mg BIW and placebo. Dosing with low fat meal.
Timing of dose initiation	Average intiation was day 24 after transplant; timing of initiation was varied; initiation of required demontration of engraftment.	50% of patients could start dosing at day 7, and 90% of patients should start dosing within 2 weeks. Earlier dosing might increase efficacy of prevention.
Treatment duration	9-13 weeks, then 8 weeks follow-up.	14 weeks, then follow-up until week 24.
Primary endpoint/ Definition of prevention failure	1) CMV disease or 2) initiation of preemptive therapy.	1) CMV disease or 2) initiation of preemptive therapy.
When to initiate preemptive treatment	When CMV DNA exceeded 200 copies/mL; or at physician discretion.	Strict protocol to follow: CMV DNA exceeds 1,000 copies/mL in any patient, or over 150 copies/mL in high-risk patients.
Efficacy on primary endpoint	10% for 100 mg BIW vs. 37% placebo, p=0.002, relative 73% reduction in prevention failure.	Powered 85% to detect 50% reduction in prevention failure.
Efficacy: Study 201 data based on SUPPRESS inclusion criteria	22% for 100 mg BIW vs. 46% placebo, relative 52% reduction in prevention failure. This models the "worst-case scenario."	---
Safety	Diarrhea is the major AE; 10% of 100 mg BIW arm, 60% of 200 mg BIW arm dropped out due to GI AE, versus 3% for placebo.	Management program to reduce GI dropout.

Sources: Chimerix, Inc., ClinicalTrials.gov, and William Blair & Company, L.L.C.

Handicapping the Probability of Success of SUPPRESS: We Estimate 80%

As summarized in exhibit 18 and the previous section, there are a number of similarities and differences between Study 201 and SUPPRESS. The inclusion criteria are similar; only high-risk R+ HSCT recipients are enrolled. Further, therapy initiation is earlier and dosing duration is longer in SUPPRESS as compared with Study 201, which might lead to better outcomes.

When applying the SUPPRESS primary endpoint and inclusion criteria to Study 201, 22% of CMX001-treated patients met treatment failure criteria, versus 46% of placebo patients—a 52% relative reduction, as illustrated in exhibit 19. This analysis constitutes a potentially worst-case scenario and supports the SUPPRESS design and powering.

As a result, we assign an 80% probability of success to SUPPRESS.

Exhibit 19
Chimerix, Inc.
Study 201 Results Based on SUPPRESS Phase III Primary Endpoint and Inclusion Criteria

Cohorts	Failures ("Worst Case")	Included in Analysis, N
CMX001		
40 mg QW	8 (40%)	20
100 mg QW	9 (38%)	24
200 mg QW	11 (34%)	32
200 mg BIW	9 (33%)	27
100 mg BIW	**10 (22%)**	**45**
Placebo	**24 (46%)**	**52**

Sources: Chimerix, Inc. and William Blair & Company, L.L.C.

Past Maribavir Failure Should Not Have Any Negative Impact on SUPPRESS Prospect

A Phase III HSCT CMV prevention study with maribavir conducted by ViroPharma failed in 2009. Maribavir is a once-daily oral drug targeting CMV protein kinase UL97. In Phase II studies, maribavir demonstrated significant reduction in CMV disease incidence versus placebo.

The main reasons for the maribavir Phase III failure include 1) the chosen dose in Phase III was likely too low; 2) the primary endpoint was CMV disease reduction 6 months post transplant; this is different from that of Study 201 or SUPPRESS which is a composite endpoint; 3) the maribavir Phase III study excluded high risk patients, and the CMV disease incidence was too low in the placebo arm (only 5%) for maribavir to demonstrate a significant relative reduction; 4) the initiation of treatment was on day 24, which some experts speculate was a little late for maribavir to come into action to prevent CMV reactivation. We note that while in CMX001's Study 201 the initiation of prevention therapy was on day 24 as well, in SUPPRESS 90% of patients will initiate prevention therapy within two weeks.

Therefore, we do not believe a past failure in the CMV prevention setting should have any negative impact on the prospect of SUPPRESS.

CMX001 Regulatory Strategy and Outlook

Fast-Track Designation for Multiple Viruses
Chimerix has received fast-track designation from the FDA for the CMV, AdV, and smallpox indications for CMX001.

Accelerated Approval Possible With SUPPRESS and Existing Database
Chimerix believes that a single SUPPRESS study, if adequately successful, plus the existing efficacy and safety database from the previous controlled and open-label studies, as well as the preclinical studies, could be sufficient to file a new drug application in the United States for an accelerated, or conditional, approval of CMX001 in CMV prevention. We expect SUPPRESS to release top-line data in early 2015, with a potential new drug application filing later in the year.

Full Approval in CMV Prevention Might Need a Phase III Pediatric Study
A second Phase III study to be conducted after approval could suffice full approval. Such a study could be a pediatric HSCT study for CMV prevention, or evaluation of CMX001 in BKV infection in both HSCT and SOT recipients. We expect Chimerix to update on the strategy for the second Phase III study during 2013 or early 2014.

Other Markets
Chimerix plans to seek regulatory approval in Canada and Europe for a label similar to that in the United States for CMX001. Discussions with various regulatory authorities are continuing.

Competitive Landscape for CMX001 in HSCT

There are two major competitive agents in development for CMV prevention: 1) letermovir from partners Merck and AiCuris and 2) TransVax from partners Astellas and Vical. Both letermovir and TransVax are entering Phase III studies, perhaps a few months behind CMX001. We note that both letermovir and TransVax are specific for CMV, while CMX001 is much broader in spectrum coverage. Further, Phase II studies for both agents excluded high-risk patients, while Study 201 of CMX001 included high-risk patients up to 35% of the study population. We expect to continue to monitor the progress of the competitive agents; the Phase III designs and data will determine relative strengths of the profiles of these agents, which will consequently impact their respective market share once commercialized.

Letermovir: An Oral, Once-Daily Therapy for CMV Prevention
Letermovir is a first-in-class agent that inhibits the formation and release of CMV viral particles via targeting the terminase complex. In October 2012, AiCuris GmbH, a spinout of Bayer Healthcare, granted worldwide rights to its human CMV portfolio of assets, including letermovir, to Merck. Letermovir has received orphan drug status for the prevention of CMV in both the United States and the European Union.

AiCuris agreement with Merck. In October 2012, AiCuris licensed letermovir (now MK-8228), as well as three additional CMV candidates, to Merck for an up-front payment of €110 million; AiCuris is eligible to receive an additional €332.5 million in milestone payments as well as royalties on worldwide sales of the products. Merck will be responsible for all development and commercialization costs of letermovir.

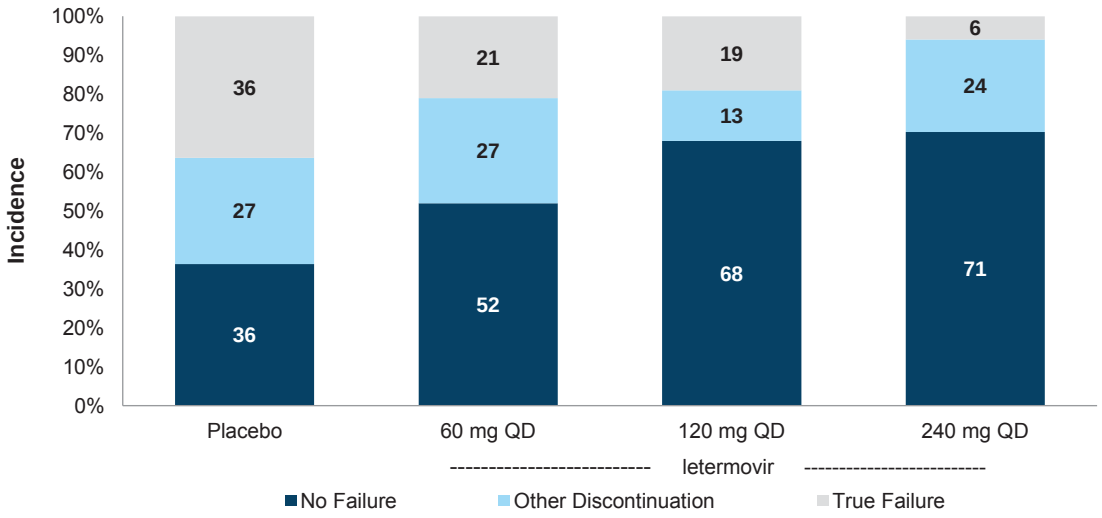
Letermovir meets co-primary endpoints in Phase IIb proof-of-concept study. In January 2010, AiCuris initiated a Phase II study evaluating letermovir versus placebo in 133 CMV-seropositive (R+) allogeneic HSCT recipients. We note that unlike Study 201 with CMX001, high-risk patients,

such as mismatched or cord blood transplant recipients, were excluded from this letermovir Phase II study. Patients enrolled in the study were randomized to receive one of three doses of letermovir (60 mg, 120 mg, and 240 mg QD) or placebo for 12 weeks. The co-primary objectives of the study were antiviral activity, defined as the incidence and time to onset of CMV prophylaxis failure (viral load above 42 DNA copies per milliliter) or CMV end-organ disease; secondary endpoints included safety and tolerability.

In February 2012, AiCuris announced that the 120 mg and 240 mg letermovir cohorts met both primary endpoints with high statistical significance compared with placebo. In the primary full analysis population, the incidence of CMV failure (either prophylaxis failure or discontinuation) before day 84 was 29.4% (p=0.007) for the 240 mg cohort and 32.3% (p=0.014) for the 120 mg cohort, versus 63.6% for placebo; the incidence of failure in the 60 mg cohort was about 48%. AiCuris also reported that high-dose letermovir met the co-primary endpoint of time to onset of prophylaxis failure when compared with placebo (p=0.02). We summarize the results of the study in exhibit 20.

We note that the viral load cut off in the letermovir study is much lower than the 200 copies per mL used in CMX001's Study 201, which might explain why the placebo failure rate was as high as 64% in these low-risk patients, while the placebo failure rate was only 37% in high-risk patients in CMX001's Study 201.

Exhibit 20
Chimerix, Inc.
Incidence of CMV Prophylaxis Failure From AiCuris's Letermovir Phase II Study



Sources: AiCuris GmbH & Co. and William Blair & Company, L.L.C.

Letermovir was considered safe and well tolerated. The percentage of patients with treatment-emergent adverse events either related to treatment or leading to discontinuation were 17.3% and 25.5% for the pooled letermovir cohorts, versus 33.3% and 57.6% for the placebo group.

TransVax: A DNA Vaccine for CMV Prevention

TransVax is a DNA plasmid-based vaccine encoding two specific antigens of CMV—glycoprotein B (gB) and phosphoprotein 65 (pp65)—co-formulated with a proprietary poloxamer delivery system and benzalkonium chloride (BAK), an agent known to increase immunogenicity. The vaccine has been shown to elicit both a cellular response to pp65 and a humoral response to gB by generating anti-gB neutralizing antibodies. In 2011, Vical granted exclusive worldwide rights to develop and

commercialize TransVax to Astellas Pharma. Astellas plans to initiate a Phase III study evaluating TransVax in HSCT patients during second quarter 2013; an additional Phase II study to evaluate TransVax in SOT patients is also anticipated to start in 2013. TransVax has received orphan drug designation in the United States for the prevention of CMV infections in HSCT and SOT recipients.

Vical's licensing agreement with Astellas. In July 2011, Vical licensed TransVax to Astellas for an up-front payment of $25 million along with $105 million in additional development milestones. Under the terms of the agreement, Astellas will be responsible for further development and commercialization of TransVax, including all costs, and will provide Vical double-digit royalties on worldwide net sales of TransVax. The agreement also provides Vical with an option to co-promote TransVax in the United States. In 2012, Vical received a $10.0 million milestone payment from Astellas related to the planned TransVax Phase III study.

TransVax Phase II proof-of-concept study meets primary endpoint; subanalysis provides additional support. In January 2006, Vical initiated a Phase II study evaluating TransVax versus placebo in 80 R+ HSCT recipients. Patients were randomized 1-to-1 to receive a series of intramuscular injections of either TransVax (dosed at 5 mg/mL) or placebo. A total of four vaccinations were administered to patients—one before transplant and three after transplant at one, three, and six months. The primary objectives of the study were the safety of TransVax and the occurrence rate of clinically significant CMV viremia in recipients.

Analysis of the per protocol population of 74 unpaired recipients (six patients were excluded from the per protocol analyses for various reasons) showed that administration of TransVax delayed and decreased the occurrence of CMV reactivation episodes compared with placebo in HSCT recipients. Specifically, the median time to initial CMV reactivation was greater than 365 days for the TransVax cohort, versus 109.5 days for the placebo group (p=0.003), and at one year post-transplant, the proportion of recipients experiencing an occurrence of CMV reactivation was 32.5% for TransVax versus 61.8% for placebo, leading to a statistically significant reduction in the incidence of CMV viremia (p=0.008). In addition, treatment with TransVax reduced the number of CMV viremia episodes compared with placebo (p=0.017).

Treatment with TransVax also led to a reduction in preemptive CMV-specific antiviral therapy and the mean number of days on antiviral therapy, but the differences in both categories were not significant. Biomarker data showed that while TransVax increased T cell responses to pp65 compared with placebo (p=0.003) at one year post-transplant, but only moderately increased T cell responses to gB compared with placebo (p=0.75).

TransVax was found to be safe and well tolerated, with no differences between groups with respect to SAEs. Only one discontinuation (angioedema) was reported in the TransVax cohort. In addition, there was no difference in the time to engraftment, graft-versus-host disease (GvHD), secondary infection, or deaths between the two treatment arms.

Vical later performed a subanalysis of the study following the determination that seven patients (two in the TransVax cohort) enrolled in the Phase II study were never infected with CMV, despite positive antibody titers at screening. The subanalysis showed that TransVax had improved results relative to the original per protocol analysis over several categories, including a reduction in the number of new treatment starts (p=0.35). Vical attributed the phenomenon of non-infected, CMV-positive patients to the likelihood that patients received prior transfusions with CMV-positive blood products.

In exhibit 21, on the following page, we summarize key data from the Phase II TransVax study.

If we apply the composite endpoint for prevention failure to this Phase II study, we conclude that TransVax did not show a statistically significant difference from placebo. We note that the composite endpoint to evaluate prevention failure used in CMX001's Study 201 and the letermovir Phase II study was incidence of CMV disease and need to initiate preemptive therapy. If we apply this endpoint to the TransVax Phase II study, it appears that 55% of TransVax treated patients experienced prevention failure versus 71% of placebo. This result is not statistically significant.

Exhibit 21
Chimerix, Inc.
Results From TransVax Phase II Study

a) Initial Per Protocol Analysis: Per Protocol Population at One Year

	TransVax N=40	Placebo N=34	Percent Change	p-value
Occurrence of CMV reactivation (≥ 500 copies/mL), n (%)	13 (32.5%)	21 (61.8%)	-47%	0.008
Time to initial CMV reactivation (days), median	>365	109.5	--	0.003
Number of CMV reactivation episodes, n (%)				
0	27 (68%)	13 (38%)		
1	8 (20%)	14 (41%)		
2	4 (10%)	3 (9%)	--	0.017
3	1 (3%)	3 (9%)		
≥ 4	0	1 (3%)		
Duration of viremia (days)	10.6 (0-68)	19.4 (0-181)	-46%	0.071
Normalized (as a percentage of time on study), mean	4.9 (0-63)	7.6 (0-49)	-36%	0.042
Occurrence of initiating CMV-specific antiviral therapy, n (%)	19 (47.5%)	21 (61.8%)	-23%	0.145
Cumulative CMV-specific antiviral therapy, days (mean)	33.7 (0-316)	40.4 (0-212)	-17%	0.45
CMV-associated disease (GI and/or pneumonia), n (%)	3 (7.5%)	3 (8.8%)	-15%	1.00

Sources: Vical Inc. and William Blair & Company, L.L.C.

b) Subanalysis: Per Protocol Population Minus Seven Subjects at One Year

	TransVax N=38	Placebo N=29	Percent Change	p-value
Occurrence of CMV reactivation (≥ 500 copies/mL), n (%)	13 (34%)	21 (72%)	-53%	0.002
Duration of viremia (days)	11.2 (0-68)	22.8 (0-181)	-51%	0.026
Normalized (as a percentage of time on study), mean	5.2 (0-63)	9.0 (0-49)	-42%	0.013
Occurrence of initiating CMV-specific antiviral therapy, n (%)	19 (50%)	21 (72%)	-30%	0.035
Cumulative CMV-specific antiviral therapy, days (mean)	32.0 (7-323)	46.7 (26-212)	-31%	0.105
Normalized (as a percentage of time on study), mean	11.5 (0-88)	17.7 (0-71)	-35%	0.074

Sources: Vical Inc. and William Blair & Company, L.L.C.

CMX001 in Smallpox

Smallpox is designated a category A bioterror agent by the U.S. Centers for Disease Control and Prevention (CDC). Variola virus, a member of the orthopoxvirus family, infects only humans and causes smallpox. Both the National Institute of Allergy and Infectious Diseases (NIAID) and the Biomedical Advanced Research and Development Authority (BARDA) have granted funding to Chimerix to develop CMX001 as a medical countermeasure against smallpox. CMX001 demonstrated potent activity against variola virus in vitro as well as encouraging activity in related viruses in animal models. The animal efficacy rule allows the FDA to approve a drug candidate based on efficacy in animal models; studies of such agents in humans are deemed unethical.

NIAID contract completed. In September 2003, Chimerix was awarded $36.3 million from NIAID to support development of an oral drug against smallpox. The support led to identification of CMX001 as the lead product candidate for clinical development. The grant was concluded in 2011.

BARDA contract; base segment due May 31, 2013. Chimerix entered into a contract with BARDA, a division of the U.S. Department of Health and Human Services in the Office of the Assistant Secretary for Preparedness and Response, for the development of CMX001 against potential smallpox outbreaks in February 2011. The contract has been amended several times, and the base performance segment is extended through May 31, 2013. If successfully completed, the base performance segment could lead to $31 million in grants. Chimerix will present animal efficacy data to the FDA and negotiate certain aspects of the smallpox animal study plan to take into account recent FDA guidance on the animal efficacy rule before May 31, 2013. BARDA must notify Chimerix 30 days before the end of the base performance segment if it intends to exercise the first optional segment. If each of the optional segments is exercised, Chimerix could receive up to $75.8 million in expense reimbursement and $5.3 million in fees. It is unclear whether BARDA would exercise the option to continue supporting development of CMX001 against smallpox.

On the safety side, data collected to date from various studies of CMX001 should provide a sufficient database to support approval of CMX001 in smallpox countermeasures. If CMX001 gains approval under the animal efficacy rule, the U.S. government might stockpile CMX001 as a medical countermeasure for potential smallpox outbreaks.

CMX157 for HIV

CMX157 is a lipid-modified prodrug of tenofovir (brand name Viread), which was licensed to Merck in July 2012. CMX157 has a much improved potency and resistance profile than Viread, is dosed once weekly, and might be devoid of the renal and bone toxicities typically observed with Viread. A Phase I study in healthy volunteers demonstrated satisfactory pharmacokinetic properties and no safety issues.

Merck Licensing Deal Executed in July 2012
In 2012, Chimerix received an up-front payment of $17.5 million from Merck for exclusive worldwide rights to CMX157. According to the agreement, Chimerix is eligible to receive an additional $151 million in development and regulatory milestones plus royalties on future sales of CMX157. Merck is responsible for all costs associated with the development and commercialization of CMX157.

Additional information is available upon request.

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Exhibit 22
Chimerix, Inc.
Income Statement
(dollars in thousands)

	2011A	2012A	2013					2014E	2015E
			Q1E	Q2E	Q3E	Q4E	FY:13E		
Revenues									
CMX001 U.S. revenue	$0	$0	$0	$0	$0	$0	$0	$0	$0
CMX001 EU royalties	-	-	-	-	-	-	-	-	-
CMX157 royalties	-	-	-	-	-	-	-	-	-
Collaboration and licensing revenue	55	17,445	-	-	-	-	-	-	-
Contract and grant revenue	12,046	16,275	2,000	1,500	-	-	3,500	-	-
Total Revenues	**12,101**	**33,720**	**2,000**	**1,500**	**0**	**0**	**3,500**	**0**	**0**
Expenses									
COGS	-	-	-	-	-	-	-	-	-
R&D expense	27,695	27,821	6,955	7,999	8,638	9,502	33,094	39,748	46,092
SG&A expense	9,398	8,682	2,279	2,393	2,513	2,638	9,823	10,765	12,300
Total Operating Expenses	37,093	36,503	9,234	10,392	11,151	12,141	42,917	50,513	58,393
Operating income	(24,992)	(2,783)	(7,234)	(8,892)	(11,151)	(12,141)	(39,417)	(50,513)	(58,393)
Interest expense, net	(212)	(776)	(187)	(187)	(187)	(187)	(749)	(749)	(375)
Fair value adjustments to warrant liability	(385)	(847)	(270)	(2,000)	(250)	(240)	(2,760)	(720)	(720)
Other income/(expense)	-	-	-	-	-	-	-	-	-
Pretax income/(loss)	(25,589)	(4,406)	(7,692)	(11,079)	(11,588)	(12,568)	(42,927)	(51,983)	(59,487)
Other comprehensive gain/(loss)	(4)	2	-	-	-	-	-	-	-
Accretion of redeemable convertible preferred stock	(9,565)	(4,357)	-	-	-	-	-	-	-
Provision for income taxes/(income)	-	-	-	-	-	-	-	-	-
Net Income/(Loss)	**($35,154)**	**($8,763)**	**($7,692)**	**($11,079)**	**($11,588)**	**($12,568)**	**($42,927)**	**($51,983)**	**($59,487)**
GAAP EPS	**($23.17)**	**($5.71)**	**($4.96)**	**($0.43)**	**($0.45)**	**($0.49)**	**($2.20)**	**($2.03)**	**($2.32)**
Weighted average shares outstanding, diluted	1,517	1,534	1,552	25,508	25,518	25,528	19,527	25,553	25,593

Sources: Chimerix, Inc. and William Blair & Company, L.L.C. estimates

Exhibit 23
Chimerix, Inc.
Balance Sheet
(dollars in thousands)

	2011A	2012A	2013					2014E	2015E
			Q1E	Q2E	Q3E	Q4E	FY:13E		
Current assets									
Cash and cash equivalents	$19,525	$29,755	$22,096	$108,962	$96,035	$82,130	$82,130	$26,401	($35,386)
Accounts receivable	4,187	783	783	783	783	783	783	283	283
Inventories, net	-	-	-	-	-	-	-	-	-
Prepaid and other current assets	1,048	983	1,033	1,083	1,133	1,183	1,183	1,183	1,383
Deferred financing costs, current portion	64	33	33	533	483	433	433	233	33
Other current assets	-	-	-	-	-	-	-	-	-
Total current assets	24,824	31,554	23,945	111,361	98,434	84,529	84,529	28,100	(33,687)
Property and equipment, net of accumulated depreciation	561	407	432	457	482	507	507	607	707
Deposits	22	22	22	100	100	100	100	50	25
Deferred financing costs, less current portion	25	48	-	-	-	-	-	-	-
Other assets	-	-	-	-	-	-	-	-	-
Total assets	**25,432**	**32,031**	**24,399**	**111,918**	**99,016**	**85,136**	**85,136**	**28,757**	**(32,955)**
Current liabilities									
Accounts payable	4,120	1,964	1,964	1,964	1,964	1,964	1,964	3,964	5,964
Accrued liabilities	2,534	906	906	906	906	906	906	906	906
Loan payable, current portion	160	4,753	4,803	4,853	4,903	4,953	4,953	4,265	-
Deferred revenue	-	-	-	-	-	-	-	-	-
Total current liabilities	6,814	7,623	7,673	7,723	7,773	7,823	7,823	9,135	6,870
Other long-term liabilities	-	337	337	337	337	337	337	337	337
Deferred revenue, net of current	-	-	-	-	-	-	-	-	-
Loan payable, less current portion	2,441	9,867	9,867	8,494	7,121	5,748	5,748	-	-
Redeemable convertible preferred stock warrant liability	6,491	7,512	7,512	-	-	-	-	-	-
Total liabilities	**15,746**	**25,339**	**25,389**	**16,554**	**15,231**	**13,908**	**13,908**	**9,472**	**7,207**
Stockholders' equity	(93,680)	(101,031)	(108,713)	95,364	83,785	71,228	71,228	19,285	(40,162)
Convertible preferred stock	103,366	107,723	107,723	-	-	-	-	-	-
Total liabilities, convertible preferred and stockholders' equity	**25,432**	**32,031**	**24,399**	**111,918**	**99,016**	**85,136**	**85,136**	**28,757**	**(32,955)**

Sources: Chimerix, Inc. and William Blair & Company, L.L.C. estimates

Exhibit 24
Chimerix, Inc.
Statement of Cash Flows
(dollars in thousands)

	2011A	2012A	2013E	2014E	2015E
Net cash from operating activities					
Net Income (Loss)	($25,589)	($4,406)	($42,927)	($51,983)	($59,487)
Adjustments					
Depreciation and amortization	270	280	300	320	350
Non-cash interest expense	50	238	250	250	250
Amortization/accretion of premium/discount on investments	118	84	100	100	100
Share-based compensation costs	1,055	1,396	1,717	2,021	2,336
Deferred lease obligation	(4)	-	-	-	-
Fair value measurement of redeemable convertible preferred stock warrant liability	385	847	-	-	-
Change in Operating Assets and Liabilities					
Accounts receivable	(4,187)	3,404	-	500	-
Prepaid and other current assets and deposits	(442)	65	(200)	-	(200)
Accounts payable and accrued liabilities	2,065	(3,784)	-	2,000	2,000
Net cash used in operating activities	**(26,279)**	**(1,876)**	**(40,760)**	**(46,792)**	**(54,652)**
Cash flows from investing activities					
Purchase of property and equipment	(321)	(126)	(100)	(100)	(100)
Purchase of short-term investments	(13,640)	(9,907)	(50,000)	-	-
Sales of short-term investments	500	-	-	-	-
Maturities of short-term investments	7,100	5,894	-	40,000	13,117
Repayment of loan to officer	125	-	-	-	-
Net cash used in (provided by) investing activities	**(6,236)**	**(4,139)**	**(50,100)**	**39,900**	**13,017**
Cash flows from financing activities					
Proceeds from issuance of redeemable convertible preferred stock and warrants	45,000	-	-	-	-
Proceeds from exercise of stock options	30	14	40	40	40
Proceeds from loan payable	-	15,000	-	-	-
Debt discount	-	(75)	-	-	-
Repayment of loan payable	(1,965)	(2,601)	(3,919)	(6,436)	(4,265)
Stock offering and deferred financing costs	(249)	(24)	107,422	-	-
Net cash provided by financing activities	**42,816**	**12,314**	**103,543**	**(6,396)**	**(4,225)**
Cash balance (Beginning of Period)	**3,306**	**13,607**	**19,906**	**32,589**	**19,301**
Difference	10,301	6,299	12,683	(13,288)	(45,860)
Cash balance (End of Period)	**13,607**	**19,906**	**32,589**	**19,301**	**(26,558)**
Marketable securities	**5,918**	**9,849**	**49,540**	**7,100**	**(8,828)**
Cash balance plus marketable securities (end of period)	**19,525**	**29,755**	**82,130**	**26,401**	**(35,386)**

Sources: Chimerix, Inc. and William Blair & Company, L.L.C. estimates

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DJIA:	14,973.96
S&P 500:	1,614.42
NASDAQ:	3,378.63

The prices of the common stock of other public companies mentioned in this report follow:

Astellas Pharma, Inc.	$53.34
Bayer AG	$106.96
Gilead Sciences, Inc. (Outperform)	$55.15
Merck & Company, Inc.	$45.67
Vical Incorporated	$3.58

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