COMPANY NOTE

Initiating Coverage

USA | Healthcare | Biotechnology

February 24, 2014

Jefferies

EQUITY RESEARCH AMERICAS

Dicerna Pharmaceuticals (DRNA)

Initiate With A Buy – We Like The Odds On These Stacked Dice(r)

BUY

Price target $48.00

Price $39.50

Key Takeaway

We believe there is significant value in DRNA's RNA interference (RNAi) technology. We see preclinical data for the lead program for primary hyperoxaluria, a rare liver/kidney disorder, as predictive of human benefit. DRNA's cancer programs are high risk. We expect critical human validation of both its technology and lead program in 2H15.

In Jan 2014, Jefferies acted as a joint bookrunner in the initial public offering of DRNA common shares.

Bull Thesis: DRNA's RNAi Platform Should Be Validated With Multiple Data Readouts In 2015. DRNA develops RNAi therapeutics, which can silence a specific gene in select cells. We see significant value in the technology alone, particularly since only a small number of companies have the freedom to operate from a patent standpoint and we believe RNAi can enable the treatment of diseases inaccessible to conventional drug technologies. The company's lead unpartnered candidates are DCR-PH1 for Type 1 primary hyperoxaluria (PH1), a rare orphan disease resulting in renal failure, and DCR-M1711 targeting MYC for solid tumors. DCR-PH1 is expected to enter a Phase 1 study in PH1 in early 2015 with data in 2H15. DRNA plans to expand its RNAi platform into other liver-targeted orphan diseases, and we expect the company to disclose the second orphan liver candidate in 2H14, with a Phase 1 study in 2H15. We believe positive early preclinical data for DCR-PH1 will provide safety and mechanistic validation for the platform and support DRNA's use of it for other liver-targeted orphan diseases to rapidly build a robust pipeline.

Bear Thesis: Lack Of Human Data, Limited Visibility Into Market Opportunities. Dicerna has not yet validated its technology in human studies. Proof-of-concept data should be available in 2H15 for both DCR-PH1 and DCR-M1711. Whether the FDA will accept an accelerated development pathway in PH1 remains uncertain. Further, it is difficult to establish the ultimate market opportunities for either of DRNA's lead programs: the true number of PH1 patients is hard to assess given its rarity and low diagnosis rates. DRNA's cancer programs are early-stage and it is unclear whether silencing MYC or KRAS will lead to a strong efficacy effect.

Valuation/Risks

We derive our $48 price target on a DCF based sum-of-parts valuation ($16 PH1 + $11 MYC + $15 Pipeline + $5 cash). Risks include: clinical, regulatory, and commercial.

Financial Summary	
Net Debt (MM):	($141.7)
Cash (MM):	$147.6

Market Data	
52 Week Range:	$46.00 - $15.00
Total Entprs. Value (MM):	$549.6
Market Cap. (MM):	$691.3
Insider Ownership:	43.3%
Institutional Ownership:	38.3%
Shares Out. (MM):	17.5
Float (MM):	3.8
Avg. Daily Vol.:	NA

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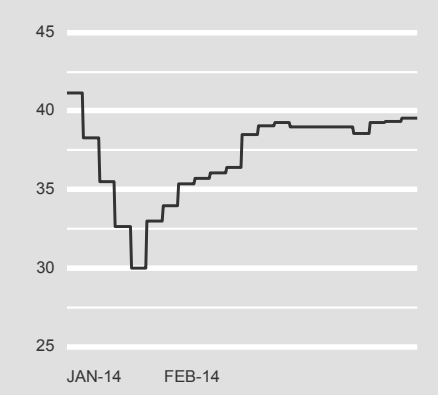
USD	Prev.	2012A	Prev.	2013E	Prev.	2014E	Prev.	2015E
Rev. (MM)	--	7.0	--	0.0	--	0.0	--	0.0
EV/Rev		78.5x						
EPS								
Mar	--	--	--	(1.55)A	--	(0.40)	--	--
Jun	--	--	--	(1.17)A	--	(0.40)	--	--
Sep	--	--	--	(0.94)A	--	(0.40)	--	--
Dec	--	--	--	(1.08)	--	(0.40)	--	--
FY Dec	--	(4.95)	--	(4.55)	--	(1.60)	--	(2.37)
FY P/E		NM		NM		NM		NM

Price Performance



Dicerna Pharmaceuticals

BUY: $48 Price Target

THE LONG VIEW

Scenarios

Target Investment Thesis

- We believe DCR-PH1 can reach $400m in the U.S.
- We expect DRNA is able to develop its MYC inhibitor DCR-M1711 for HCC
- We assume DRNA develops a pipeline basket of 10 orphan drugs with launches between 2021-2027
- Target Price: $48 = $16 PH1 + $11 MYC + $15 Pipeline + $5 cash

Upside Scenario

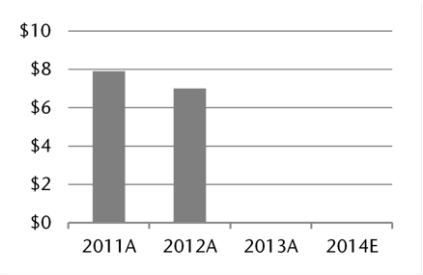
- There could be considerable upside to DCR-PH1 sales if launch drives PH1 diagnoses / treatment
- DCR-M1711 could address solid tumors more broadly
- Profitable co-promote of KRAS program with KHK
- Success with multiple pipeline assets
- Target Price: $87 = $33 PH1 + $20 MYC + $29 Pipeline + $5 cash

Downside Scenarios

- Dicerna's RNAi chemistry or delivery technologies have a narrow window of safety
- DCR-PH1 fails to produce meaningful improvements leading to FDA delays
- DCR-M1711 MYC targeting not safe or efficacious
- Pipeline assets fail
- Target Price: $12 = $7 PH1 + $0 MYC + $0 Pipeline + $5 cash

Long Term Analysis

Revenue (millions)



Source: Company Reports, Jefferies LLC

Long Term Financial Model Drivers

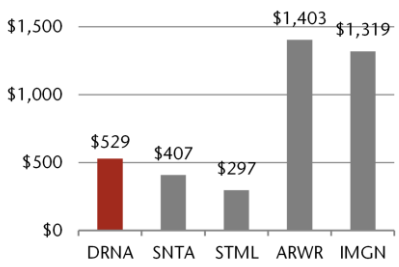
Earnings CAGR	**55%**
Revenue Growth	**37%**

Other Considerations

With several eagerly anticipated product launches, anemic pipelines at pharma, and an increasingly conservative FDA stance, we believe small and mid-cap biotech could lead sector performance in 2014. We see a premium placed on late-stage and marketed products. M&A interest could also factor into the performance of the sector, particularly among small-cap and mid-cap companies with later stage programs.

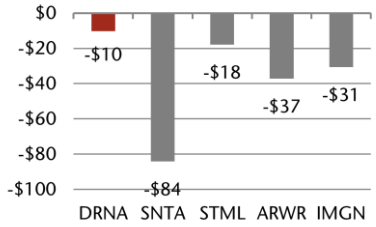
Peer Group

Group EVs



Source: Jefferies LLC

2012 Net Income



Source: Jefferies LLC

Recommendation / Price Target

Ticker	Recommendation	PT
DRNA	Buy	$48
SNTA	Buy	$19
STML	Buy	$60
ARWR	Buy	$30
IMGN	Buy	$21

Catalysts

- 2H14: Disclose second orphan liver candidate
- 1H15: Initiation of Phase 1 study of DCR-PH1
- 2H15: DCR-PH1 Phase 1 data

Company Description

Dicerna Pharmaceuticals is a Watertown, MA-based therapeutics company focused on developing RNA interference (RNAi) technologies targeting liver and cancer. Dicerna has partnered two oncology development programs with the global pharmaceutical company Kyowa Hakko Kirin Co., Ltd. (KHK) targeting KRAS and CKAP5. DRNA's unpartnered programs are DCR-PH1 for Type 1 primary hyperoxaluria, a rare orphan disease resulting in renal failure, and DCR-M1711 targeting MYC for solid tumors.

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Executive Summary

Dicerna is a Watertown, MA-based RNA therapeutics company with development programs in liver and cancer. Dicerna is using its proprietary RNA interference (RNAi) technology platform to build a broad pipeline in both liver disease and cancer, both on its own and in collaboration with partners. In indications such as rare diseases in which only a small sales force is necessary, Dicerna expects to retain commercial rights in key markets. In oncology and larger disease areas, the company has or intends to partner its product candidates while retaining significant portions of the commercial rights in North America. To date, Dicerna has partnered two oncology development programs with the global pharmaceutical company Kyowa Hakko Kirin Co., Ltd. (KHK, TSE: 4151, 1109 JPY, NC) and is eligible to receive royalties on worldwide net sales for these product candidates. With KHK, Dicerna also has an option to co-promote any product candidate targeting the oncogene KRAS, the more advanced of the two programs, in the U.S. for 50% of the profit. The company's lead unpartnered products are DCR-PH1 for Type 1 primary hyperoxaluria (PH1), a rare orphan disease resulting in renal failure, and DCR-M1711 targeting MYC for solid tumors. DCR-PH1 is the first orphan drug candidate from Dicerna's RNAi platform and is expected to enter a Phase 1 study in PH1 in early 2015 with data in 2H15. Dicerna also plans to expand its RNAi platform into other liver-targeted diseases, similar to the strategy employed by Alnylam (ALNY, $90.64, NC). The specific targets Dicerna plans to develop have not yet been disclosed for competitive reasons, though we expect the company to disclose the second orphan liver candidate in 2H14, with Phase 1 studies to be started by late 1H15. Dicerna expects multiple clinical data readouts in 2015.

Valuation

We derive our $48 price target for Dicerna primarily based on the prospective value of its RNAi platform with a probability-adjusted value for Dicerna's lead products, DCR-PH1 and DCR-M1711. We assume a key component of Dicerna's value is derived from its RNAi technology platform at $15 per share. **See Chart 1**. To estimate the platform opportunity, we have taken a basket of 10 approved orphan drugs and averaged their historical and projected sales to generate an average sales curve for a typical orphan drug, with peak worldwide sales of ~$800 million. We do not adjust for inflation or higher drug prices. We assume Dicerna attempts to develop 10 orphan drugs over time with potential launches from 2021-2027, each with a 15% probability of success. This effectively assumes that Dicerna is able to develop 1.5 drugs from its technology. For each product, we assume Dicerna shoulders full worldwide development and marketing costs with R&D costs of $90m, COGS of 10% and SG&A reaching 15% of peak sale. For DCR-PH1, we build out a dedicated valuation. We believe sales of DCR-PH1 could peak at $840m worldwide in 2030 if it can demonstrate normalization of oxalate levels. Our model assumes that Dicerna commercializes DCR-PH1 in the U.S. itself, with peak sales reaching $407m, and licenses rights in Europe and Asia, with total milestones of $300 million and tiered-royalties of 20-30%. We assume a 35% probability of success for DCR-PH1 to reach a valuation of $15 for DCR-PH1. This discount reflects the early-stage nature of the program and the uncertainty in the actual number of patients. We believe there is greater overall potential in the pipeline compared to PH1 and expect positive Phase 1 data from DCR-PH1 to de-risk our pipeline estimates. Every 5% incremental de-risking of the pipeline adds $5.15 per share. We also build a dedicated model for DCR-M1711 for liver cancer alone. However, given the high risk nature of the program, we have assigned a 20% probability of success and this program adds $11 per share to our valuation. If both the pipeline and the two lead programs were completely de-risked, we estimate that Dicerna should be valued at $209 per share at a minimum. We note that there remains significant

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upside to our valuation from other indications for DCR-M1711 and we ascribe no value to the rest of the programs in the KHK collaboration, including KRAS where Dicerna would be eligible to opt in for 50% of the U.S. profits at the time of regulatory filing.

Chart 1: Dicerna DCF Assumptions

Primary Hyperoxaluria 1	($M)	$924
Risk Adjustment	35%	**$323**
Key Assumptions		
Discount Rate	12%	
Terminal Growth Rate	4%	
DCR-M1711	($M)	$1,076
Risk Adjustment	20%	**$215**
Key Assumptions		
Discount Rate	12%	
Terminal Growth Rate	3%	
Pipeline NPV	($M)	$2,044
Risk Adjustment	15%	**$307**
Key Assumptions		
Number of Products	10	
Discount Rate	12%	
Terminal Growth Rate	5%	
Cash		**$104**

Total Dicerna Value	**$949**
Shares outstanding	20
Value per share	$48

Source: Jefferies estimates, company data

Risks

In addition to standard risks associated with an early-stage biotechnology company, we note that Dicerna is subject to specific risks related to its technology, the lead DCR-PH1 development program for PH1, and the cancer programs.

Platform Controversies

- **Street Ascribes Value To The Dicer Substrates And Delivery Technology, But Targets, Clinical Risk And Market Opportunities Are Uncertain.** Dicerna's Dicer substrate (DsiRNA) platform and delivery systems (EnCore Lipid Nanoparticles and conjugate approaches) underpin the company's platform and drug candidates. However, there are no human data to date directly validating the efficacy or safety of this approach. From preclinical animal toxicology data for the cancer candidate, the dose limiting toxicity in mice was liver enzyme elevations, which started to occur at doses of 10 mg/kg each week, and the dose-limiting toxicity in monkeys was complement-mediated disorders (coagulopathies) starting at doses of 4 mg/kg weekly. This may provide at least a 50-fold safety window relative to the predicted human

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dose exposure for PH1 (which the company believes could be dosed at 0.3 mg/kg once every three weeks based on the preclinical data in mice). The safety window may be smaller in the cancer indications where higher doses may be needed, but treatment is also likely of short duration in nature and may be acceptable in light of the disease and the clinical benefit. In addition, we are encouraged by the progress of other companies in the RNAi field, which have proven that early toxicity and delivery challenges with RNAi can be overcome. In addition, Dicerna's specific targets and diseases remain undisclosed, making it difficult to assess the risks, market opportunity or competitive landscape around these programs. That said, we believe that, as more details emerge, it is likely investors will shift focus to these liver targeted orphan diseases especially if Dicerna can find an indication where a biomarker may be accepted by the FDA as a surrogate endpoint for approval, or at least where reduction in target protein expression will be interpreted by investors as a highly predictive translation to clinical benefit. We see this as the case for the lead program, DCR-PH1 for the treatment of PH1.

- **Potential Competition.** Arrowhead is the other RNAi company that has a license (non-exclusive) to DsiRNA therapeutics from the City of Hope, and in addition, there are several other RNAi players using different RNAi chemistries and delivery technologies. One common theme to date is that companies, due to delivery challenges, have predominantly focused their initial pipeline efforts on liver diseases and cancers. Players such as Alnylam have already announced official programs in several liver target orphan diseases. Other competition includes Arrowhead (ARWR, $22.45, Buy), Tekmira (TKMR, $21.49, NC), Silence Therapeutics (LSE SLN, 3.59 GBP, NC). Dicerna and other RNAi players have all indicated that they are not likely to pursue a fast follower strategy. This could be something that changes over time, however, if large market opportunities open up and companies see a potential clinical advantage to a second-in-class competitor. Each of the RNAi companies will have filed IP around sequences that best inhibit any particular target prior to disclosing the pipeline program, but Dicerna has indicated that, in its case, these sequence patents will likely only cover 25-base long DsiRNAs against that target and that companies with other RNAi chemistry (e.g. Alnylam's 21-base long sequence) could still find RNA sequences that are not covered under Dicerna's patents. One of the key motivators for a fast follower may be to improve upon administration: Dicerna's lipid delivery technology requires intravenous administration and success in a pipeline program may spur competitors to try to develop a subcutaneous formulation, particularly for the treatment of chronic diseases such as PH1. Were all companies developing RNA interference technologies to avoid pursuing the same indications, we still believe there could be enough liver-targeted diseases to allow RNAi companies to develop strong pipelines with no near-term risk of overlap or competition.

- **Intellectual Property Risk.** Although we understand Dicerna to be the exclusive licensee of the DsiRNA patent estate from the City of Hope as well as a license holder to two patent estates covering the use of RNAi, the Carnigie Institution of Washington "Fire & Mello" patent estate and the Plant Bioscience Limited "Baulcombe" patent estate, we note that Dicerna may be potentially infringing a patent currently held by Alnylam in Europe. Dicerna noted that it had been made aware of potential infringement of Alnylam's patents and patent applications in March 2010, but disputed the claims raised over the course of multiple discussions and note that it has not received further correspondence from Alnylam since 2010 on this claim. The European patent assigned to

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Alnylam (EP '061 patent) covers RNAi constructs, including DsiRNA molecules, and has been validated in Austria, Germany, Ireland, Liechtenstein, Switzerland, and the United Kingdom. This patent had been challenged by Sirna Therapeutics (acquired by Merck [MRK, $56.03, Hold] in 2006), but the Opposition Division of the European Patent Office (EPO) upheld all claims in August 2009. As the decision was on appeal, Alnylam announced its acquisition of Sirna in January 2014, suggesting that these claims will not be pursued and the appeal will be terminated with the EPO without the possibility of continuing the opposition, as neither Dicerna nor any other third-party company had entered into the opposition as a party. Dicerna noted that it could challenge the EP '061 patent in each validated European country, but it is unclear if it will succeed. Dicerna noted that patent applications covering similar grounds are being pursued in the U.S. and other jurisdictions. Were these patents to be issued and upheld, Dicerna would be at risk of being sued and forced to pay damages or seek licenses from the patent holders for the duration of the remaining patent life – expected to be through 2022 for EP '061 and foreign counterparts. We believe that Dicerna could likely negotiate reasonable terms with Alnylam and further note that any royalties would only be payable from approval to 2022, which is a relatively short timeframe to pay a royalty. Further, we note that the full extent of this risk may not be known until patents issue, as IP-seekers are not necessarily immediately required to disclose their existence. Under current rules, the common time for disclosure of a patent application is 18 months after the earliest finding for which priority is claimed.

PH1 Controversies

- **Mechanism of action is not fully validated.** DCR-PH1 is thought to work in PH1 by preventing the overproduction of oxalate through the targeting of the enzyme glycolate oxidase (HAO1) upstream of oxalate formation. The efficacy of this mechanism has been supported by reducing excreted levels of urinary oxalate to near baseline levels in a genetic PH1-model mouse with elevated urinary oxalate. Despite promising activity in this mouse model, we note that Dicerna has not yet shown efficacy of DCR-PH1 in humans with the disease and initial proof-of-concept data are not expected until mid-to-late 2015. It is also unclear what degree of efficacy will be realized and whether normalization of oxalate levels can be achieved with DCR-PH1.
- **Sizing of the PH1 market opportunity.** A key risk for Dicerna is the actual size of the PH1 market opportunity, estimates for which vary by nearly an order of magnitude depending on the estimates used from a prevalence range of 1-3/1,000,000 from scientific journal articles to an incidence of 8/1,000,000 using company estimates derived from scientific literature regarding allele frequency. Further, we note there is apparent geographic variability in incidence rates, with NIH estimates varying from 1/100,000-1/250,000 and EU estimates for PH1 of roughly 1/120,000 live births, based on the expected genetic incidence. There is the additional complication of adequately identifying these PH1 patients for treatment, as we note a registry organized by the Mayo Clinic in 2003 has identified only 287 PH1 patients, as of June 2013, but Dicerna estimates that roughly 1,000 patients have been identified in disease registries in North America and Europe based on its own market research with PH1 patient advocacy groups that manage patient registries. Nevertheless, Dicerna contends that the true prevalence of PH1 is underreported, suggesting possible upside to our current estimated market opportunity. We currently model 300 and 600 patients in the U.S. and EU, respectively, in line with the company's market

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research, but see considerable risk to the market opportunity given the range of prevalence estimates and potential difficulty establishing diagnoses.

- **Pricing in PH1 remains unclear.** We believe a major value driver for the PH1 program, as with most drugs being developed for small indications, is the ability to take aggressive orphan drug pricing with the assumption that any individual payor in the U.S. will have so few patients affected that there is unlikely to be scrutiny. Given our expectation that the PH1 indication is limited to roughly 900 patients in the U.S. and EU markets, considered to be an ultra-orphan disease, we are assuming that the company charges roughly $425,000 per patient per year. We note that some drugs, such as Sanofi's (SAN FP, €74.00, Buy) Myozyme for Pompe's disease (which affects 10,000 patients), can cost $700,000 on average in the U.S. for adults, and we have found average pricing data in Europe suggesting an even higher cost. For instance, the Netherlands has requested pricing adjustments for Myozyme noting that it had been commanding costs of up to 700,000 euros per year per patient. Both the ability to price the drug freely and to retain support of payors both in the U.S. and in Europe are crucial assumptions underlying our market opportunity estimates.

- **Development risk around urinary oxalate as a primary endpoint for the PH1 trials.** Dicerna has articulated a strategy whereby the urinary oxalate levels will serve as the endpoint for its clinical trials and regulatory approval. We remain somewhat cautious about this endpoint given that the excretion of urinary oxalate is known to be variable, especially early in the course of PH1, and some patients with PH1 do not even have markedly elevated levels of urinary oxalate. We believe that, although oxalate elevation is considered a constitutive symptom of PH1, it directly leads to kidney failure and is a precursor to more permanent damage from PH1 through the deposition of oxalate in tissues (systemic oxalosis) and particularly in the skeleton. However, we note that in light of the FDA decision to request that Alnylam include additional data from a modified composite Neuropathy Impairment Scale (mNIS+7) prior to approval of patisiran (ALN-TTR02) for the treatment of transthyretin (TTR)-familial amyloid polyneuropathy (FAP) despite having shown robust and significant levels of reduction in serum TTR protein, we believe that it remains a question whether the FDA would accept an improvement in urinary oxalate levels as adequate for approval, even if it were statistically significant. That said, the TTR example could be different in that the mNIS+7 scale had been previously used as a regulatory endpoint, whereas no drug has ever been approved for PH1. Therefore, there remains a question of what could be regarded as a suitable endpoint for approval for PH1. Beyond urinary oxalate, it is unclear if a better endpoint exists. A recent New England Journal of Medicine article highlighted a symptomatic assessment for PH1 including kidney stones that consisted of more than 95% calcium oxalate monohydrate and finding of oxalate crystals in kidney-biopsy, but measurement of resolution of these may be complicated. While other urinary metabolites such as glycolate and l-glycerate are considered helpful, they are not regarded as conclusive, occurring only in a fraction of PH1 patients. Plasma levels of oxalate are thought to remain relatively normal until patients have their kidney function substantially impaired (e.g. stage 3b chronic kidney function – GFR of 30-45ml/min/1.73m^2). The use of a surrogate endpoint may introduce several risks, including the potential for patient-to-patient variability in urinary oxalate levels potentially compounded by the likely small number of patients in the clinical trial. There could be increased uncertainty on the regulatory acceptability of this endpoint if urinary oxalate levels are reduced with DCR-PH1 but not entirely normalized (i.e. we know that healthy individuals have a urinary oxalate level <0.45 mmol/1.73m^2/day, versus >1

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mmol/1.73m^2/day for a PH1 patient, but what constitutes a clinically meaningful reduction in urinary oxalate within this range). In the event of a clinical endpoint trial being requested by the FDA, it is unclear what would be measured and over what time frame. There are several well-accepted clinical measures of renal function (glomerular filtration rate), but the rates of kidney decline can vary significantly from patient to patient and may require too large of studies relative to the size of the patient population. Overall, we remain encouraged that Dicerna could convince the FDA to accept urinary oxalate as an endpoint for approval, given the unmet medical need, the likely endorsement of this endpoint by expert nephrologists, and the ultra-orphan nature of the disease.

Cancer Controversies

- **Will MYC knockdown translate to an overall survival benefit?** Dicerna has shown promising early single-agent activity with DCR-M1711 in a human hepatocellular carcinoma (HCC) mouse xenograft model. The company has been able to visualize strong and well-distributed target MYC mRNA knockdown by RNAi in tumor samples with significant reduction in MYC RNA at both 5mg/kg and 0.5mg/kg when compared to placebo (PBS) and HPRT1. That said, the company has yet to show any human data with the U.S. all-comers Phase 1 trial to be initiated in 1Q14 and the Asian HCC trial later in the year. Although we expect an early indication of response rates by 2015, it is unclear if MYC alone will yield single agent responses and we do not believe there will be enough data to feel comfortable that this MYC knockdown translates into a meaningful benefit in overall survival until randomized Phase 2 trials are initiated in 2016. Further we do not know what degree of MYC amplification or reduction will be necessary for DCR-M1711 to show an improvement, thus we cannot be certain what fraction of the 33-50% of HCC patients with amplified MYC would benefit from this therapy, leading to some ambiguity in the ultimate market opportunity for the drug. Similar questions arise for the KRAS program; although there is strong rationale targeting it as an oncogene, it is unclear if KRAS is a driving oncogene in tumors and blocking it will lead to an overall survival benefit.

- **KRAS Development Program With KHK: Low-Risk, But Little Control.** In an early partnership with Kyowa Hakko Kirin Co. Ltd., Dicerna partnered rights to its KRAS program in addition to other undisclosed oncology targets. Although Dicerna retains an opt-in right for a 50:50 profit share or co-promotion in the U.S. at time of NDA filing, which effectively de-risks the program for Dicerna. Nevertheless, this gives Dicerna limited control over the development course for this asset, which to-date has shown full tumor regression in KRAS-dependent xenograft model. Based on KHK's recent presentation, it seems like the company intends to advance the compound into several cancers, starting with the poorly vascularized pancreatic and bile duct cancer, and then potentially moving development into colorectal and non-small cell lung cancer. Although we believe the timing of the opt-in for KRAS was ideal, exposing Dicerna to little risk for the program, we believe the limited agency and visibility in the program are less than optimal, and the economics associated with the second CKAP5 target are fairly poor with royalties in the 8-10% range. It is our understanding that despite potential additional targets under this agreement Dicerna intends to renegotiate economic terms prior to pursuing any further collaborations with KHK. We see the KRAS target as a free call option for Dicerna, and do not include it in our current valuation.

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Pipeline Controversies

- **Pipeline Risky, As Targets Have Yet To Be Identified, Technology Still Unvalidated.** Despite promising early preclinical efficacy signals for Dicerna's assets, we believe there is considerable risk in the pipeline that consists largely of undisclosed and otherwise unidentified orphan drug targets. We believe that while the Dicerna platform technology could be remarkably promising, the prospects for these pipeline assets are largely dependent on Dicerna's ability to successfully develop its lead assets without yielding a systemic safety or efficacy concern, and while defending both its intellectual property and freedom to operate from competing firms.

Review of RNA Interference and DsiRNA Therapeutics

RNA interference (RNAi) is a mechanism in cells that inhibits the expression of a specific gene and affects the production of a resulting protein. In the past, development of RNAi therapeutic products has been limited by the instability of RNA molecules in the blood, the inability of these molecules to access targeted cells or organs, as well as the inability to gain entry into the cell cytoplasm, where they carry out their function. Thus, delivery technology is required to protect RNA in the blood stream following administration, allow efficient and targeted delivery to the intended cells and enable cellular uptake and release into the cytoplasm of the cell. To date, few companies have been able to successfully deliver RNA to its intended target. Dicerna's approach uses slightly longer, asymmetric double-stranded RNAs, Dicer substrate RNAs (DsiRNAs). Unlike classic 21-mer siRNAs that are loaded into the RNA-induced silencing complex (RISC) loading complex before processing (the approach used by Alnylam), DsiRNAs enter the RNAi pathway earlier at the Dicer enzyme; this enhances incorporation into RISC and orientation for cleavage of the target mRNA.

Background of Dicerna's DsiRNA Program

RNAi is a highly potent and specific mechanism for silencing the activity of a targeted disease-driving gene. RNAi offers the potential to go beyond traditional therapeutic modalities, such as small molecules and monoclonal antibodies, and to attack targets that are expressed exclusively inside cells and lack good small-molecule binding pockets, which are not easily drugged with small molecules and monoclonal antibodies. Dicerna's RNAi approach is based on proprietary double-stranded RNA molecules, called Dicer Substrate siRNA (DsiRNA), which the company believes maximizes RNAi potency. Dicerna licensed the dicer substrate technology from City of Hope (COH) in November 2007 in conjunction with a Series A financing. In March 2009, Dicerna secured an exclusive worldwide right to sublicense the dicer substrate technology.

Dicerna's pipeline IP centers around several in-licensed patent estates as well as its own patents and patents pending protecting various claims to methods and compositions of matter related to its proprietary technology. We understand Dicerna to be the exclusive licensee of the DsiRNA patent estate from the City of Hope as well as a license holder to two patent estates covering the use of RNAi, the Carnigie Institution of Washington "Fire & Mello" patent estate and the Plant Bioscience Limited "Baulcombe" patent estate. The City of Hope license includes the core DsiRNA patent (U.S. 8,084,599), titled "methods and compositions for the specific inhibition of gene expression by double-stranded RNA," describes RNA structures having a 25 to 30 nucleotides sense strand, a blunt end at the 3' end of the sense strand and a one to four nucleotides overhang at the 3' end of the antisense strand, which is set to expire July 17, 2027. In addition to DRNA's own three U.S. patents, U.S. 8,349,809 (issued in January 2013 with an expiration date of January 2030), U.S. 8,372,816 (issued in February 2013 with an expiration date of April 2030) and U.S. 8,513,207 (issued in August 2013 with an expiration date of May 2030), the

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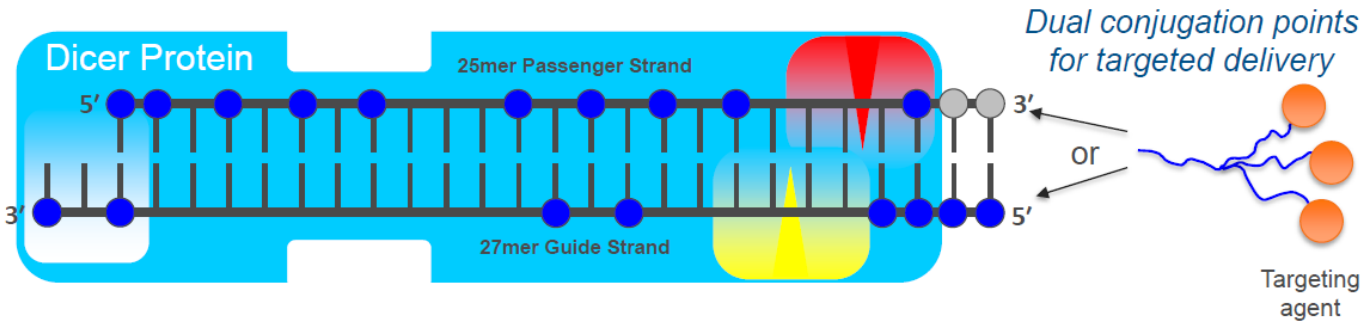
company has numerous patent applications covering specific DsiRNA sequences that drive activity against high value disease targets, including MYC, KRAS, CTNNB1 (ß-catenin), and other targets. We believe DRNA will be able to establish IP protection beyond 2030 by building patent protection around specific compounds and indications.

Exhibit 1: DsiRNA Structure



Source: Company presentation

The chemical structure of a DsiRNA is designed to be an ideal substrate for processing by the enzyme Dicer, the initiation point for RNAi in the human cell cytoplasm. Unlike first generation RNAi molecules, which mimic the output product of a Dicer enzyme processing event, DsiRNAs enter the RNAi pathway at this natural initiation point, earlier than the point at which 21-base long siRNAs used by Alnylam are incorporated. The asymmetrical nature of the DsiRNA encourages preferential use of the correct RNA strand of the DsiRNA, which increases the efficacy of the RNAi mechanism. This increased efficacy is believed to have two benefits. First, for some targets, it may increase the potency of Dicerna's DsiRNA molecules relative to other molecules used to induce RNAi. Further, Dicerna believes that, compared to 21-mers, DsiRNAs produce more viable sequence "hits" with high predicted potency.

Dicerna's Delivery Technology Overview

Dicerna has internally developed EnCore lipid nanoparticles, a proprietary system for the delivery of DsiRNAs to the liver and to solid tumors. In fact, this is the area where the company has spent the majority of its funding to date. EnCore consists of a core of lipid and DsiRNA encased by an outer lipid shell that confers an overall positive charge. The lipids used in the outer envelope help to mediate the accumulation, internalization and release into the cytoplasm of the DsiRNA hidden in the core of the particle. The lipid core enables binding of high levels of DsiRNA payload and protects it from degradation. These delivery particles are highly potent, have low toxicity, and are amenable to manufacturing at scale. Even at doses as low as 13.1µg/kg, silencing at the 50% level can be produced, which is 100-fold to 1,000-fold below the dose level at which dose-limiting toxicities would be expected. Dicerna initially began its work in oncology, as it was unsure if it had a therapeutic index for chronic dosing in normal liver, but it has since overcome toxicity challenges and has data demonstrating efficacy in animal models both in solid tumors and in normal liver tissue.

Dicerna's delivery of RNA to the target can be explained in three steps, the first being biodistribution, which results in accumulation of the EnCore nanoparticle exploited through its properties of being lipid. The next step is binding and internalization, in which the nanoparticle gains entry to the desired target cells via receptor-mediated

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internalization. In tumors, lipid nanoparticles are directly bound and internalized via tumor cell surface receptors, while delivery to other tissues requires a targeting agent to bind to an internalized receptor to gain entry. Lastly is cytoplasmic release, whereby the RNAi payload is unloaded from the endosome into the cytoplasm caused by ionizable lipids in the nanoparticle that results in fusion with the endosomal membrane, releasing the contents into the cytoplasm, where all of the RNAi machinery is located.

To date, Dicerna has tested a non-clinical formulation of the EnCore delivery particle in mice (multi-dose), rats (single-dose), and cynomolgous monkeys (multi-dose). In the monkeys, dosed bi-weekly for five doses ranging from 1-4 mg/kg, there were four significant observations at the highest dose (4 mg/kg), including general morbidity (organ changes were not severe by histopathology), non-severe liver enzyme (AST) elevations in all animals (no elevations of ALT, another liver enzyme), and elevation of cytokines IL-6 and MCP-1. As a result, the maximum tolerated dose (MTD) was determined to be 3 mg/kg on a bi-weekly basis.

Dicerna also has a Direct Targeted DsiRNA that enables targeted delivery of RNAi without the need for a lipid delivery vehicle. The DsiRNAs molecule has two distinct conjugation points, which can be used to attach targeting agents or other agents that facilitate delivery. It has been shown that both conjugation points can be used simultaneously without inhibiting processing by the Dicer enzyme. The Direct Targeted Delivery system mediates the process of delivery in a manner similar to EnCore, but does so as a single molecular entity instead of a particle. First, the strands of the DsiRNA are conjugated to a targeting agent as well as an agent that allows them to enter the cytoplasm from their delivery vehicle. After delivery to the cell interior, the Dicer enzyme cleaves the molecule just as with an unconjugated DsiRNA. Dicerna anticipates that future product candidates will utilize these conjugation points to further improve the delivery of DsiRNAs. The initial conjugate is likely to be n-acetylgalactosamine (GalNAc), which has been used by Alnylam to achieve efficient liver-specific delivery of siRNA. This would enable Dicerna to pursue a subcutaneous delivery approach for its pipeline candidates, as the EnCore lipid system requires intravenous administration.

Dicerna's Platform Beyond DCR-PH1

While the company has not yet provided specifics as to the intended disease targets beyond PH1, management has indicated it is also planning to extend its technology into additional liver-targeted orphan diseases. These are indications where presumably the silencing of genes could be easily interpreted to lead to clinical benefit, if not serve as a surrogate endpoint for regulatory approval. Dicerna has discussed the acceleration of its liver-targeted disease pipeline, but has yet to disclose the targets or diseases. We expect the company to disclose an additional orphan liver candidate in 2H14, and following the completion of preclinical toxicity studies, we expect the next liver-targeted candidate to enter clinical trials in 2015, with a Phase 1 study in 2H15.

Competitive Companies With RNAi Delivery Technologies

Dicerna's RNAi Technology is Differentiated From Competitors

Dicerna's technology differs from the approach being taken by most of its competitors, given its use of dicer substrates, rather than the 21-mer siRNA approach used by competitors. The leader in the space, Alnylam, began its development with 21-mer siRNA sequences and a lipid-based delivery technology licensed from Tekmira, but is now using its proprietary GalNAc system that conjugates the siRNA to a sugar molecule (rather than a lipid) to allow for subcutaneous delivery for liver-targeted genes.

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Alnylam

Alnylam (ALNY, $90.64, NC), is the current leader in the RNAi space, with proprietary siRNAs and two delivery systems, a lipid nanoparticle (LNP) technology licensed from Tekmira (see below for a description of Tekmira) and a proprietary technology that conjugates a sugar molecule called "GalNAc" to the siRNA molecule. While the LNP technology has been validated through clinical studies, it is accomplished via intravenous delivery versus GalNAc, which enables subcutaneous delivery. The company's lead product, ALN-TTR for TTR amyloidosis, is currently in Phase 3 trials. By the end of 2015, Alnylam expects to have more than five RNAi therapeutic programs in clinical development. The programs include: ALN-TTR02, an intravenously delivered RNAi therapeutic targeting transthyretin (TTR) for the treatment of TTR-mediated amyloidosis (ATTR) in patients with familial amyloidotic polyneuropathy (FAP); ALN-TTRsc, a subcutaneously delivered RNAi therapeutic targeting TTR for the treatment of ATTR in patients with familial amyloidotic cardiomyopathy (FAC); ALN-AT3 for the treatment of hemophilia and rare bleeding disorders (RBD); ALN-PCS for the treatment of hypercholesterolemia; ALN-AS1 for the treatment of acute intermittent porphyria (AIP); ALN-TMP for the treatment of beta-thalassemia and iron-overload disorders; ALN-CC5 for the treatment of complement-mediated diseases, and other programs. Notably, Alnylam recently announced its acquisition of Sirna Therapeutics from Merck, which consists of intellectual property and RNAi assets including pre-clinical therapeutic candidates as well as siRNA conjugate and other delivery systems. It is assumed that the acquisition was largely done to consolidate the Tushcl IP estate, which covers 21-mer siRNAs and chemical modifications.

Arrowhead

Arrowhead (ARWR, $22.45, Buy) has licenses to both Alnylam's 21-mer siRNA technology and to Dicer substrates through the City of Hope, as well as access to Meroduplex and UNA chemistry. The differentiating feature of Arrowhead's technology is its dynamic polyconjugate (DPC) delivery platform, which differs from the approach being taken by most of its competitors. The DPCs allow for active breakdown of endosomal membranes allowing siRNAs to be released more efficiently into the cytoplasm relative to other technologies, which could enhance potency of ARWR's siRNA candidates. ARWR's lead program, ARC-520, is for hepatitis B infection. The company plans on announcing a new orphan liver disease in 2Q14, but has indicated that this does not overlap with disclosed programs from any other RNAi company, including Dicerna.

Tekmira

Tekmira (TKMR, $21.49, NC) is a RNAi company that has a license to use Alnylam's payload technology (Tekmira has rights under Alnylam's intellectual property to develop thirteen RNAi therapeutic products) and has a proprietary lipid nanoparticle (LNP) technology. Tekmira is focused on products for the treatment of Ebola, oncology, Marburg's disease, and liver- focused targets.

- TKM-PLK1 (oncology): Ph 1/2 ongoing in patients with advanced Gastrointestinal Neuroendocrine Tumors (GI-NET) or Adrenocortical Carcinoma (ACC) with results mid-2014; Initiate a Phase 1/2 clinical trial in Hepatocellular Carcinoma in the 1H14;
- TKM-Ebola: Phase 1 to begin 1Q14, with results in 2H14;
- TKM-HBV: IND in 2H14; Phase I data in 2015;
- TKM-ALDH2 (alcohol use disorder): IND 2H14; Ph 1 data in 2015.

Silence Therapeutics

Silence Therapeutics (LSE SLN, 3.59 GBP, NC) is a U.K.-based company that uses a blunt-end dsRNA payload (AtuRNAi) and a lipid nanoparticle delivery technology that embeds

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siRNA into lipid bilayer particles. The company has three siRNA delivery systems: AtuPLEX for the vascular endothelium, DACC for the pulmonary endothelium, and DBTC for the liver. The Company's lead drug candidate is Atu027, a liposomal formulation in clinical development for pancreatic and head and neck cancer. Silence has completed a Phase 1 trial in solid tumors and has an ongoing Phase 1b/2a trial of Atu027 + gemcitabine for the treatment of pancreatic cancer.

Quark

Quark is a privately held company that has licensed its RNAi payload technology (blunt dsRNA) from Silence Therapeutics (described above) and Alnylam, and is focused on developing unformulated RNA duplexes for the treatment of ocular and kidney diseases. PF-655, partnered with Pfizer (PFE, $31.46, Buy) is in Phase 2 studies for diabetic macular edema (DME) and wet age-related macular degeneration (AMD). QPI-1002 (I5NP) is in Phase 2 development for acute kidney injury and delayed graft function, and Novartis (NOVN VX, CHF73.85, Hold) has an option to an exclusive worldwide license for all indications.

RXi

RXi (RXII, $4.80, NC) is a public company that focuses on the use of RNAi for the treatment of scarring. The company's sRNA payload was developed by adding a long extension to one strand of modified RNAi (modified to incorporate lipophilic and stabilizing moieties), and is delivered topically without a delivery technology. The company is pursuing anti-scarring and ocular indications. RXi's first RNAi product candidate, RXI-109, entered clinical trial in 2012 and is currently in Phase 2 testing. RXI-109 is a self-delivering RNAi compound that targets connective tissue growth factor (CTGF), a key regulator of fibrosis and scar formation, and is currently being developed to reduce scar formation in the skin following surgery.

PH1 Background

Dicerna is developing DCR-PH1 as a treatment for type 1 primary hyperoxaluria (PH1), a rare inherited autosomal recessive disorder of metabolism in the liver that results in irreparable kidney damage, by targeting the liver metabolic enzyme glycolate oxidase. In animal models in which one or both of the genes encoding glycolate oxidase in the liver have been inactivated, this inactivation has led to reduction or elimination of the key pathology of PH1. PH1 afflicts an estimated 5,000 people worldwide (1-3 per million in the U.S., per the New England Journal of Medicine) and results in accelerated severe renal disease and early mortality. That said, based on the frequency of occurrence of disease mutations in the population, the expected genetic incidence is eight per million of population, suggesting PH1 is potentially underdiagnosed. The Oxalosis and Hyperoxaluria Foundation (OHF) registry suggests there are currently north of 265 PH1 patients globally (out of 361 patients with any type of PH), although Dicerna believes that these numbers may not capture all patients contained within a registry based on its discussions with patient advocacy officials. We understand that the OHF itself believes that 1,000 patients with PH1 in the U.S. and EU may be contained in registries, and that the number is underreported likely due to lack of funding. Morbidity varies with disease presentation occurring in around 19% of affected individuals before age four to six months with severe disease, often associated with failure to thrive, nephrocalcinosis, anemia, and metabolic acidosis. Approximately 54% of affected individuals present in late childhood or early adolescence, usually with symptomatic nephrolithiasis and the remainder of affected individuals present in adulthood with recurrent kidney stones. The median age of onset of the disease is 5.5 years and data from the Rare Kidney Stone Consortium suggests that the median age at diagnosis of end stage renal failure (ESRD) is 24 years of age. Notably, 20%-50% of patients have ESRD at the time of diagnosis. The

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company expects to select a clinical candidate in 1H14, followed by IND filing and the start of clinical trials in 1Q15 and data from a Phase 1 study in patients by 2H15. This will be a single-dose study testing a range of doses and will likely enroll a total of 15-20 patients. Notably, the company expects to measure urinary oxalate levels as a biomarker. Normalization of urinary oxylate levels would provide proof-of-concept of gene silencing and would likely be viewed as highly predictive of clinical benefit by investors. We believe these early data would likely be taken to the regulatory agencies in 2H15 to discuss an accelerated pathway to approval, with the potential for a Phase 2/3 trial to start in late 2015/early 2016. We note that Dicerna plans to raise the question on the use of urinary oxalate as the primary endpoint for approval during its pre-IND meeting with the FDA in 1H15, but we do not believe the FDA would likely provide a definitive response until the end-of-Phase 2 meeting.

Primary Hyperoxaluria Overview. Type 1 primary hyperoxaluria (PH1) is a rare, inherited autosomal recessive disorder of metabolism in the liver that results in irreparable kidney damage. PH1 is caused by excess accumulation in the kidneys of an insoluble chemical called oxalate in the form of its calcium salt. Normal individuals have urinary oxalate excretion rates of <0.45 mmol/1.73m^2/day, versus >1 mmol/1.73m^2/day in patients with PH1. There are two main sources of oxalate: collagen breakdown and diet. It was previously thought that dietary oxalate accounted for little of the urinary oxalate levels (<10%), but Holmes et al [2001] showed that between 24% and 53% of urinary oxalate is attributable to oxalate from the diet [Holmes & Assimos 2004]. That said, patients with PH absorb even less oxalate than do healthy individuals and hence the fraction of dietary oxalate excreted in their urine is <5%. In patients with PH1, there is a loss of function in the AGXT gene that encodes the enzyme AGT1, which breaks down oxalate. In individuals with PH1, crystals of calcium oxalate form in the kidneys, leading to chronic formation of kidney stones, subsequent fibrosis, and ultimately leads to accelerated kidney failure. Diagnosis relies on measurement of urine oxalate to creatinine ratio, plasma oxalate concentration, molecular genetic testing, and ultimately, an assay of AGT catalytic activity from liver biopsy. AGXT is the only gene known to be associated with PH1 and targeted mutation analysis of specific AGXT mutations can detect 50%-70% of mutations, while sequence analysis can detect at least one AGXT mutation in 100% of affected individuals.

Typical PH1 interventions include massive intake of water to dilute the oxalate and the use of Vitamin B-6 supplementation, which modestly increases the solubility of oxalate, but neither of these approaches will stop disease progression. Responsiveness to vitamin B-6 supplementation is defined as a decrease in the level of urinary oxalate by more than 30% from the point of treatment initiation. The Gly170Arg (the most common – 30% of PH1 patients) and Phe152Ile (second most common, 9% of PH1 patients) genotypes are associated with a significant and sustained reduction of urinary oxalate levels during treatment with B-6, which leads to improvement in the overall prognosis. Responsiveness to B-6 has also been observed in patients with the Ile244Thr genotype. Vitamin B-6 supplementation can be discontinued after three months if oxalate levels have not fallen. Patients are often also supplemented with calcium at each meal, which helps to decrease urinary calcium oxalate with altering calcium excretion as well as thiazides and potassium citrate that can decrease urinary calcium excretion and inhibit stone formation. Further, significant intake of vitamins C and D is avoided to prevent stone formation. Reduction in dietary oxalate is typically ineffective in this patients population, given the relatively large proportion of oxalate is derived from collagen breakdown, particularly in PH patients.

Patients eventually enter end-stage renal disease (ESRD) at a median age of 33. Even daily dialysis at this point does not full prevent the accumulation of oxalate in the bone, skin, heart and retina, which results in concomitant complications. Currently, there are limited

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therapeutic options for most patients with PH1 aside from a dual liver-kidney transplant, a risky procedure that presents a challenge in identifying a donor and is associated with high co-morbidity rates. Even in those U.S. patients treated with dual-organ transplant, five-year overall survival is 64%. For patients treated with kidney transplant alone, five-year survival is 45%.

There are three forms of primary hyperoxaluria in which the underlying defects have been identified - PH1, PH2, and PH3 - which are each caused by an enzyme deficiency and each affects a different intracellular organelle, although all result in overproduction of oxalate. Primary hyperoxaluria type 2 (PH2) is caused by deficiency of the cytosolic enzyme glyoxylate reductase (GR), which catalyzes the reduction of glyoxylate and hydroxypyruvate, impairing the removal of glyoxylate and resulting in the overproduction of oxalate and L-glyeric acid and the increased secretion of both metabolites in the urine. PH2 is rarer than PH1, as PH1 accounts for 80% of PH patients. The diagnosis of PH2 can be established by assay of GR enzymatic activity in liver. From a small cohort of individuals with PH1 and PH2 from one center, PH1 as a group appears to differ from PH2 in the following respects: PH2 is considered a less aggressive disease than PH1 (even when onset is early), PH1 has statistically higher urinary oxalate excretions and more stone-forming activity (thus requires more frequent stone removal), individuals with PH1 have statistically lower urine osmolalities and lower urine concentration of calcium, citrate, and magnesium, and in PH1, urinary glycolate and oxalate are elevated while in PH2, urinary L-glycerate and oxalate are elevated (though exceptions exist). Primary hyperoxaluria type 3 (PH3) is characterized by elevated oxalate and glycolate and has been seen in about 5-10% of affected individuals in what appears to be a primary hyperoxaluria with normal AGT and GR enzymatic activity. Oxalate overproduction in patients with PH3 is caused by loss-of-function of the mitochondrial 4-hydroxy-2-oxoglutarate aldolase (HOGA) enzyme, and the excess oxalate might be generated via hydroxyproline metabolism. Patients with PH3 most often present with recurrent urolithiasis during the first decade of life. Although hyperoxaluria with or without hypercalciuria does not seem to disappear over time, it seems that PH3 starts to be clinically silent later in life and does not progress to end stage renal failure. Because excess oxalate occurs in all three forms of PH, DCR-PH1 could be used as treatment across the disease spectrum, but we believe that orphan drug pricing may become more scrutinized particularly in PH3 patients, which does not lead to end stage renal failure.

Rationale for DCR-PH1. There is a strong rationale for Dicerna to focus its RNAi technology on developing product candidates for the treatment of PH1. The hydroxyproline breakdown metabolic pathway that is disrupted in PH1 consists of a number of enzymes. The ultimate enzyme in the pathway, alanine-glyoxylate aminotransferase 1 (AGT1), is mutated in patients with PH1. When AGT1 is mutated, oxalate begins to build up, resulting in progressive loss of kidney function and, ultimately, kidney failure. The enzyme prior to AGT1 in the pathway is glycolate oxidase (HAO1). Inhibition of glycolate oxidase prevents the formation of the oxalate precursor and the buildup of oxalate. Inhibiting glycolate oxidase will cause glycolate, another byproduct, to be produced in excess, but this is much more soluble than oxalate and has not been associated with significant toxicity in animal models. There are also believed to be two human cases of excess glycolate production discovered through genome sequencing projects, which provides some real world evidence of the safety of excess glycolate. One of these patients was reported in an abstract from the American Society of Nephrology meeting in 2013. The patient and his brother were evaluated for psychomotor delay associated with anisocoria, alacrima and in the brother, also achalasia (triple A syndrome, unrelated to the metabolic abnormality). Urinary organic acid profile performed in the 8-year old patient showed markedly increased urinary glycolic acid excretion (2,000 mmol/mol creatinine; normal reference for age: 18-92) with normal excretion of oxalate,

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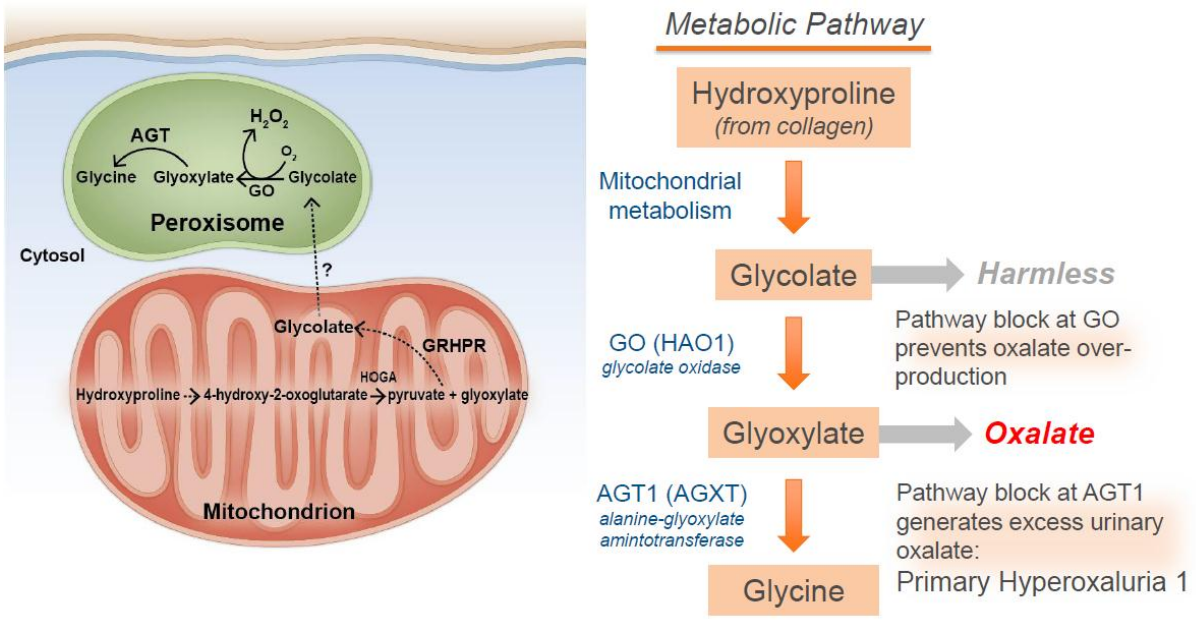
citrate, glycerate and glycine. Abdominal ultrasound showed normal sized kidneys with normal echotexture and direct sequencing of the HAO1 gene, encoding glycolate oxidase, revealed a mutation (not found in the brother affected by triple A syndrome or four healthy sisters, all having normal urinary glycolate excretion). Thus, it was determined that the loss-of-function mutations in the HAO1 gene, encoding glycolate oxidase, are responsible for asymptomatic isolated glycolic aciduria without hyperoxaluria. That said, the safety of excess glycolate production will likely remain a focus for the FDA.

In a mouse model, it was shown that glycolate oxidase (HAO1) is a valid target for the treatment of PH1. Double knockout mice missing both the AGT1 gene (the cause of PH1) and the HAO1 gene (the purported solution to PH1) were shown to be phenotypically normal, with urinary oxalate levels in line with wild-type mice. Relative to the AGT1 knockout mice, there was a profound impact of double knockout on urinary oxalate levels, with a return to non-toxic levels and a robust response to a dietary oxalate challenge. Importantly, the mice also demonstrated normal breeding, Mendelian rates, and histology. Additionally, the company has demonstrated HAO1 knockdown in vivo by EnCore delivered DsiRNAs. The peak observed knockdown of HAO1 was 97% in mouse liver after a single intravenous dose using either of two DsiRNA candidates. In these treated mice the company observed a significant reduction in oxalate levels in the urine. In treated mice, the urinary oxalate levels were returned to near baseline levels, similar to normal mice.

Exhibit 2: PH1 Disease Biology and Treatment Strategy



Source: Company presentation

Other programs in development for PH1. It has been suggested in publications that enzyme replacement therapy could be used to treat PH1, though the location of the enzyme of target is in the peroxisome, so this may make PH1 less amenable to enzyme therapy. Others have suggested bacteria to treat the disease by interfering with the GI tract, but this has not shown robust data to date. There have also been attempts at developing a small molecule inhibitor, though a suitable inhibitor has yet to be identified.

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OxThera AB, a Stockholm-based privately-held biopharmaceutical company, has initiated a 24-patient study in Primary Hyperoxaluria with its drug Oxabact, an oral product composed of highly concentrated freeze-dried live bacteria (Oxalobacter formigenes) administered in capsules to control oxalate ingested through diet.

MYC Background

Dicerna is developing DCR-M1711, a wholly owned RNAi for the treatment of cancers driven by targeting the MYC oncogene. MYC is upregulated in a wide variety of tumor types and this upregulation has been shown to be related to the presence and severity of cancer. Inhibition of MYC has exhibited strong anti-tumor effects in numerous animal models of human cancers. Notably, the company has shown strong single-agent efficacy with IV DCR-M1711 in human hepatocellular carcinoma orthotopic xenograph models. The data demonstrated that, after seven doses of DCR-M1711 over two weeks, there was a dose dependent antitumor effect with up to 79% tumor reduction by weight with the highest dose. A therapy that reduces or eliminates elevated MYC activity has the potential to generate therapeutic benefits for patients with various tumor types that include MYC amplifications or other elevations of MYC activity. Dicerna is investigating DCR-M1711 in a variety of tumor types with the initial focus on hepatocellular (liver) carcinoma.

It has been shown that MYC overexpression selectively amplifies expression of genes that are already expressed, which is why it is thought that reversion to normal MYC levels might be therapeutic. While MYC itself does not transform cells, it makes it easier for them to divide and is thus a facilitator of proliferation. MYC amplification rate by tumor is as follows: hepatocellular 50%, breast 80%, colorectal 70%, gynecological 90%, bladder 33%, prostate 70%, gastric 47%, small cell lung 20%. Knocking out MYC has been demonstrated to cure lung tumors in mice induced by KRAS. Studies done in mice have showed strong single-agent efficacy in liver cancer with DCR-M1711 delivered by IV injection. With complete knockout in mice, the liver grew back to a normal volume within the expected timeframe.

Dicerna has been completing IND enabling studies for its lead candidate as well as preparation of GMP batches (Phase 1 product is ready to go). Dicerna has also identified clinical sites and plans to file the IND soon and ship drug thereafter. The plan is to initiate Phase 1 studies in all-comers in a U.S.-based trial in 1Q14, followed by studies in liver cancer patients in Taiwan in 2H14 after preliminary safety data has been established in the U.S. study. In 1H15, Dicerna will also initiate a two-week window study in patients with hepatocellular carcinoma after determination of the maximum tolerated dose, in which first-line hepatocellular carcinoma patients will receive one dose of drug prior to surgery. A sample of the resected liver following surgery will be used to determine levels of MYC knockdown. The company is not requiring MYC amplification for this study (but MYC will be measured). Dicerna expects data from this window study in mid-to-late 2015.

MYC has not been successfully approached by conventional small molecule drugs and is not amenable to antibody therapeutics. Attempts to develop small molecules have been challenging due to MYC's role as a transcription factor (rather than an enzyme) and the absence of a good binding pocket or active site on the MYC protein. Antibodies are not a good option against MYC since MYC is not expressed on the cell surface.

Overview of Hepatocellular Carcinoma (HCC). HCC accounts for 85-90% of primary liver cancers and represents a significant market opportunity given the high unmet medical need. Liver cancer is the third leading cause of cancer-related deaths worldwide with over 695,000 annual deaths according to research published in 2010 by

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the International Agency for Research on Cancer, and annual HCC incidence rates are increasing worldwide.

Currently, there are limited treatments for HCC, particularly when it is advanced or unresectable. Early-stage HCC is generally treated with surgery that has the potential to be curative; however, given the nonspecific symptoms characteristic in HCC, the large majority of patients are diagnosed only after HCC is at an advanced stage. Advanced HCC has limited treatment options and is associated with poor patient outcome and high mortality. Chemotherapies have historically demonstrated poor efficacy in HCC. Nexavar is the only approved systemic therapy in liver cancer, with data showing 2.8 months life extension versus placebo, but is not well-tolerated (diarrhea, fatigue, infection, hair loss, skin reaction, rash, weight loss, gastrointestinal side effects, hypertension). Sales in 2012 were $780m in liver cancer (it is also approved in renal cell carcinoma and other indications; total sales were >$1b). In all indications, dosing is 2 tablets 2x daily until it is no longer clinically beneficial or unacceptable tox. The cost of treatment is $10,525 per month. In addition to approved therapies, Tekmira has announced that it expects to initiate a multicenter, single arm, open label dose escalation Phase 1/2 study for TKM-PLK1 in HCC in 1H14 and Kadmon Corporation (a private, NY, NY based company) is evaluating salirasib (KD032) in Phase 2 trials for the treatment of KRAS-specific non-small cell lung cancer, pancreatic cancer and other solid tumors.

KRAS Background

Dicerna, in collaboration with Kyowa Hakko Kirin (KHK), is developing DsiRNAs targeting the oncogene KRAS. KRAS is a key oncogenic signaling molecule that is involved in cell proliferation, survival, and invasion and metastasis. KRAS mutations are often associated with poor prognosis (aggressive disease and resistance to current therapies), with mutations occurring in 90% of pancreatic tumors, 40% of colorectal tumors and 25% of non-small cell lung tumors. Data to date has shown full tumor regressions of KRAS-dependent xenograph models using two different KRAS DsiRNAs. Regarding development, KHK is fully responsible for development, has selected the contract manufacturer, is planning to or has begun manufacturing, quality control, and stability testing, and expects to file an IND in 2014. The next step will be to move into Phase 1 testing, expected to begin in early 2015. Notably, the delivery does not use Dicerna's EnCore platform, but rather a similar lipid delivery technology.

Partnership with KHK. The partnership was signed in December 2009, with potential for $1.4b in value and up to 12 targets, focused on oncology and inflammation. Two products, the KRAS program and CKAP5 program, have advanced to development stage, and two additional targets have been explored. Additional target selection requires Dicerna's approval. KHK can nominate additional targets, but Dicerna has the sole discretion to accept and does not plan to do so at the current terms. Regarding the KRAS program, Dicerna has an opt-in at the time of U.S. regulatory filing for a 50/50 profit share and co-promotion rights in the US, for which Dicerna would pay half of the development expenses plus a small premium. On ex-U.S. sales, Dicerna receives high single-digit-to-low double-digit royalties on ROW sales. If Dicerna opts out of the co-promote, it would be paid a high single-digit/low double-digit royalty in both the US and ROW. Phase 1 trials for the KRAS program are expected to start in 1H15 and an IND for CKAP5 will follow six months later. Dicerna is also developing a candidate for an additional, undisclosed third oncology target in collaboration with KHK. Phase 1 studies for this program are expected to begin in 2H15.

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Management

Douglas Fambrough, Ph.D. - CEO - Dr. Fambrough co-founded Dicerna in 2007 and currently serves as CEO. Previously, Dr. Fambrough was a General Partner with the life science venture capital firm Oxford Bioscience Partners. His investments include first generation RNAi pioneer Sirna Therapeutics, organized by Dr. Fambrough and two other investors in 2000, where he served on their Board of Directors until the company was acquired by Merck for $1.1 billion in 2006. Before joining Oxford, he was a genomic scientist at the Whitehead/MIT Center for Genome Research. Dr. Fambrough graduated from Cornell and obtained his Ph.D. in Genetics at the University of California, Berkeley.

Bob D. Brown, Ph.D. - Chief Scientific Officer - Dr. Brown joined Dicerna as SVP, research in 2008. Previously, Dr. Brown was at Genta, where he was VP, research and technology and had more than 75 issued patents and patent applications. Dr. Brown also worked directly with clinicians and key opinion leaders performing the studies on trial design, execution and interpretation of results. Additionally, Dr. Brown was co-founder and VP of R&D at Oasis Biosciences. Dr. Brown holds a Ph.D. in molecular biology from the University of California, Berkeley, and a B.S. in chemistry and B.S. in biology from the University of Washington.

Jim Dentzer - CFO - Jim Dentzer joined Dicerna as CFO in December 2013. Previously, Jim was the CFO of Valeritas. Prior to Valeritas, he was the CFO of Amicus Therapeutics, where he led the company through a Series D financing and subsequent IPO. In prior positions, he spent six years as corporate controller of Biogen Idec and six years in various senior financial roles at E.I. du Pont de Nemours in the U.S. and Asia. Jim holds a B.A. in Philosophy from Boston College and an M.B.A. from the University of Chicago.

David Miller, Ph.D. - VP, Business Development - Dr. Miller joined Dicerna as senior director, business development in 2008. Previously, Dr. Miller was director of business development for Pioneer Valley Life Sciences Institute. Prior to this, Dr. Miller was senior director, business development at Synta Pharmaceuticals and director, business development at Altus Biologics. Additionally, Dr. Miller was a founder and the head of research for EcoScience Corporation and a corporate officer. He began his career at Genetics Institute. Dr. Miller holds a Ph.D. in molecular biology and biochemistry from Harvard and B.S. in biochemistry and biophysics from the University of California, Davis.

Paul I. Nadler, M.D., F.C.P., F.A.C.P. - Acting Chief Medical Officer - Dr. Nadler joined Dicerna as acting chief medical officer in March 2013. Dr. Nadler serves as the Managing General Partner for Nadler Pharma Associates, a consulting firm specializing in preclinical and clinical development, since 1998 and his medical training is in Internal Medicine, Oncology, and Clinical Immunology and Allergy. Prior to founding Nadler Pharma, he served as Chief Medical Officer for Kern McNeill International, Vice President, Medical and Regulatory Affairs with Protein Design Labs, Vice President, Scientific Planning and Analysis and Vice President, Medical Research with Sandoz Research Institute, Vice President and Medical Director with Knoll Pharmaceuticals, BASF Group and Director of Clinical Immunology and Head, Section of Immunology and Virology, Department of Medical Research with Hoffmann La Roche. Dr. Nadler holds an M.D. from Washington University School of Medicine and trained in Internal Medicine at Barnes Hospital/Washington University Medical Center and spent five years in the Immunology Branch of the National Cancer Institute, National Institutes of Health.

James B. Weissman - Chief Business Officer - James Weissman joined Dicerna as chief business officer in January 2012. Prior to Dicerna, he was VP of business development at MannKind, responsible for leading the company's activities related to

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licensing, new products and strategic planning. Prior to MannKind, Mr. Weissman held leadership positions in both business development and marketing at Pfizer Pharmaceuticals in Tokyo, most recently as senior director of marketing. Mr. Weissman holds a Bachelor of Science from Bates College in Maine.

Initial Public Offering

In January 2014, Jefferies acted as a joint book-running manager in an initial public offering of 6.9m Dicerna common shares at $15.00 per share. The financing had net proceeds of $92.9m to fund preclinical and clinical trials of proprietary product candidates, continued technology platform developments, working capital and general corporate purposes, as well as potential acquisitions or in-licensing activities.

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DRNA: Historical and Projected Revenue and Earnings

December 31 Fiscal Year ($000s, except per share)	2012	1Q13A	2Q13A	3Q13A	4Q13E	2013	1Q14E	2Q14E	3Q14E	4Q14E	2014	2015	2016	2017	2018	2019	2020	2021	2022
End User Sales																			
U.S. PH1 Sales		0	0	0	0	0	0	0	0	0	0	0	0	15,581	63,225	106,783	154,514	201,448	245,079
Ex-U.S. PH1 Sales		0	0	0	0	0	0	0	0	0	0	0	0	22,073	89,895	155,040	224,326	293,117	355,972
WW PH1 Sales		0	0	0	0	0	0	0	0	0	0	0	0	37,654	153,120	261,823	378,840	494,565	601,051
WW DCR-M1711 Sales		0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	66,150	667,229	1,829,961
Dicerna Reported Revenue																			
U.S. PH1 Sales		0	0	0	0	0	0	0	0	0	0	0	0	15,581	63,225	106,783	154,514	201,448	245,079
Ex-U.S. PH1 Royalties		0	0	0	0	0	0	0	0	0	0	0	0	4,415	19,974	36,512	57,298	77,935	96,792
DCR-M1711 Royalties		0	0	0	0	0	0	0	0	0	0	0	0	0	0	9,922	100,084	274,494	451,193
Milestones		0	0	0	0	0	0	0	0	0	0	0	50,000	125,000	25,000	25,000	125,000	75,000	75,000
Other Revenue		0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Revenue	**7,015**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**50,000**	**144,996**	**108,199**	**178,217**	**436,897**	**628,877**	**868,063**
Operating expenses:																			
COGS	0	0	0	0	0	0	0	0	0	0	0	0	0	1,558	5,058	7,475	10,816	14,101	17,156
% of Product Sales	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*10%*	*8%*	*7%*	*7%*	*7%*	*7%*
Research and Development	11,551	2,455	2,455	2,455	5,000	12,364	5,000	5,000	5,000	5,000	20,000	36,000	51,000	62,000	68,200	75,020	90,024	108,029	129,635
% of Revenue	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*43%*	*63%*	*42%*	*21%*	*17%*	*15%*
SG&A	4,589	1,192	1,192	1,192	1,400	4,977	2,000	2,000	2,000	2,000	8,000	11,000	18,000	33,000	38,000	41,000	44,300	47,115	49,021
% of Revenue	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*23%*	*35%*	*23%*	*10%*	*7%*	*6%*
Total Operating Expenses	16,265	3,647	3,647	3,647	6,400	17,341	7,000	7,000	7,000	7,000	28,000	47,000	69,000	96,558	111,258	123,495	145,140	169,245	195,811
Income (Loss) from operations	**(9,250)**	**(3,647)**	**(3,647)**	**(3,647)**	**(6,400)**	**(17,341)**	**(7,000)**	**(7,000)**	**(7,000)**	**(7,000)**	**(28,000)**	**(47,000)**	**(19,000)**	**48,438**	**(3,059)**	**54,722**	**291,757**	**459,632**	**672,253**
Other income (expense):																			
Preferred stock warrant remeasurement	469	73	73	73	73	292	0	0	0	0	0	0	0	0	0	0	0	1	2
Interest income	*2*	*0*	*0*	*0*	*0*	1	*55*	*46*	*37*	*28*	166	687	1,071	1,406	1,510	1,628	2,211	3,380	5,151
Loss on extinguishment of debt		(106)	(106)	(106)	(106)	(424)	0	0	0	0	0	0	0	0	0	0	0	0	0
Interest expense	*(1,342)*	*(253)*	*(253)*	*(253)*	*(69)*	(829)	*(69)*	*(69)*	*(69)*	*(69)*	(277)	0	0	0	0	0	0	0	0
Total other income (expense)	(871)	(286)	(286)	(286)	(102)	(960)	(15)	(23)	(32)	(41)	(111)	687	1,071	1,406	1,510	1,628	2,211	3,381	5,153
Pre-tax income	**(10,121)**	**(3,933)**	**(3,933)**	**(3,933)**	**(6,502)**	**(18,301)**	**(7,015)**	**(7,023)**	**(7,032)**	**(7,041)**	**(28,111)**	**(46,313)**	**(17,929)**	**49,844**	**(1,550)**	**56,350**	**293,967**	**463,013**	**677,405**
Tax	*0*	*0*	*0*	*0*	*0*	*0*	*0*	*0*	*0*	*0*	*0*	*0*	*0*	*18,442*	*0*	*20,850*	*108,768*	*171,315*	*250,640*
Tax rate														*37%*	*0%*	*37%*	*37%*	*37%*	*37%*
Less: Accretion and dividends on redeemable convertible preferred stock	(4,097)	(793)	(793)	(793)	0	(2,379)	0	0	0	0	0	0	0	0	0	0	0	0	0
Net loss	**(14,218)**	**(4,726)**	**(4,726)**	**(4,726)**	**(6,502)**	**(20,680)**	**(7,015)**	**(7,023)**	**(7,032)**	**(7,041)**	**(28,111)**	**(46,313)**	**(17,929)**	**31,402**	**(1,550)**	**35,501**	**185,199**	**291,698**	**426,765**
Non-GAAP EPS	**$ (4.95)**	**$ (1.55)**	**$ (1.17)**	**$ (0.94)**	**$ (1.08)**	**$ (4.55)**	**$ (0.40)**	**$ (0.40)**	**$ (0.40)**	**$ (0.40)**	**$ (1.60)**	**$ (2.37)**	**$ (0.80)**	**$ 1.31**	**$ (0.06)**	**$ 1.40**	**$ 7.05**	**$ 10.71**	**$ 15.09**
Pro forma weighted average shares outstanding	2,046	3,046	4,046	5,046	6,046	4,546	17,528	17,578	17,628	17,678	17,603	19,553	22,378	24,003	24,678	25,428	26,278	27,228	28,280

Source: Company data, Jefferies Group LLC estimate
2/21/2014

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DRNA: Historical and Projected Changes in Financial Position

December 31 Fiscal Year ($000s, except per share)	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
CASH FLOWS FROM OPERATING ACTIVITIES:									
Net loss	(10,121)	(20,680)	(28,111)	(46,313)	(17,929)	31,402	(1,550)	35,501	185,199
Adjustments to reconcile net loss to net cash used in operating acti									
Deferred revenue	(1,014)								
Depreciation	551	523	575	632	696	765	842	926	1,019
Stock-based compensation									
Loss on extinguishment of debt	-								
Non-cash interest expense	-								
Amortization of debt discount	201								
Amortization of debt issuance costs	24								
Utilization of NOL Carryforwards		-	-	-	-	18,442	-	20,850	-
Decrease in fair value of preferred stock warrant	(469)								
Changes in operating assets and liabilities:									
Research and license receivable	(4,857)	5,018	-	-	-	-	-	-	-
Prepaid expenses and other current assets	28								
Accounts payable	(216)								
Accrued expenses and other current liabilities	49								
Deferred rent	(38)								
Net cash used in operating activities	(15,737)	(15,140)	(27,536)	(45,680)	(17,233)	50,609	(708)	57,276	186,218
CASH FLOWS FROM INVESTING ACTIVITIES–									
Purchases of property and equipment	(120)	(256)	(307)	(369)	(3,686)	(4,055)	(4,461)	(4,907)	(5,397)
Net cash used in investing activities	(120)	(256)	(307)	(369)	(3,686)	(4,055)	(4,461)	(4,907)	(5,397)
CASH FLOWS FROM FINANCING ACTIVITIES:									
Proceeds from issuance of common stock	7	11	92,955	112,800	108,288	-	-	-	-
Repurchase of restricted common stock	-	(5)							
Payments of deferred issuance costs	-	(173)							
Proceeds from issuance of redeemable convertible preferred stock	-	57,000							
Redeemable convertible preferred stock issuance costs	-	(220)							
Proceeds from long-term debt	-	-							
Proceeds from bridge loan financing	-	3,000							
Payments of long-term debt issuance costs	-	-							
Payments of long-term debt fees	(136)	-							
Repayments of long-term debt principal	(2,834)	(4,140)	(4,587)	(439)					
Net cash provided by (used in) financing activities	(2,963)	55,473	88,368	112,361	108,288	-	-	-	-
INCREASE (DECREASE) IN CASH AND CASH EQUIVALENTS	**(18,820)**	**40,077**	**60,524**	**66,312**	**87,369**	**46,554**	**(5,169)**	**52,369**	**180,821**
CASH AND CASH EQUIVALENTS–Beginning of period	**22,490**	**3,670**	**43,747**	**104,272**	**170,584**	**257,952**	**304,506**	**299,338**	**351,707**
CASH AND CASH EQUIVALENTS–End of period	**3,670**	**43,747**	**104,272**	**170,584**	**257,952**	**304,506**	**299,338**	**351,707**	**532,528**

Source: Company data, Jefferies Group LLC estimate
February 21, 2014

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DRNA: Historical Condensed Balance Sheets

($000s)	12/31/2011	12/31/2012	9/30/2013
ASSETS			
CURRENT ASSETS:			
Cash and cash equivalents	22,490	3,670	54,712
Research and license agreement receivable	161	5,018	-
Prepaid expenses and other current assets	372	344	413
Total current assets	**23,023**	**9,032**	**55,125**
NONCURRENT ASSETS:			
Property and equipment0net	1,314	883	683
Restricted cash	264	264	264
Other assets	36	12	-
Total noncurrent assets	**1,614**	**1,159**	**947**
TOTAL ASSETS	**24,637**	**10,191**	**56,072**
LIABILITIES AND STOCKHOLDERS' EQUITY			
CURRENT LIABILITIES:			
Accounts payable	1,414	1,198	1,557
Current portion of long-term debt	2,834	4,140	4,471
Deferred revenue	830	-	-
Deferred rent	-	63	14
Accrued expenses and other current liabilities	221	270	518
Total current liabilities	**5,299**	**5,671**	**6,560**
NONCURRENT LIABILITIES:			
Long-term debt-net of current portion	8,735	4,660	1,407
Preferred stock warrant liability	800	331	436
Deferred revenue-net of current portion	184	-	-
Deferred rent-net of current portion	101	-	-
Total noncurrent liabilities	**9,820**	**4,991**	**1,843**
TOTAL LIABILITIES	**15,119**	**10,662**	**8,403**
Total stockholders' equity (deficit)	**9,518**	**(471)**	**47,669**
TOTAL LIABILITIES AND STOCKHOLDERS' EQUITY	**24,637**	**10,191**	**56,072**

Source: Company data, Jefferies Group LLC, Inc. estimates
February 21, 2014

Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

Company Description

Dicerna Pharmaceuticals is a Watertown, MA-based therapeutics company focused on developing RNA interference (RNAi) technologies targeting liver and cancer. Dicerna has partnered two oncology development programs with the global pharmaceutical company Kyowa Hakko Kirin Co., Ltd. (KHK) targeting KRAS and CKAP5. DRNA's unpartnered programs are DCR-PH1 for Type 1 primary hyperoxaluria, a rare orphan disease resulting in renal failure, and DCR-M1711 targeting MYC for solid tumors.

Analyst Certification

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

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Company Specific Disclosures

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Meanings of Jefferies Ratings

Buy - Describes stocks that we expect to provide a total return (price appreciation plus yield) of 15% or more within a 12-month period.

Hold - Describes stocks that we expect to provide a total return (price appreciation plus yield) of plus 15% or minus 10% within a 12-month period.

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

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Risk which may impede the achievement of our Price Target

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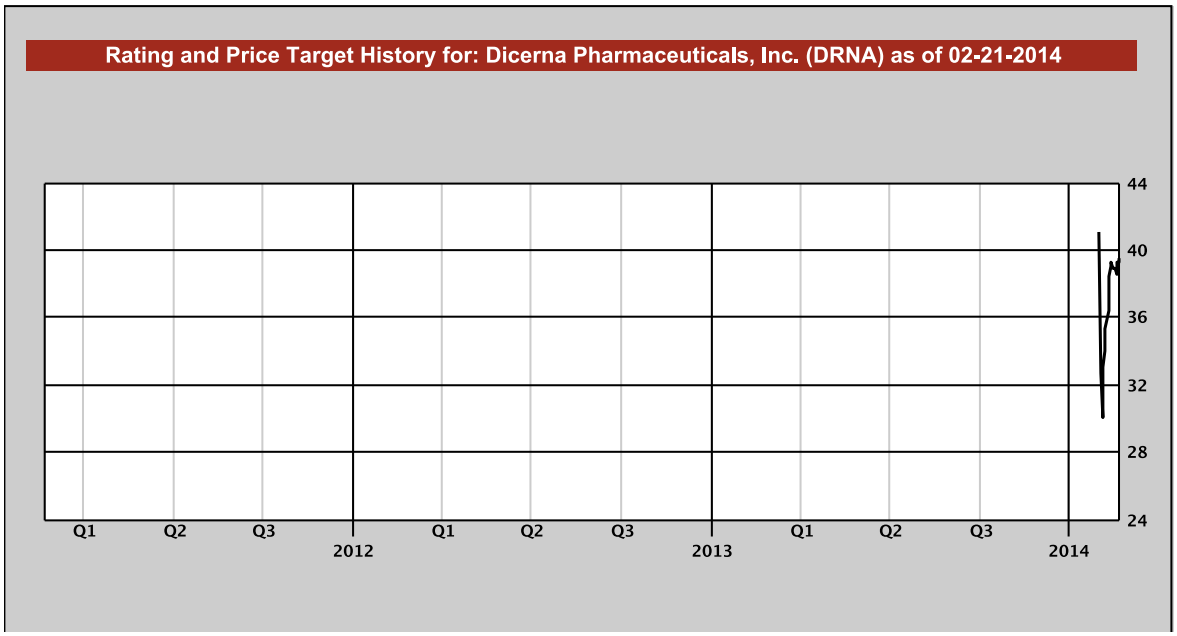
Other Companies Mentioned in This Report

- Arrowhead Research Corporation (ARWR: $22.45, BUY)
- ImmunoGen Inc. (IMGN: $17.48, BUY)
- Merck & Co. (MRK: $56.03, HOLD)
- Novartis AG (NOVN VX: CHF73.85, HOLD)
- Pfizer, Inc. (PFE: $31.46, BUY)
- Sanofi (SAN FP: €74.00, BUY)
- Stemline Therapeutics, Inc. (STML: $29.82, BUY)
- Synta Pharmaceuticals Corp. (SNTA: $6.44, BUY)



Distribution of Ratings

Rating	Count	Percent	IB Serv./Past 12 Mos. Count	IB Serv./Past 12 Mos. Percent
BUY	892	49.45%	213	23.88%
HOLD	763	42.29%	126	16.51%
UNDERPERFORM	149	8.26%	4	2.68%

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