Netherlands | Healthcare | Biotechnology

31 January 2018

Kiadis (KDS NA)

Nearing Green Light for ATIR; Initiating at Buy with €22 PT

Key Takeaway

ATIR could improve the safety and effectiveness of "half-matched" haplo-ID bone marrow transplants, further expanding their use. We expect EU conditional approval on Phase II data in 1Q19E, with final Phase III results by 1H20E. Assuming \$475m peak sales US+EU commercialising ATIR itself, our NPVs suggest the current share price significantly undervalues this opportunity, factoring-in likely future dilution given current cash is sufficient until early-2019E.

Jefferies acted as Sole Bookrunner for the Oct 2017 private placement raising c.€18m gross proceeds from 2.25m new shares at €8.

ATIR addresses an unmet need: Haematopoietic stem cell transplants (HSCT) can offer a cure for some serious disorders but it can be challenging to find matched donors, whereas haploidentical are widely available. Current protocols mitigate the life-threatening risk of graft versus host disease (GvHD), but typically also subdue graft versus leukaemia (GvL) antitumour and anti-infective benefits. ATIR aims to minimize GvHD while retaining the benefits, lowering the risk of relapse and complications. Phase II confirmed this potential, comparing very favourably for GvHD and relapse risks relative to literature reports for current standard-of-care PTCy, in our view.

Nearing green light for ATIR to boost HSCT: We forecast haplo-ID HSCT to more than double by 2026E, driven by protocols such as PTCy and potentially ATIR, for which we expect launch from 2H19E EU and 2022E US. Assuming 20% peak ATIR penetration with €150k/\$250k average Revenue/patient we derive \$240m/\$235m EU/US peak sales for c.€21/€10 per share NPV at 80%/50% probability. "Best" case we believe ATIR peak sales could near-\$2bn.

Adequate funds to reach key EU decision: Our model suggests cash is sufficient to fund burn until early-2019E, excluding any possible out-licensing deals or other income. Importantly this should be beyond the EU approval and potential Phase III HATCY interim analysis YE18-1H19E depending on the rate of patient enrolment. However, incremental funds are necessary for S&M and completing the Phase III required for US filing, in our view.

Valuation/Risks

Our €22/share Price Target is based on a sum-of-the-parts valuation comprising probability-adjusted NPVs for ATIR in the US and EU, together with Net Cash, less potential dilution to ensure sufficient funds until YE2020E. Risks include: (1) clinical or regulatory setbacks; (2) commercial execution risks; and (3) securing adequate funds to maximise value.

| EUR | Prev. | 2016A | Prev. | 2017E | Prev. | 2018E | Prev. | 2019E |
|-----------------------|-------|--------|-------|--------|-------|--------|-------|--------|
| Rev. (MM) | | 0.0 | | 0.0 | | 0.0 | | 3.6 |
| EV/Rev | | | | | | | | 54.2x |
| EBIT (MM) Adjusted | | (11.4) | | (17.5) | | (23.6) | | (27.4) |
| EV/EBIT | | NM | | NM | | NM | | NM |
| Cash Position | | 14.6 | | 29.5 | | 3.8 | | 2.5 |
| EPS Adjusted | | | | | | | | |
| FY Dec | | (1.08) | | (1.26) | | (1.52) | | (2.42) |
| FY P/E | | NM | | NM | | NM | | NM |

RIIV

Price target €22.00 Price €10.96^

| Financial Summary | |
|---------------------------|----------------|
| Net Debt (MM): | €5.6 |
| Long-Term Debt (MM): | €14.6 |
| Cash & ST Invest. (MM): | €10.7 |
| Market Data | |
| 52 Week Range: | €12.40 - €5.11 |
| Total Entprs. Value (MM): | NM |
| Market Cap. (MM): | NM |
| Shares Out. (MM): | 17.3 |
| Float (MM): | 14.1 |
| Avg. Daily Vol.: | 202,139 |

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Price Performance



^Prior trading day's closing price unless otherwise noted.

KDS NA Initiating Coverage

31 January 2018

Kiadis

Buy: €22 Price Target

Scenarios

Base Case

- Novel protocols such as ATIR drive ongoing growth of haploidentical HSCT given "half matched" donors are readily available and GvHD risks can be mitigated
- We forecast \$475m peak ATIR sales in US+EU assuming 20% penetration of haplo-ID HSCT procedures, with Kiadis commercialising the product itself in these regions for a highly profitable opportunity
- Price Target €22/share comprising NPVs for ATIR in the US and Europe plus Net Cash, less potential dilution to ensure sufficient funds until YE2020E

Investment Thesis / Where We Differ

Our financial model suggests the €11m cash at 30 June 2017, together with €18m gross proceeds from the October private placement and €10m net debt financing from Kreos Capital in August, is sufficient to fund cash burn at least into early-2019E. Importantly this is beyond the EU CHMP opinion and conditional approval, in addition potentially to the Phase III HATCY interim analysis

Upside Scenario

- EU regulatory approval of ATIR could add c.€5/share
- Positive Phase III HATCY results for ATIR could boost our sum-of-the-parts by at least €6/share
- Higher 30% peak ATIR penetration in both the US and Europe could add €21/share
- These potential catalysts could boost our NPV derived Price Target to €54/share, still including the potential dilution to ensure sufficient funds until YE2020E

Catalysts

- EU CHMP opinion on ATIR for haploidentical HSCT is likely at the September or October meetings
- EU conditional approval of ATIR during 1Q19E
- Updates on patient enrolment in the ATIR Phase III study
- Interim analysis of the Phase III HATCY trial around YE18-1H19E, with final results 1H20E

Downside Scenario

- EU regulatory rejection or a significantly delayed opinion of ATIR could remove at least €10/share
- If the Phase III HATCY study for ATIR fails this could lower our sum-of-the-parts by at least €11/share
- Lower 10% peak ATIR penetration in both the US and Europe could remove around €20/share
- These potential setbacks could reduce our NPV derived Price Target to a negligible value

Long Term Analysis

2018E Net Cash (€m)

Long Term Financial Model Drivers 2016-21E Revenue CAGR n/m 2016 Net Cash (€m) (2.6) 2017E Net Cash (€m) 3.0

(21.8)

Initiating Coverage

31 January 2018

Investment Summary

Kiadis develops innovative cell therapies for safer and more effective bone marrow transplants. Its sole clinical product ATIR improves haploidentical "half-matched" stem cell transplants and may expand their use, providing an important anti-cancer effect and ability to fight infections, while also reducing the life-threatening risk of graft versus host disease (GvHD). We anticipate an EU CHMP opinion by 4Q18E based on Phase II data, for conditional approval during 1Q19E. The recently initiated Phase III HATCY study is likely to have an interim analysis around YE18-1H19E, with final results 1H20E, for potential US launch by 2022E. We forecast \$475m peak sales in US+EU assuming 20% penetration, with Kiadis commercialising ATIR itself for a highly profitable opportunity. Current cash is only sufficient until early-2019E, in our view, but by this time ATIR should be EU approved, hence despite this overhang we are initiating with a Buy rating given an NPV-based Price Target of €22 per share suggests substantial potential upside.

Jefferies acted as Sole Bookrunner for the October 2017 private placement raising c.€18m gross proceeds from issuing 2.25m new shares at €8 per share.

HSCT can be curative: Haematopoietic stem cell transplants (HSCT) can offer a cure for some serious disorders but autologous (own cells) procedures are frequently precluded by the patients' health, requiring use of an allogeneic donor, which also provides a valuable graft versus leukaemia (GvL) benefit. Matched relatives or unrelated donors can be challenging to find, hence the appeal of widely available "half matched" haploidentical HSCT. Their use is rapidly expanding, aided by protocols that mitigate the life-threatening risk of GvHD, but these typically also subdue GvL and anti-infective effects, raising the risk of relapse and complications. ATIR may minimise GvHD while retaining the benefits.

Nearing green light for ATIR to boost HSCT: We forecast haplo-ID HSCT to more than double by 2026E to over 6k and 4k in the EU and US, respectively, mostly driven by its increasing share, plus a +3.5% market CAGR. Assuming 20% peak ATIR penetration with €150k and \$250k average Revenue/patient we derive \$240m and \$235m peak sales, with a potential incremental \$75m in RoW markets excluded from base case estimates.

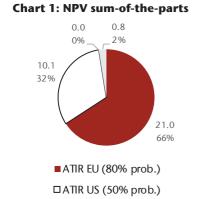
"Best" case ATIR peak sales could near-\$2bn: This assumes novel protocols like ATIR drive faster growth of haplo-ID HSCT for 50% more procedures, peak penetration reaches 40%, cost:benefit justifies higher average Revenue/patient of €250k/\$350k, and ATIR is adopted for the c.11% of HSCT outside of treating blood cancers.

Valuation

Our €22 per share Price Target is based on a sum-of-the-parts valuation comprising probability-adjusted NPVs for ATIR in the US and Europe, together with Net Cash, less potential dilution to ensure sufficient funds until YE2020E using the most aggressive forecasts for cash burn, excluding any potential collaboration income.

Risks

- Clinical or regulatory setbacks: Efficacy or safety concerns in the ATIR Phase III HATCY study could significantly dent our NPVs, with slower patient enrolment also potentially trimming forecasts. A rejection or regulatory delays by the European CHMP regulatory authority would have a substantial adverse impact.
- Commercial execution risks: Kiadis and/or potential future partners will need to successfully navigate the potentially complex price/reimbursement environment, persuade physicians to change their current treatment paradigm, and mitigate possible competitive threats.
- Securing adequate funds to maximise value: Our forecasts suggest current cash is sufficient until early-2019E, likely beyond the EU regulatory decision and potential Phase III interim analysis, but incremental funds are required to complete the pivotal trial and build-out a commercial infrastructure.



Source: Jefferies estimates excluding adjustment for potential dilution to ensure sufficient funds to YE2020E

■ Net Cash

■ATIR RoW (0% prob.)

Initiating Coverage

31 January 2018

Shining a light on stem cell transplants

Kiadis develops innovative cell therapies for safer and more effective bone marrow transplants. Its sole clinical product ATIR is produced by the selective depletion of the donor's alloreactive T cells and is then administered to patients (the host) after a haploidentical "half-matched" stem cell transplant to provide an important anti-cancer effect and ability to fight infections, while also reducing the life-threatening risk of graft versus host disease (GvHD). We anticipate a European CHMP opinion by 4Q18E based on Phase II data, for potential conditional approval during 1Q19E and launches from 2H19E. The recently initiated Phase III HATCY study is likely to have an interim analysis around YE18-1H19E, with final results 1H20E, for potential US launch by 2022E. We forecast \$240m and \$235m peak sales in Europe and the US, respectively, assuming 20% penetration with Kiadis commercialising ATIR itself for a highly profitable opportunity.

Depleting T cells from the graft prior to a haploidentical transplant cuts the risk of GvHD, as T cells from the non-identical donor recognise the recipient tissues (the host) as foreign. However, donor T cells are also beneficial, essential for the "graft versus leukaemia" effect killing residual tumour cells and also enabling the patient to fight infections. ATIR consists of a "safe" subset of T cells to be given to patients after a T cell depleted stem cell transplantation from a haploidentical donor to provide these benefits but still mitigate the risk of GvHD without the need for prophylactic immunosuppressants. The initial indication is adult leukaemias, as blood cancers represent c.89% of transplant procedures.

- Peak sales: \$240m in Europe, \$235m in the US, and potentially a conservative \$75m in other markets, which we currently exclude pending visibility on possible commercial strategies
- NPV: c.€21 per share for Europe and c.€10 per share for the US assuming 80% and 50% probabilities of success, respectively
- News flow: European CHMP opinion by 4Q18E for conditional approval during 1Q19E; updates on enrolment of the Phase III HATCY study with an interim analysis likely around YE18-1H19E

Key considerations when evaluating the ATIR Phase III and future adoption

- We believe Phase III is adequately powered based on Phase II: The pivotal trial's primary endpoint is GvHD and relapse free survival at 12 months, known as GRFS, versus the standard-of-care post-transplant cyclophosphamide (PTCy or "Baltimore protocol"). In the ATIR Phase II '007 trial the GRFS was 13/23 patients (57%), whereas based on literature reports we estimate the GRFS using PTCy to be around 37%. The Phase III HATCY study is 80% powered for a 20% difference, hence we are optimistic ATIR can demonstrate a statistically significant benefit over PTCy.
- Assume survival rates are similar but other benefits significant: Literature suggests one-year survival using PTCy is broadly in the range of 60%, around the figure reported for ATIR in the Phase II study. We do not believe ATIR needs to demonstrate a survival benefit to be adopted given the important clinical relevance of a significantly lower GRFS. Phase II data suggest ATIR has the potential to substantially reduce the incidence of chronic GvHD, which is associated with high morbidity-mortality, and relapse compared to PTCy, in addition to lower rates of acute GvHD.
- Risk of higher drop-out rate pre-transplant in ATIR cohort: Eligible patients and donors enrolled in the Phase III randomised to the ATIR arm receive apheresis 14 days prior to the HSCT conditioning, as during this period Kiadis manufactures the product. In this two-week period there may be a risk patients'

Initiating Coverage

31 January 2018

health deteriorates or they withdraw from the study, amongst other scenarios, thereby failing to receive a transplant. This could confound analysis of ATIR's efficacy compared to that of PTCy.

- Patient enrolment may be slower than anticipated: After initiating the Phase III in December 2017, Kiadis must successfully activate the clinical trial sites for physicians to then commence screening patients. We envisage it may take up to two years to fully enrol the study but delays to this timeline could adversely impact our forecasts.
- Challenges changing the standard-of-care: It often can be more challenging to drive adoption of a new procedure compared with a novel drug, in our view. ATIR is expected to be an outpatient product infused after hospitalisation for haploidentical HSCT but its use first requires clinicians to perform apheresis of both the patient and donor around 14 days prior to the conditioning regimen. In contrast, haploidentical HSCT using the "Baltimore protocol" can be initiated shortly after a donor is available. Furthermore, physicians using PTCy typically administer steroids as a standard-of-care if there are signs of GvHD, which should be avoided when using the ATIR protocol.
- New therapies could perhaps drive a decline in HSCT: Recently launched drugs and potential future generations of treatments, including modalities such as CAR T, could substantially improve response rates and survival. In theory, this could reduce the number of HSCT procedures performed, particularly given the relative convenience of administering a novel drug. We regard this to be a fairly unlikely near-term scenario as transplants are well established, offer patients a possible cure, and new therapies may be used as a bridge to a successful HSCT.

Potential sources of upside to our base case forecasts for ATIR

- **Greater proportion of patients able to undergo HSCT:** We understand up to 35% of patients eligible for HSCT are unable to find a matched donor and fail to receive a transplant. Adoption of PTCy has driven growth of haploidentical procedures, both cannibalising use of matched related and unrelated donor (MRD and MUD) transplants but also expanding the market. Novel treatment protocols could further accelerate use of haploidentical HSCT, as "half-matched" donors are readily available for most patients, thereby boosting the +3.5% market CAGR we forecast based on the recent trend. We assume the current trend of more widespread use of haploidentical donors continues, almost doubling as a proportion of procedures from 2017E to 2030E.
- **Higher penetration of ATIR for haploidentical HSCT:** Our peak penetration is only 20% in both the US and Europe. We believe the most significant challenge to ATIR adoption is likely to be PTCy, given the need to change the current paradigm (as discussed above), rather than emerging competitive threats, such as Zalmoxis and BPX-501.
- Higher price per ATIR transplant: Our estimates of average revenue per patient around €150k in Europe and \$250k in the US could prove conservative, particularly given possible competitor Zalmoxis recently secured a reimbursement price in Italy of €149k per infusion and in Germany of €163,900 per infusion. As an outpatient drug infused after HSCT hospitalisation, we envisage ATIR to be billed separately to payers, rather than bundled into the total fee for the transplant procedure.
- Use for indications beyond blood cancers: If ATIR proves to be a safe and effective product for haploidentical HSCT of patients with blood cancers then we envisage longer-term it would also likely be adopted for transplants treating other disorders, such as β-thalassemia, sickle cell disease, severe aplastic anaemia, and primary immune deficiencies. Around 11% of HSCT procedures are for these indications.

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KDS NA Initiating Coverage 31 January 2018

€22 Price Target using NPV sum-of-the-parts

Similar to other biotech stocks in our coverage universe, we believe the most appropriate valuation methodology for Kiadis is a fundamental NPV sum-of-the-parts. Hence, our Price Target comprises NPVs for ATIR in both the US and Europe, in addition to Net Cash. We then adjust our valuation to reflect the potential dilution from a capital increase to ensure sufficient funds until at least YE2020E. Data and/or potential out-licensing deals could crystallise significant value, and provide upside to our valuation.

Table 1: Kiadis sum-of-the-parts valuation

| | | Peak | Value | Adj. Valu | | e EUR | |
|--------------------------------|------------------------------|--------------|---------|-----------|---------|-----------|--|
| | Indication | Sales (\$mn) | (EURmn) | Prob. | (EURmn) | per share | |
| ATIR101 | Haploidentical HSCT (Europe) | 240 | 455 | 80% | 364 | 21.0 | |
| | Haploidentical HSCT (US) | 235 | 350 | 50% | 175 | 10.1 | |
| | Haploidentical HSCT (RoW) | 75 | 47 | 0% | 0 | 0.0 | |
| Net Cash/(Debt) | | | 14 | 100% | 14 | 0.8 | |
| Valuation | | | 866 | | 553 | 32.0 | |
| Potential Dilution for Funding | Min. Yrs of Cash | 3.0 | | 43% | (64) | (9.7) | |
| Potential Diluted Valuation | | | | | | 22.3 | |

Source: Jefferies estimates

Table 2: Sources of upside potential and downside risk

| rubie zi sources or apside potentiar a | | | |
|--|----------------------------|-------------------------------|-----------|
| | | EUR | EUR |
| | Upside | per share Downside | per share |
| ATIR EU regulatory decision | Approved | 5.3 Rejected or delayed | (10.5) |
| ATIR Phase III HATCY results | Positive | 6.1 Fails | (11.3) |
| ATIR peak penetration (20% base case) | Higher 30% peak in US & EU | 21.0 Only 10% peak in US & EU | (20.7) |
| Potential Upside/(Downside) | | 32.3 | (42.6) |
| Potential Valuation | | 64.3 | (10.6) |

Source: Jefferies estimates. Note our Long View scenarios include potential dilution for funding

Adequate funds to reach European approval decision

Our financial model suggests the €11m cash reported at 30 June 2017, together with the c.€18m gross proceeds from the recent private placement in October and €10m net debt financing from Kreos Capital in August, is sufficient to fund cash burn at least into early-2019E. We do not include possible future out-licensing deals in our base case forecasts. Importantly current cash should be sufficient to fund burn until beyond the European CHMP opinion and conditional approval, in addition to the Phase III HATCY interim analysis dependent on the rate of patient recruitment.

We include within cash flow forecasts the repayments due to Kreos Capital for the €15m debt facility at a 10% annual fixed interest rate. The first tranche of €10m is interest only for the first nine months from August 2017, before then amortising equally in monthly instalments for the remaining 36 months. The second €5m tranche, triggered by raising at least €20m additional funds before 1 July 2018, is interest-only for the first 12 months, before then amortising equally in monthly instalments over the remaining 36 months. At the time of the agreement, Kreos Capital also received 253,617 warrants for new shares in Kiadis at an initial exercise price of €6.36. Pursuant to the terms of the facility, we assume the debt is repaid during 2018-21E.

Valuation modestly depressed by license fees

The Theralux platform on which ATIR is based utilises intellectual property and know-how that was originally licensed from the University of Montreal, Canada. Pursuant to the terms of the agreement(s), Kiadis owes a 5% royalty on global sales of products using Theralux such as ATIR.

During 2010, Hospira (now part of Pfizer) licensed rights to ATIR in specific geographies, but this agreement was terminated in 2012 and all rights returned. Kiadis has obligations totalling \$26m at YE15 increasing 1.5% per annum, repayable via a \$3m milestone due

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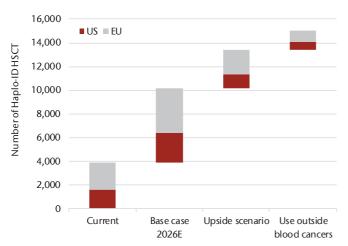
31 January 2018

on a sublicense or first commercial sale, and 5% royalties on sales. Once repaid Kiadis owes Hospira a 3% royalty on sales outside North America, South America, and China, for a total 8% long-term royalty stack on sales in these territories.

"Best" case scenario suggests ATIR peak sales could near-\$2bn

Our "best" case assumes novel protocols drive more rapid growth of haploidentical HSCT, ATIR penetration peaks at 40% not 20%, and average Revenue per patient is a higher \$350k/€250k. Furthermore, we also assume if ATIR proves to be safe and provides a significant clinical benefit for patients versus current standard-of-care, then it could also be adopted for haploidentical HSCT of diseases other than blood cancers. Overall under this scenario we envisage around 50% more haploidentical HSCT are performed around the time of ATIR peak penetration, with nearly 6,000 in the US and over 9,000 in Europe. We believe these could represent a realistic upside scenario given the number of HSCT overall, excluding autologous transplants, is expected to surpass 11,000 in the US and 20,000 in Europe.

Chart 2: "Best" case upside scenario suggests ATIR peak sales could near-\$2bn in the US and Europe combined



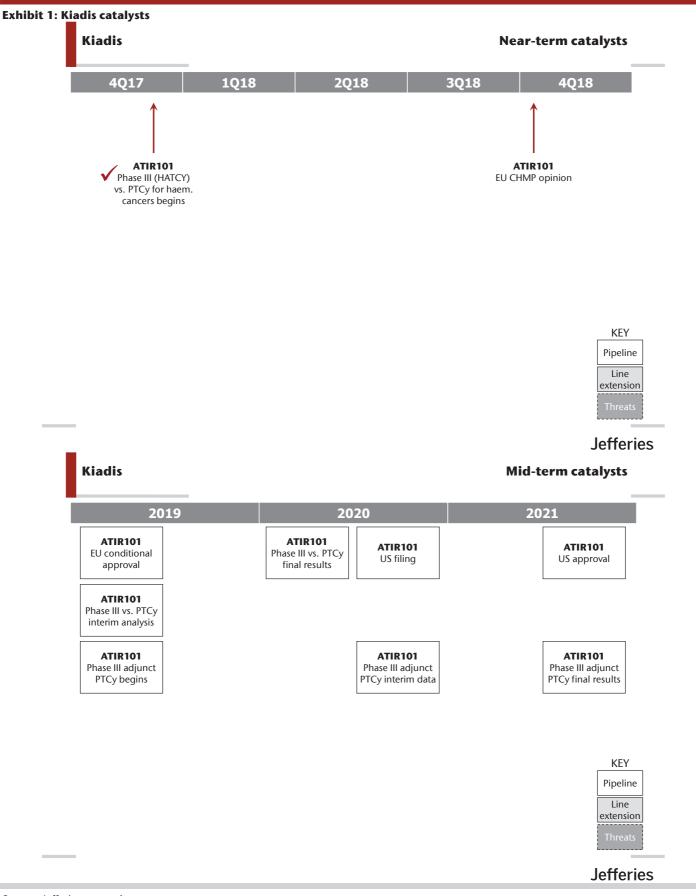
| Peak upside scenario | | | | | | | | |
|----------------------|-----------------------------|--------|--------|--|--|--|--|--|
| US (\$m) | 5,950 halpo-ID HSCT at peak | | | | | | | |
| | Av. Revenue/Patient | | | | | | | |
| Penetration | \$250k | \$300k | \$350k | | | | | |
| 20% | 295 | 355 | 415 | | | | | |
| 30% | 445 | 535 | 625 | | | | | |
| 40% | 595 | 710 | 830 | | | | | |

| EU (\$m) | 9,100 halpo-ID HSCT at peak | | | | | | | |
|-------------|-----------------------------|---------------------|---------|--|--|--|--|--|
| | Av. | Av. Revenue/Patient | | | | | | |
| Penetration | EUR150k | EUR200k | EUR250k | | | | | |
| 20% | 335 | 450 | 560 | | | | | |
| 30% | 505 | 675 | 840 | | | | | |
| 40% | 675 | 895 | 1,120 | | | | | |

| US+EU (\$m) | Av. Revenue/Patient | | | | | | |
|-------------|---------------------|-------|-------|--|--|--|--|
| Penetration | Base case | | | | | | |
| 20% | 630 | 805 | 975 | | | | |
| 30% | 950 | 1,210 | 1,465 | | | | |
| 40% | 1,270 | 1,605 | 1,950 | | | | |
| | | | | | | | |

Source: Jefferies estimates

KDS NA Initiating Coverage 31 January 2018



Source: Jefferies research

Initiating Coverage

31 January 2018

ATIR nearing the green light in Europe

ATIR is produced by the selective depletion of the donor's alloreactive T cells and is administered to patients after a haploidentical "half matched" stem cell transplant to provide an important anti-cancer effect and ability to fight infections, while also reducing the risk of graft versus host disease. We anticipate a European CHMP opinion by 4Q18E based on Phase II data, for potential conditional approval during 1Q19E and launches from 2H19E. The recently initiated Phase III HATCY study is likely to have an interim analysis around YE18-1H19E, with final results 1H20E, for potential US launch by 2022E. We forecast \$240m and \$235m peak sales in Europe and the US, respectively, assuming 20% penetration for around 1,200 and 900 haplo-ID transplants. We assume Kiadis commercialises ATIR itself in the US and Europe, focusing on the c.120+ main transplant centres in each geography, for a highly profitable opportunity given the relatively small target patient populations. "Best" case our scenario analysis suggests peak sales could near \$2bn if novel protocols drive more rapid growth of haploidentical HSCT, ATIR penetration reaches 40%, the clinical benefit commands a higher price, and use extends beyond blood cancers.

- Peak sales: \$240m in Europe, \$235m in the US, and potentially a conservative
 \$75m in other markets, which we currently exclude pending visibility on possible commercial strategies
- NPV: c.€21 per share for Europe and c.€10 per share for the US assuming 80% and 50% probabilities of success, respectively
- News flow: European CHMP opinion by 4Q18E for conditional approval during 1Q19E; updates on enrolment of the Phase III HATCY study with an interim analysis likely around YE18-1H19E

ATIR consists of a "safe" subset of T cells to be given to patients after stem cell transplantation to provide an important graft versus tumour effect and the ability to fight infections, but lowering the risk of graft versus host disease without the need for prophylactic immunosuppressants. The initial indication is adult leukaemias, as blood cancers represent c.89% of transplant procedures.

Stem cell transplants can be curative

Haematopoietic stem cell transplantation (HSCT) is an established treatment for a number of conditions, including malignant haematological diseases and other blood disorders. HSCT can offer a cure for many of these life-threatening conditions. An intravenous infusion of haematopoietic stem and progenitor cells is designed to establish marrow and immune function in patients. There are three types of HSCT based on the source of the stem cells.

- Autologous HSCT blood stem cells are taken from the recipient's own body
- Allogeneic HSCT blood stem cells from a related (siblings, parents, cousins, etc.) or unrelated donor
- Syngeneic HSCT blood stem cells are derived from an identical twin so is essentially the same as an autologous procedure.

The use of autologous stem cells is mainly limited by the patients' own well-being and absence of malignant/diseased cells to contaminate the graft.

Allogeneic HSCT requires procuring bone marrow from a matched donor, i.e. a person with matched or partially matched human leukocyte antigen (HLA). HLA proteins are found on the surface of most cells and identify tissue type. There are a limited number of

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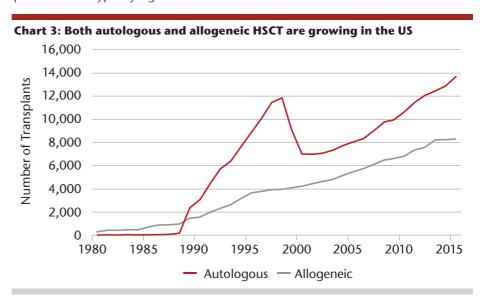
31 January 2018

alleles of the HLA system, hence matched donors can be related to the patient, or found in international registries and be unrelated to the patient. These are known as a matched related donor (MRD) or unrelated donor (MUD).

Haploidentical donor transplants are the only option for patients without an HLA-matched donor as half-matched stem cells are used. Most patients have a family donor who is at least a 50% HLA match, hence haploidentical HSCT (haplo-HSCT) is becoming increasingly common as protocols/techniques are developed to mitigate its potentially serious shortfall; risk of graft-versus-host disease (GvHD).

The main advantage of allogeneic HSCT for haematologic malignancies is the profound therapeutic effect mediated by the donor T cells, which can eradicate residual cancer cells in the recipient; 'graft versus leukaemia' (GvL) phenomenon (also known as graft versus tumour). Given this benefit, the donor alloreactive T cells may be depleted prior to the HSCT, reducing the risk of GvHD, with a donor lymphocyte infusion (DLI) then administered at a later timepoint to reinstall GvL activity. A longer time interval between transplantation and DLI lowers the risk of GvHD but raises the risk of a relapse. The complete removal of donor T cells also abrogates the recipient's ability to fight a variety of viruses soon after transplant, such as cytomegalovirus, Epstein-Barr virus, varicella zoster, and others.

Stem cells can be derived from three sources; (1) bone marrow, (2) peripheral blood, and (3) umbilical cord blood. Over time peripheral blood has become more commonly used than bone marrow due to its ease of extraction and high cell count. However, the risk of GvHD is greater with peripheral blood transplants. Studies suggest cord blood does not have to be as closely matched as bone marrow or peripheral blood, albeit the cost of procurement is typically higher.



Source: Jefferies research based on CIBMTR summary slides 2016

Haplo-HSCT use may be impaired by the high risk of GvHD with unmanipulated grafts, or increased non-engraftment, slow immune reconstitution and infections if T cell depletion is employed

KDS NA Initiating Coverage 31 January 2018

Exhibit 2: Comparison of allogeneic versus autologous stem cell transplantation

| Allogeneic | Autologous |
|--|--|
| Advantages | |
| No tumor contamination risk or damage from prior chemo Graft versus tumor effect Useful for patients with bone marrow dysfunction | Donor not required if peripheral bone marrow is healthy No immunosuppression lowering infection risk No GvHD Dose-intensive regimen can be used for older patients |
| Disadvantages | |
| Dose-intensive regimen has toxicities limiting use Limited availability and/or long wait for donor Greater risk from GvHD and infectious complications | Not feasible if peripheral blood stem cells or marrow involved No graft versus tumor effect Not all patients can be mobilised to give adequate cell counts |

Source: Jefferies research

Exhibit 3: Comparison of MUD HSCT with haplo-HSCT

MUD Haplo

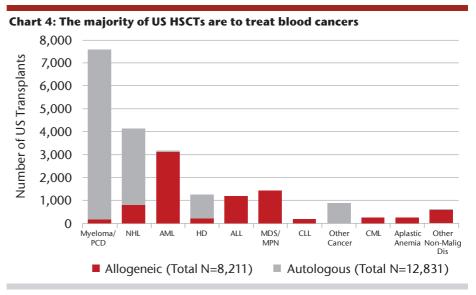
Donor availability 20%-80% (ethnic variation) >95%

Time to graft acquisition Slower via registries Faster

Time from collection to infusion Longer if shipped Shorter

Ease of repeat donations May be challenging Easier

Source: Jefferies research



Source: Jefferies research based on CIBMTR summary slides 2016

Initiating Coverage

31 January 2018

GvHD is a major cause of transplantrelated mortality, limiting the success of HSCT; in patients who are resistant to steroids, morbidity and mortality is significant

GvHD a significant obstacle to successful HSCT

Acute and chronic graft-versus-host disease (GvHD) are immune-mediated complications of allogeneic HSCT. GvHD is a major cause of non-relapse related mortality, limiting the success of HSCT in a considerable proportion of patients, whilst also inducing substantial morbidity affecting quality of life.

GvHD occurs when T cells from the non-identical donor (the graft), recognise the recipient tissues (the host) as foreign, initiating an immune reaction that causes disease in the transplant recipient, principally targeting the skin, gastrointestinal (GI) tract and liver.

Standard treatment is with steroids, however if it does not resolve and the disease is steroid-resistant (SR-GvHD), the morbidity and mortality rate is high, estimated to be up to 80%. Thus, there is an urgent clinical need to develop treatments for patients suffering from SR-GvHD whilst maintaining the graft versus tumour effect required to prevent a potential rise in relapse-related mortality.

Grading of aGvHD includes a stage between 1 and 4 for the individual organs, which are then combined for an overall grade, from I to IV.

| Exhibit 4: Grading of acute GvHD | | | | | | | |
|----------------------------------|---|------|---------------------|------|---------------------------------|--|--|
| | | Exte | nt of organ involve | emer | nt | | |
| Stage | Skin | | Liver (bilirubin) | | GI (stool output/day) | | |
| 0 | No GvHD rash | | <2mg/dL | | <500ml/day or persistent nausea | | |
| 1 | Maculopapular rash <25% BSA | | 2-3mg/dL | | 500-999ml/day | | |
| 2 | Maculopapular rash 25-50% BSA | | 3.1-6mg/dL | | 1000-1500ml/day | | |
| 3 | Maculopapular rash >50% BSA | | 6.1-15mg/dL | | >1500ml/day | | |
| 4 | Generalised erythema plus bullous formation | | >15mg/dL | | Severe abdominal pain +/- ileus | | |
| Grade | Skin | | Liver (bilirubin) | | GI (stool output/day) | | |
| 1 | Stages 1-2 | | None | | None | | |
| II | Stage 3 | or | Stage 1 or | or | Stage 1 | | |
| III | - | | Stage 2-3 | or | Stages 2-4 | | |
| IV | Stage 4 | or | Stage 4 | | | | |

Source: Jefferies research. Note: BSA is body surface area

No standard of care for steroid resistant aGvHD

Despite prophylactic measures, aGvHD remains relatively common. The incidence of aGvHD ranges from 10-80% depending upon several variables, including the donor type (related versus unrelated, matched versus mismatched or haploidentical), the type of conditioning, the donor's sex and the stem cell source (peripheral blood versus bone marrow). Response rates to initial treatment with steroids are under 50% for patients with grade II-IV GvHD, and yet there remains no standard of care or approved drug for second-line treatment. Second-line agents generally fall into broad categories, including cytostatic agents (mycophenolate, pentostatin), immunomodulating agents (mTor inhibitors, extracorporeal photopheresis) and biologic therapies (alemtuzumab, infliximab, denileukin). In this second-line treatment setting, response rates have generally been much lower, around 20% to 30%.

There is no consensus on treatment for the c.50% of patients with steroid-resistant disease

¹ Garnett C, et al. (2013) Treatment and management of graft-versus-host disease: improving response and survival. Therapeutic Advances in Haematology; 4(6):366-378

² Drobyski WR, et al. (2011). Tocilizumab for the treatment of steroid refractory graft-versus-host disease. Biol Blood Marrow Transplant; 17(12):1862-1868

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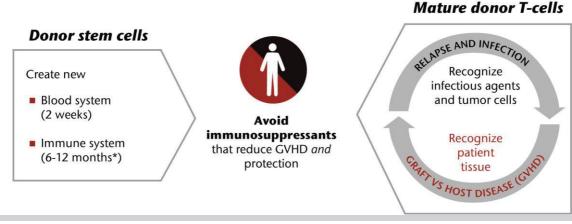
31 January 2018

Balance of risk versus benefits required

The complete abrogation of GvHD following T cell depleted-HSCT (most commonly by CD34+ selection) illustrates the pivotal role of donor-derived T cells in aGvHD.³ However, donor T cells are also beneficial, and in fact are essential for the 'graft versus leukaemia' (GvL) phenomenon (also known as graft versus tumour); a powerful component of allogeneic HSCT treatment contributing to the eradication of residual leukaemia cells. Donor T cells also play a key role in mediating reconstitution of the patient's adaptive immune system. Accordingly, although reducing the rate of GvHD, T cell depleted HSCT has not led directly to improved survival due to infections secondary to delayed immune reconstitution, graft rejection and increased rates of disease relapse.⁴

Thus, a careful balance is required for a successful GvHD treatment, namely sufficient impact to mitigate GvHD but permitting enough immune function to provide GvL and control infection, and ultimately must not lead to an increase in disease-relapse.

Exhibit 5: The challenge is to mitigate the risk of GvHD while still gaining the benefits of donor T-cells' GvL effects



Source: Jefferies research. * T-cells typically have low survival without mature donor T-cells

Posttransplant cyclophosphamide gaining traction

Posttransplant cyclophosphamide (PTCy) involves administering the chemotherapeutic soon after the HSCT to reduce the incidence of GvHD without the need for T cell depletion. The selective action of cyclophosphamide is on rapidly proliferating T cells, such as donor and host alloreactive CD4+ cells, while sparing others and enabling a rapid immune reconstitution to possibly preserve the GvL effect.

O'Donnell et al at John Hopkins University in Baltimore conducted a clinical trial that set the stage for the use of PTCy, hence its alternative name as the "Baltimore protocol"⁵. Patients undergoing haploidentical HSCT received a T cell replete graft followed by cyclophosphamide from day-3 in combination with other immunosuppressants (tacrolimus, mycophenolate mofetil [MMF], etc).

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³ Hatano R, et al. (2013). Prevention of acute graft-versus-host disease by humanised anti-CD26 monoclonal antibody. British Journal of Haematology; 162:236-277

⁴ Booth C, et al. (2013) T cell depletion in paediatric stem cell transplantation. Clin Exp Immunol; 172(2):139-147

⁵ P.V. O'Donnell (2002). Nonmyeloablative bone marrow transplantation from partially HLAmismatched related donors using posttransplant cyclophosphamide. Biol Blood Marrow Transplant, 8 (2002); 377-386

Initiating Coverage

31 January 2018

Importantly comparisons of haplo-HSCT using PTCy with MUD HSCT suggests similar overall outcomes, with perhaps lower incidences of chronic GvHD given the use of cyclophosphamide⁶.

In the US, use of unrelated donor (MUD) transplants surpassed related donors (MRD) in 2006 as the former increasingly dominated allogeneic HSCTs, likely driven by the prevalence of donor registries and cord blood inventories, together with improved outcomes. However, since 2012 the numbers of MUD and MRD transplants have been in decline, driven by a rise in the use of haploidentical HSCT. This is likely driven by the use of PTCy protocols. A similar trend is evident in Europe.

Chart 5: Retrospective comparison of outcomes with haplo PTCy vs. MUD

60%

50%

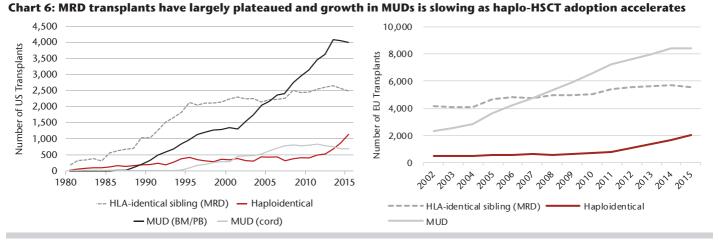
40%

30%

10%

Acute GvHD II-IV Chronic GvHD Relapse Survival

Source: Jefferies research based on Fuchs, 2017. Averages are estimated across the studies and indications despite different durations of follow-up and patient populations.



Source: Jefferies research based on EBMT publications and CIBMTR 2016 summary slides

Jefferies

⁶ E Fuchs (2017). Related haploidentical donors are a better choice than matched unrelated donors: Point. Blood advances

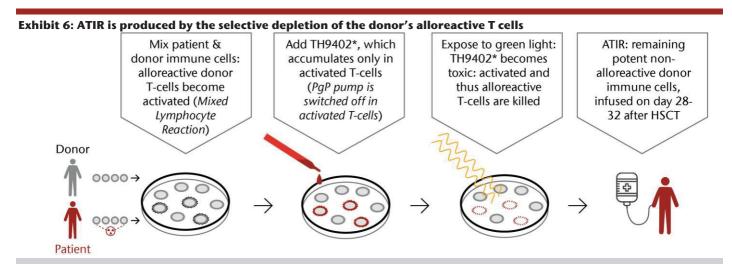
Initiating Coverage

31 January 2018

ATIR: Selective depletion of T cells

Kiadis manipulates the donor immune cells using a mixed lymphocyte reaction, i.e. mixing the donor and host's immune cells resulting in the alloreactive donor T cells becoming activated, recognising the host's cells as foreign. A proprietary selective rhodamine derivative, TH9402, is then added which selectively accumulates only in activated T cells, as their Pgp (permeability glycoprotein 1) cell membrane pump is switched off. TH9402 becomes cytotoxic under green light, hence by exposing the mixture of immune cells the donor's alloreactive T cells are killed. The remaining donor T cells retain populations with GvL effect and the ability to fight infections. The process takes around five days, of which two involve active operations, and is performed in simple clean rooms with laminar airflow cabinets. The final product is frozen in liquid nitrogen and the ATIR is infused on day 28-32 after the HSCT.

Kiadis leased an existing commercial manufacturing facility in Amsterdam, The Netherlands during December 2017. We understand this is equipped to produce gene therapies, with process development and quality control laboratories installed. Existing contract manufacturing agreements will be maintained but this in-house site allows significant future capacity expansion, with only minimal capex investments.

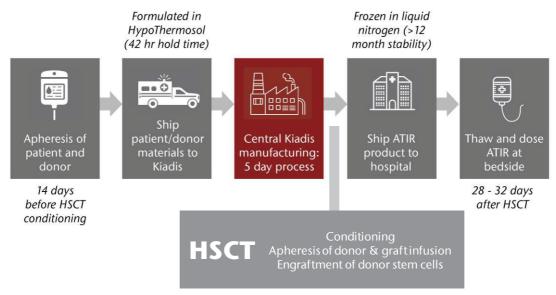


Source: Jefferies research. * TH9402 is a proprietary selective rhodamine derivative

Initiating Coverage

31 January 2018

Exhibit 7: ATIR can be readily incorporated into the protocol for HSCT



Source: Jefferies research

ATIR101 for blood cancers filed in Europe with Phase III just initiated

Kiadis' focus is to pursue ATIR for blood cancers, initially adult acute leukaemia % of HSCT. ATIR is currently being investigated in the Phase III HATCY study (CR-AIR-009) but has been filed for conditional approval in Europe based on the Phase II (CR-AIR-007) efficacy trial, plus a CR-AIR-006 analysis versus a matched historical control group. The European Marketing Authorisation Application was filed in April 2017, with day-120 questions received in September. We anticipate a CHMP opinion by 4Q18E for potential approval during 1Q19E and launches from 2H19E.

Kiadis receive FDA Regenerative Medicine Advanced Therapy (RMAT) designation in September 2017, which should enable more regular interactions with the regulatory authority and increases the likelihood of an expedited review process.

The Phase III trial has been designed after consultations with both FDA at an end-of-Phase II meeting and the EMA to support full approval. The first patient was enrolled in early-December 2017 and the study is anticipated to take at least two years to be fully recruited for final results during 1H20E. An interim analysis at 50% of planned events is likely to occur around YE18-1H19E.

Phase II results supported EU filing for conditional approval

The Phase II (CR-AIR-007) was an open-label, single-arm study enrolling 23 AML/ALL patients at four sites in Europe and Canada during 2013-16. All subjects received a CD34+ (T cell depleted) haplo-HSCT followed by a single dose of 2m cells/kg ATIR with no prophylactic immunosuppression.

Overall survival at 12 months was 14/23 61%, with two relapses (9%) and non-relapse mortality (NRM) of 7/23 30%. There were no acute GvHD of Grade III-IV, with only three cases (13%) of Grade II, and one episode (4%) of chronic GvHD.

Initiating Coverage

31 January 2018

Exhibit 8: Detailed ATIR Phase II (CR-AIR-007) study results

Population Adult ALL & AML

In 1st remission with high-risk features or in 2nd+ remission

HSCT HID CD34+ T-cell depleted (CliniMACS)

N 23 (16 AML & 7 ALL)
Cytogenetic risk profile 9 (39%) intermediate; 14 (61%) adverse
Disease risk index 10 (43%) intermediate; 13 (57%) high
Sites 4 Europe & Canada

Administration Days 28-32
Dose 2m cells/kg

GvHD prophylaxis None

Immune reconstitution 100% Median time (days) 12

At 12 months

Overall survival 60.9% 14/23 with 2 deaths on relapse, 5 TRM infections & 2 TRM other

Relapse 8.7% 2/23
Non-relapse mortality (NRM) 30.4% 7/23
Acute GvHD grade III-IV 0.0% 3/23 grade II
Chronic GvHD moderate-severe 4.3% 1 patient

GRFS 56.5%

Source: Jefferies research

On longer follow-up with all patients assessed to 24 months there have been another two relapses reported and three transplant-related mortality events due to infections (pneumonia, sepsis and septic shock) for 9/23 39% overall survival. We note all three cases of death due to infections were in immunosuppressed patients, two of which received donor lymphocyte infusions and then developed severe acute GvHD, and the other being the subject with chronic GvHD.

A historical observational cohort of 35 patients, matched by indication and clinical trial site to those in '007, was selected under a protocol supported by EMA scientific advice. Overall survival at 12 months for this historical control cohort was 20%, suggesting a favourable comparison versus ATIR.

A second Phase II study (CR-AIR-008) investigated the effect of a second ATIR infusion around day 70-74, designed to prolong the protection from transplant-related mortality. However, after 11 of the planned 15 patients were enrolled, with six subjects having received two ATIR infusions, it was reported that some patients administered two doses developed severe acute GvHD, compared to no cases after only a single dose. Consequently, the remaining four patients are to be treated with only a single dose as per protocol. To our knowledge no definitive explanation has been presented for this outcome, but it is perhaps not unreasonable to propose the study's original rationale was flawed as T cells likely persist for at least six months, negating the benefit of a second ATIR dose. It may be the case that two infusions represent an "overdose", raising the risk of alloreactive donor T cells to cause acute GvHD.

Phase III initiated in December 2017

The Phase III HATCY trial aims to enrol 195 adult patients with acute leukaemia at 40-50 sites in the US, Canada and Europe. Subjects are randomised 1:1 to receive either a CD34+ HSCT followed by a single dose of 2m cells/kg ATIR at day 28-32, or a T-cell replete HSCT with post-transplant cyclophosphamide and immunosuppressants (PTCy, the "Baltimore protocol").

The primary endpoint is GvHD and relapse-free survival, known as GRFS and defined as survival without chronic GvHD requiring immunosuppression, acute GvHD Grade III-IV, or relapse. The study is event-driven with a primary analysis at 93 GRFS events and an interim analysis planned at the halfway point, when the sample size could be adjusted if

Initiating Coverage

31 January 2018

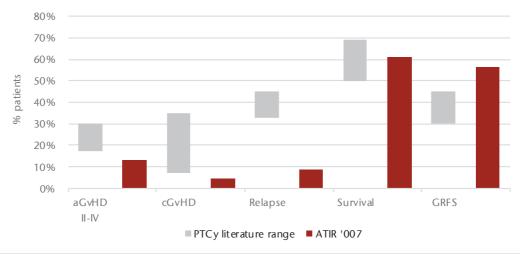
deemed necessary. The trial is 80% powered for a 20% difference in GRFS with ATIR versus PTCy assuming approximately 60% vs. 40%.

Secondary endpoints include overall survival, progression-free survival, relapse-related mortality, and transplant-related mortality.

Key considerations when evaluating the Phase III and future adoption

• We believe Phase III is adequately powered based on Phase II: GRFS at 12 months in the Phase II '007 trial was 13/23 57%. Based on literature reports we estimate the GRFS using PTCy to be around 37% on average. We note for the five patients with sufficient follow-up after receiving only a single ATIR dose in the CR-AIR-008 study the 12-month GRFS is 80% (4/5), hence combining the '007 and '008 trials the GRFS with ATIR rises to 61% (17/28). Based on these Phase II data and literature reports for PTCy, we are optimistic ATIR can demonstrate a statistically significant benefit in GRFS over PTCy to meet the Phase III primary endpoint assuming the trial is 80% powered for a 20% difference.





Source: Jefferies research; PTCy results from McCurdy et al 2017, Ciurea et al 2015, Ciurea et al 2012, Devillier et al 2016, Di Stasi et al 2014, Solh et al 2016, Sugita et al 2015, Solomon et al 2012

- Assume survival rates are similar but other benefits significant: Literature suggests one-year OS using PTCy is broadly in the range of 60%, around the figure reported for ATIR in the Phase II study. We do not believe ATIR needs to demonstrate a survival benefit to be adopted given the important clinical relevance of a significantly lower GRFS. Phase II data suggest ATIR has the potential to substantially reduce the incidence of chronic GvHD and relapse compared to PTCy, in addition to lower rates of acute GvHD. We envisage a benefit on chronic GvHD to be particularly important for patients, physicians, and payers. The five-year mortality rate for severe chronic GvHD is around 50% and there is typically a profound adverse impact on quality of life. Literature reports for PTCy suggest the risk of moderate-severe cGvHD is around 20%-25% compared with 4% (one patient) in the ATIR Phase II study.
- Risk of higher drop-out rate pre-transplant in ATIR cohort: Eligible patients and donors enrolled in the Phase III randomised to the ATIR arm receive apheresis 14 days prior to the HSCT conditioning, as during this period Kiadis manufactures the product. In this two-week period there may be a risk patients' health deteriorates or they withdraw from the study, amongst other scenarios, thereby failing to receive a transplant. This could confound analysis of ATIR's efficacy compared to that of PTCy by reducing reported outcomes, as under the

Initiating Coverage

31 January 2018

pre-specified intent-to-treat (ITT) analysis any patient enrolled in the trial will be included within the calculations. We understand a modified ITT analysis will also be performed in the subpopulation that received a transplant.

- Patient enrolment may be slower than anticipated: After initiating the Phase III in December 2017, Kiadis must successfully activate the clinical trial sites for physicians to then commence screening patients. We envisage it may take up to two years to fully enrol the study but delays to this timeline could adversely impact our forecasts.
- Challenges changing the standard-of-care: Typically, we believe it can be more challenging to drive adoption of a new procedure compared with a novel drug. ATIR is expected to be an outpatient product infused after hospitalisation for haploidentical HSCT but its use first requires clinicians to perform apheresis of both the patient and donor around 14 days prior to the conditioning regimen and then elect a CD34+ T-cell deplete transplant. In contrast haploidentical HSCT using the "Baltimore protocol" can be initiated shortly after a donor is available. Furthermore, physicians using PTCy typically administer steroids as a standard-of-care if there are signs of GvHD, which should be avoided when using the ATIR protocol.
- New therapies could perhaps drive a decline in HSCT: Recently launched drugs and potential future generations of treatments, including modalities such as CAR T, could substantially improve response rates and survival. In theory, this could reduce the number of HSCT procedures performed, particularly given the relative convenience of administering a novel drug. We regard this to be a fairly unlikely near-term scenario as transplants are well established, offer patients a possible cure, and new therapies may be used as a bridge to a successful HSCT.

Significant potential market opportunity

We estimate over 2,700 haploidentical HSCT for blood cancers will be performed in Europe this year and over 1,800 in the US. We forecast the number of HSCT procedures for blood cancers to continue expanding at around a +3.5% CAGR, with an increasing proportion utilising haploidentical donors. Our model assumes the proportion of haploidentical donors steadily rises to double its current adoption, from c.6.5% 2017E in Europe to around 14% by 2030E, and from over 8% in the US to c.17% over the same period. This represents a +12% five-year CAGR 2018-23E of haploidentical HSCT procedures for blood cancers in both regions, which could prove conservative given the +19% EU and +23% US five-year CAGR 2013-18E.

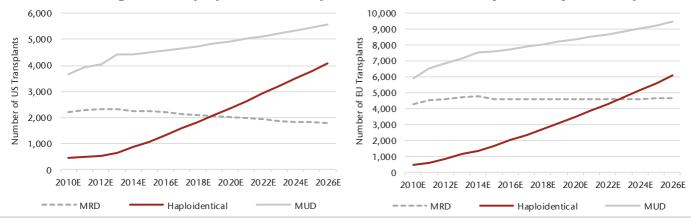
Our base case peak penetration for ATIR in Europe and the US is 20% in 2026E and 2027E, respectively. We believe this fairly reflects the potential challenges in driving physicians to shift from established treatment protocols, notably PTCy, in addition to possible competitive threats (see later). We estimate average Revenue per patient to Kiadis of €150k in Europe and \$250k in the US.

We forecast \$240m peak sales in Europe and \$235m in the US, reflecting the greater number of transplants performed in the EU, which more than offsets a lower list price. In other international markets, we provisionally estimate \$75m peak sales assuming a modest 10% penetration and \$150k average Revenue per patient. We assume Kiadis would likely partner ATIR in these regions, significantly reducing the profitability of sales. Pending visibility on the commercial strategy for international markets we assign no NPV to these regions in our sum-of-the-parts valuation.

Initiating Coverage

31 January 2018

Chart 8: We forecast growth of haplo procedures to outpace the rise in HSCT driven by use of PTCy and other protocols



Source: Jefferies estimates with historical figures based on EBMT publications and CIBMTR 2016 summary slides

| Table 3: ATTRIVI | giodai sales model and | ilicensing obligations |
|------------------|------------------------|------------------------|
| | | |

| (EUR millions Dec YE) | 2018E | 2019E | 2020E | 2021E | 2022E | 2023E | 2024E | 2025E | 2026E | 2027E |
|---|------------|---------|---------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|
| EU Haploidentical HSCT for Blood Cancers | 2,716 | 3,092 | 3,481 | 3,883 | 4,298 | 4,726 | 5,167 | 5,622 | 6,090 | 6,571 |
| Patient Growth | 15.4% | 13.8% | 12.6% | 11.5% | 10.7% | 10.0% | 9.3% | 8.8% | 8.3% | 7.9% |
| ATIR101 Penetration | | 0.8% | 3.1% | 6.2% | 9.6% | 13.7% | 17.1% | 19.0% | 20.0% | 20.0% |
| ATIR EU Patients | | 24 | 108 | 242 | 412 | 647 | 884 | 1,068 | 1,218 | 1,314 |
| Average Revenue per Patient p.a. (EUR) | | 150,000 | 150,000 | 150,000 | 150,000 | 150,000 | 150,000 | 150,000 | 150,000 | 150,000 |
| ATIR101 EU Sales (EURmn) | | 3.6 | 16.2 | 36.3 | 61.7 | 97.0 | 132.5 | 160.2 | 182.7 | 197.1 |
| US Haploidentical HSCT for Blood Cancers | 1,836 | 2,100 | 2,369 | 2,643 | 2,922 | 3,205 | 3,491 | 3,781 | 4,074 | 4,369 |
| Patient Growth | 16.4% | 14.4% | 12.8% | 11.6% | 10.5% | 9.7% | 8.9% | 8.3% | 7.7% | 7.2% |
| ATIR101 Penetration | | | | 2.3% | 5.7% | 9.6% | 13.7% | 17.1% | 19.0% | 20.0% |
| ATIR US Patients | | | | 61 | 168 | 307 | 478 | 647 | 774 | 874 |
| Average Revenue per Patient p.a. | | | | \$250,000 | \$250,000 | \$250,000 | \$250,000 | \$250,000 | \$250,000 | \$250,000 |
| ATIR101 US Sales (\$mn) | | | | 15.2 | 42.0 | 76.7 | 119.4 | 161.6 | 193.5 | 218.4 |
| RoW Haploidentical HSCT for Blood Cancers | 1,357 | 1,665 | 2,003 | 2,353 | 2,716 | 3,092 | 3,481 | 3,883 | 4,298 | 4,726 |
| Patient Growth | 20.2% | 22.7% | 20.3% | 17.5% | 15.4% | 13.8% | 12.6% | 11.5% | 10.7% | 10.0% |
| ATIR101 Penetration | | | | 1.1% | 2.9% | 4.8% | 6.8% | 8.6% | 9.5% | 10.0% |
| ATIR RoW Patients | | | | 27 | 78 | 148 | 238 | 332 | 408 | 473 |
| Average Revenue per Patient p.a. | | | | \$150,000 | \$150,000 | \$150,000 | \$150,000 | \$150,000 | \$150,000 | \$150,000 |
| ATIR101 RoW Sales (\$mn) | | | | 4.1 | 11.7 | 22.2 | 35.7 | 49.8 | 61.2 | 70.9 |
| ATIR101 WW Sales (\$mn) | | 4.4 | 20.0 | 63.8 | 129.6 | 218.2 | 318.1 | 408.5 | 479.5 | 531.8 |
| Licensing Obligations | | | | | | | | | | |
| % Sales Paid to University of Montreal | | 5.0% | 5.0% | 5.0% | 5.0% | 5.0% | 5.0% | 5.0% | 5.0% | 5.0% |
| % Sales Paid to Hospira to Repay Loan | | 5.0% | 5.0% | 5.0% | 5.0% | 5.0% | 5.0% | 5.0% | 5.0% | 5.0% |
| Hospira Loan Outstanding 1-Jan incl 1.5% inc p | .a. (\$mn) | 27.2 | 27.4 | 26.8 | 23.9 | 17.7 | 6.9 | 0.0 | 0.0 | 0.0 |
| % Ex-NA/SA/China Sales Paid to Hospira After Loa | n | 3.0% | 3.0% | 3.0% | 3.0% | 3.0% | 3.0% | 3.0% | 3.0% | 3.0% |
| Kiadis Milestone to Hospira on Launch/License (El | JRmn) | (2.4) | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Kiadis EU Licensing Obligations (EURmn) | | (2.8) | (1.6) | (3.6) | (6.2) | (9.7) | (12.2) | (12.8) | (14.6) | (15.8) |
| Kiadis US Licensing Obligations (EURmn) | | 0.0 | 0.0 | (1.2) | (3.4) | (6.2) | (4.9) | (6.6) | (7.9) | (8.9) |
| Kiadis RoW Licensing Obligations (EURmn) |) | 0.0 | 0.0 | (0.3) | (1.0) | (1.8) | (1.5) | (3.0) | (3.7) | (4.3) |

Source: Jefferies estimates

ATIR has Orphan Drug Designation in both the US and Europe, which ensures at least seven and ten years' market exclusivity, respectively. We understand the composition patent "P015" including methods for reducing GvHD expires in October 2021, with patent "P019" on rhodamine derivatives extending to January 2024, and method of use IP "P016" until around December 2024 if granted. Given Orphan Drug protection is likely to extend beyond these patent lifetimes, only potential "P040" covering an improved photodynamic process is worth consideration, in our view, which if granted could expire around February 2036. We believe the greater barriers to entry for possible future "generic" versions of ATIR may be the proprietary "know how" for the manufacturing

Initiating Coverage

31 January 2018

process, release assays, and cell handling procedures. Given these hurdles, our NPVs assume around a 20-year commercial lifetime for ATIR from launch in all geographies.

Potential sources of upside to our forecasts

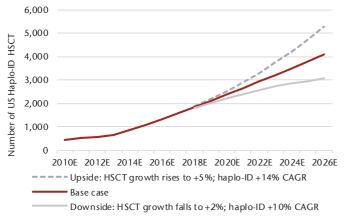
Our "best" case scenario suggests ATIR peak sales could near \$2bn in the US and Europe combined on assuming novel protocols drive more rapid growth of haploidentical HSCT, ATIR penetration of 40% from 20%, and higher \$350k/€250k average Revenue per patient. We also assume if ATIR proves to be safe and provides a significant clinical benefit for patients versus current standard-of-care, then it could also be adopted for haploidentical HSCT of diseases other than blood cancers. Overall our "best" case scenario assumes around 50% more haploidentical HSCT are performed around the time of ATIR peak penetration, with nearly 6,000 in the US and over 9,000 in Europe. We believe these could represent a realistic upside scenario given the number of HSCT overall, excluding autologous transplants, is expected to surpass 11,000 in the US and 20,000 in Europe.

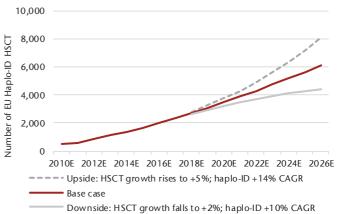
- **Greater proportion of patients able to undergo HSCT:** We understand up to 35% of patients eligible for HSCT are unable to find a matched donor and fail to receive a transplant. Novel treatment protocols could enable a proportion of this population to receive a HSCT given haploidentical donors are readily available, thereby boosting the +3.5% market CAGR.
- Accelerating growth of haploidentical HSCT driven by new protocols:
 We assume the current trend of more widespread use of haploidentical donors
 continues, almost doubling as a proportion of procedures from 2017E to 2030E.
 This rate may prove overly conservative.
- Penetration of ATIR for haploidentical HSCT: Our peak penetration is only 20% in both the US and Europe. We believe the most significant challenge to ATIR adoption is likely to be PTCy, given the need to change the current paradigm (as discussed above), rather than emerging competitive threats.
- Higher price per ATIR transplant: Our estimates of average Revenue per patient around €150k in Europe and \$250k in the US could prove conservative, particularly given possible competitor Zalmoxis (see below) recently secured a reimbursement price in Italy of €149k per infusion and in Germany of €163,900 per infusion. As an outpatient drug infused after HSCT hospitalisation, we envisage ATIR to be billed separately to payers, rather than bundled into the total fee for the transplant procedure (i.e. a Medicare Part B J-code rather than inclusion within DRG codes in the US). Hospitals could potentially save on lower use of cyclophosphamide and related complications, such as relapse, haemorrhagic cystitis, and GvHD.
- Use for indications beyond blood cancers: If ATIR proves to be a safe and effective product for haploidentical HSCT of patients with blood cancers then we envisage longer-term it would also likely be adopted for transplants treating other disorders, such as β-thalassemia, sickle cell disease, severe aplastic anaemia, and primary immune deficiencies. Around 11% of HSCT procedures are for these indications.

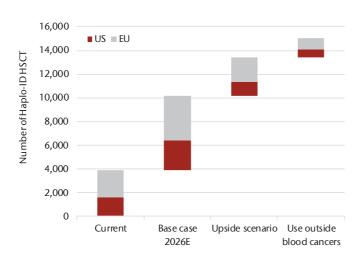
Initiating Coverage

31 January 2018

Chart 9: Scenario analysis for ATIR peak sales in US/Europe and "best" case upside







| Peak upside scenario | | | | | | | | |
|----------------------|-----------------------------|--------|--------|--|--|--|--|--|
| US (\$m) | 5,950 halpo-ID HSCT at peak | | | | | | | |
| | Av. Revenue/Patient | | | | | | | |
| Penetration | \$250k | \$300k | \$350k | | | | | |
| 20% | 295 | 355 | 415 | | | | | |
| 30% | 445 | 535 | 625 | | | | | |
| 40% | 595 | 710 | 830 | | | | | |

| EU (\$m) | 9,100 halpo-ID HSCT at peak | | | | | | | |
|-------------|-----------------------------|---------------------|---------|--|--|--|--|--|
| | Av. | Av. Revenue/Patient | | | | | | |
| Penetration | EUR150k | EUR200k | EUR250k | | | | | |
| 20% | 335 | 450 | 560 | | | | | |
| 30% | 505 | 675 | 840 | | | | | |
| 40% | 675 | 895 | 1,120 | | | | | |

| US+EU (\$m) | Av. Revenue/Patient | | | | | | |
|-------------|---------------------|-------|-------|--|--|--|--|
| Penetration | Base case | | | | | | |
| 20% | 630 | 805 | 975 | | | | |
| 30% | 950 | 1,210 | 1,465 | | | | |
| 40% | 1,270 | 1,605 | 1,950 | | | | |

 $Source: Jefferies\ estimates\ with\ historical\ HSCT\ figures\ based\ on\ EBMT\ publications\ and\ CIBMTR\ 2016\ summary\ slides$

Potential future development plans

- Combined with "Baltimore protocol": In future Kiadis may initiate a trial investigating ATIR as an adjunctive treatment to PTCy, i.e. after the "Baltimore protocol". Patients would receive a T cell replete HSCT, with ATIR infused at a later timepoint. In theory, we envisage this may reduce relapse rates given the benefit of ATIR, albeit without mitigating the c.25% acute GvHD rates that are typical using PTCy.
- Indications beyond blood cancers: We do not believe Kiadis is likely to pursue development of ATIR for indications outside haematological malignancies (named ATIR201) in the near future, despite the Phase I/II protocol in β-thalassemia major paediatric patients being approved in a few European countries. It seems strategically rational to initially prioritise pursuing a paediatric investigation plan in blood cancers to maximise the opportunity for ATIR101, particularly given c.85% of HSCT are for these indications.

Initiating Coverage

31 January 2018

Potential competitive threats

We caution against comparing across clinical trials, particularly when Phase I/II studies across a selective number of clinical sites and in relatively small patient populations. Nevertheless, it is perhaps instructive to consider the relative key efficacy metrics and safety concerns for the key competitive threats to ATIR. From a market-access perspective, we note the ATIR Phase III study directly compares the therapy versus a standard-of-care PTCy, providing data which we do not believe will be available for competitors.

| | ATIR101 | Zalmoxis (TK cells) | | BPX-501 & rimiducid | | |
|------------------------------|-----------------------|-------------------------|---------------|---------------------|--------------------|--|
| Trial | CR-AIR-007 | TK007 | TK007+008 | BP-004 | (update) | |
| Population | Adult | Adult | Adult | Paediatric | | |
| | ALL & AML | ALL, AML & other mali | gnancies | ALL & AML | | |
| HSCT HID | CD34+ T-cell depleted | In vivo TCD & B cell de | epletion | abTCR/CD19+ B | cell-depletion | |
| N | 23 | 30 (excl 22 untreated) | 37 | 47 | 112 (53 malignant) | |
| Sites | 4 EU/Can | 7 EU & 1 Israel | | 3 EU | | |
| Administration | Days 28-32 | Day 21+ (median 43) | (median ~40) | Day 14+/-4 | | |
| Dose | 2m cells/kg | 10m cells/kg up to 4 n | nonthly doses | 1m cells/kg | | |
| GvHD prophylaxis | None | None | None | None | None | |
| Immune reconstitution | 100% | 77% | | 100% | | |
| Median time (days) | 12 | 31 | | 16 | | |
| At 12 months | | | | | | |
| Overall survival | 61% | 40% | 49% | 89% | 85% | |
| Leukaemia free survival | | | 37% | 83% | | |
| Relapse | 9% | 33% | 41% | 15% | | |
| Non-relapse mortality (NRM) | 30% | 30% | 22% | 3% | | |
| Acute GvHD grade III-IV | 0% | 7% | 9% | 5% | | |
| Chronic GvHD moderate-severe | 4% | 3% | 6% | 3% | | |

Source: Jefferies research. Zalmoxis data from presentation at ASH December 2014 and EMA assessment report June 2016. BPX-501 results reported at EHA June 2017 and updated at ASH December 2017

BPX-501 T cells engineered with suicide safety switch

Bellicum Pharmaceuticals (BLCM, \$8.7, Buy) utilises its Chemical Induction of Dimerisation (CID or CaspaCIDe) platform to introduce safety switches to control toxicities of T cell based immunotherapies. The CaspaCIDe switch is controlled by the administration of rimiducid. BPX-501 is an adjunct T cell therapy with the CaspaCIDe switch for patients undergoing allogeneic HSCT. Upon occurrence of GvHD the CaspaCIDe switch is activated by administering rimiducid causing apoptosis of activated donor T cells, for resolution of the GvHD. BPX-501 T cells are injected 14±4 days after the HSCT.

The Phase I CASPALLO study investigated BPX-501 in 10 paediatric patients undergoing allogeneic HSCT. Rimiducid administration resolved GvHD within 24-48 hours in four patients who developed the condition, with nearly 90% of the BPX-501 T cells depleted within two hours of its use. There was no recurrence of acute or chronic GvHD in three surviving patients after long-term follow-up.

Data from the ongoing Phase I/II BP-004 trial in 112 paediatric patients were reported at the EHA conference in June and the recent ASH clinical meeting, 9-12 December 2017. Patients received BPX-501 after an α/β T cell and CD19+ B cell depleted haplo-HSCT.

Final results from the Phase I/II BP-004 study, plus a comparator observation study in MUD transplants, could form the basis of a European regulatory filing. For the US, a controlled clinical trial in adults with AML is planned.

Initiating Coverage

31 January 2018

Zalmoxis T cells engineered with suicide gene

MolMed (MLM IT, €0.59, NC) produces TK by genetic engineering of T cells from the allogeneic HSCT donor using a retroviral vector to express a truncated form of the human low affinity nerve growth factor receptor (∆LNGFR) and the Herpes Simplex I virus thymidine kinase (HSV-TK Mut2). The HSV-TK suicide gene makes the cells sensitive to antiviral ganciclovir/valganciclovir. At the onset of GvHD, ganciclovir/valganciclovir can then by administered to kill the donor T cells. The cells are modified to express ∆LNGFR for identification purposes. TK is administered around 40 days after the HSCT to promote a sustained reconstitution of the host's immune system. Up to four monthly doses can be administered until immune reconstitution (CD3+ cell count over 100/mcl).

The Phase I/II TK007 study in 52 adult patients with haematological malignancies investigated TK after haplo-HSCT. Immune reconstitution was achieved in 23 of the 30 patients treated with TK in a median time of 21 days after the last TK infusion. Patients received 1-4 infusions of TK cells with a median of two. Acute GvHD was reported in 10/30 patients, with all but two cases being Grade I-II, and there was one episode of severe chronic GvHD. Treatment of acute GvHD was required in all but one case, with full control of the clinical manifestations achieved in all nine patients either when treated with ganciclovir/valganciclovir alone (n=3) or in combination with standard immunosuppressives (n=6).

A randomised Phase III trial TK008 is progressing slowly in adult leukaemia patients undergoing halpo-HSCT but TK received conditional approval in Europe during August 2016 as Zalmoxis. This was based on a matched-pair analysis using EBMT data for 37 TK treated patients (23 from TK007 and 14 from TK008). MolMed partnered with Dompé (private) in July 2017 to commercialise Zalmoxis in Europe. A reimbursement price of €149k per infusion was recently established in December 2017 with the Italian AIFA, and c.€164k during January 2018 for Germany.

Other competing technologies for selective T-cell depletion

For example, Miltenyi Biotec (private) has the automated CliniMACS Cell Separation System using immunomagnetic beads. CliniMACS CD34+ cell enrichment has been used for over 20 years to manipulate grafts for HSCT. More recently innovative T cell depletion strategies have been developed to maintain graft facilitating and anti-infective functions. Recent data presented at the ASH clinical conferences 2016 & 2017 demonstrated the favourable survival rates and absence of severe GvHD using a $TCR\alpha/\beta$ and CD19 depleted graft, also enriched for natural killer (NK) and $TCR\gamma/\delta$ effector cells. We note MMF was used for GvHD prophylaxis until day-30.

These innovative T cell depletion techniques still remove $TCR\alpha/\beta$, which likely adversely impacts the beneficial GvL effect. We find conflicting reports on the capabilities of $TCR\gamma/\delta$ effector cells to produce a GvL effect but NK cells do have a role.

Initiating Coverage

31 January 2018

Financial Models

Table 4: Kiadis Profit and Loss Model

| | | 2017 | | | | | | |
|--|----------|---------|---------|---------|---------|----------|---------|--------------|
| (EUR millions except EPS Dec YE) | 2016A | 1H17A | 2H17E | 2017E | 2018E | 2019E | 2020E | 202 1 |
| ATIR EU Sales | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 3.6 | 16.2 | 36. |
| ATIR US Sales | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 12. |
| License & Other Revenue | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0. |
| Revenue | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 3.6 | 16.2 | 48. |
| Cost of Sales | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | (4.2) | (7.1) | (18. |
| Gross Profit | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | (0.6) | 9.1 | 29. |
| otal Operating Expenses | (11.4) | (8.2) | (9.4) | (17.5) | (23.6) | (26.7) | (27.1) | (38 |
| R&D Expenses | (8.2) | (5.9) | (6.7) | (12.6) | (16.3) | (15.5) | (10.8) | (9 |
| General & Admin. Expenses | (3.2) | (2.3) | (2.6) | (4.9) | (7.0) | (7.7) | (8.3) | (8 |
| Sales & Marketing Expenses | 0.0 | 0.0 | 0.0 | 0.0 | (0.3) | (3.5) | (8.0) | (20 |
| o/w Acquisition-related Amortisation/Write-dow | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0 |
| Other Operating Income | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0 |
| perating Exceptionals | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0 |
| perating Income | (11.4) | (8.2) | (9.4) | (17.5) | (23.6) | (27.4) | (18.0) | (8. |
| Adjusted Operating Income | (11.4) | (8.2) | (9.4) | (17.5) | (23.6) | (27.4) | (18.0) | (8. |
| let Financial Income | (3.4) | (0.4) | (1.8) | (2.2) | (2.7) | (14.4) | (8.3) | (12 |
| xceptionals | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | C |
| ncome from Associates & JVs | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0 |
| Pretax Profit | (14.8) | (8.5) | (11.1) | (19.7) | (26.3) | (41.8) | (26.3) | (21. |
| Adjusted Pretax Profit | (14.8) | (8.5) | (11.1) | (19.7) | (26.3) | (41.8) | (26.3) | (21. |
| axation | (0.0) | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0 |
| linority Interests | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | C |
| let Income from Continuing Operations | (14.8) | (8.5) | (11.1) | (19.7) | (26.3) | (41.8) | (26.3) | (21 |
| let Income from Discontinued Operations | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0 |
| let Income | (14.8) | (8.5) | (11.1) | (19.7) | (26.3) | (41.8) | (26.3) | (21. |
| Adjusted Net Income | (14.8) | (8.5) | (11.1) | (19.7) | (26.3) | (41.8) | (26.3) | (21. |
| VA Basic Shares (mn) | 13.8 | 14.0 | 17.3 | 15.6 | 17.3 | 17.3 | 17.3 | 17 |
| VA Shares Diluted (mn) | 13.8 | 14.0 | 17.3 | 15.6 | 17.3 | 17.3 | 17.3 | 17 |
| PS (EUR) | (1.1) | (0.6) | (0.6) | (1.3) | (1.5) | (2.4) | (1.5) | (1. |
| Adjusted EPS (EUR) | (1.1) | (0.6) | (0.6) | (1.3) | (1.5) | (2.4) | (1.5) | (1. |
| Piluted EPS (EUR) | (1.1) | (0.6) | (0.6) | (1.3) | (1.5) | (2.4) | (1.5) | (1 |
| Piluted Adjusted EPS (EUR) | (1.1) | (0.6) | (0.6) | (1.3) | (1.5) | (2.4) | (1.5) | (1. |
| 6 Change Year over Year | | | | | | | | |
| evenue | n/a | n/a | n/a | n/a | n/a | n/a | 350.3% | 199.1 |
| ost of Sales | n/a | n/a | n/a | n/a | n/a | n/a | 68.5% | 164.4 |
| Gross Profit | n/a | n/a | n/a | n/a | n/a | n/a | 1533.6% | 226.3 |
| otal Operating Expenses | (28.7%) | 61.4% | 47.3% | 53.5% | 35.0% | 13.1% | 1.3% | 41.7 |
| R&D Expenses | 6.4% | 54.7% | 52.9% | 53.7% | 29.5% | (4.9%) | (30.7%) | (12.0 |
| General & Admin. Expenses | (61.4%) | 81.8% | 34.6% | 53.0% | 42.9% | 10.0% | 8.0% | 7.0 |
| Sales & Marketing Expenses | n/a | n/a | n/a | n/a | n/a | 1066.7% | 128.6% | 150.0 |
| perating Income | 28.7% | (61.4%) | (47.3%) | (53.5%) | (35.0%) | (15.8%) | 34.3% | 51.7 |
| djusted Operating Income | 28.7% | (61.4%) | (47.3%) | (53.5%) | (35.0%) | (15.8%) | 34.3% | 51.7 |
| let Financial Income | (652.2%) | 73.8% | 10.0% | 36.3% | (23.0%) | (442.1%) | 42.0% | (47.9 |
| retax Profit | 10.1% | (32.2%) | (33.6%) | (33.0%) | (33.6%) | (58.8%) | 37.0% | 20.2 |
| djusted Pretax Profit | 10.1% | (32.2%) | (33.6%) | (33.0%) | (33.6%) | (58.8%) | 37.0% | 20.2 |
| Net Income | 10.1% | (32.2%) | (33.5%) | (33.0%) | (33.6%) | (58.8%) | 37.0% | 20.2 |
| djusted Net Income | 10.1% | (32.2%) | (33.5%) | (33.0%) | (33.6%) | (58.8%) | 37.0% | 20.2 |
| PS (EUR) | 21.2% | (27.5%) | (8.1%) | (17.0%) | (20.8%) | (58.8%) | 37.0% | 20.2 |
| Adjusted EPS (EUR) | 21.2% | (27.5%) | (8.1%) | (17.0%) | (20.8%) | (58.8%) | 37.0% | 20.2 |

Source: Jefferies estimates; company data

Initiating Coverage

31 January 2018

Table 5: Kiadis Cash Flow Model

| 2. Kiduis Casii How Model | | | | | | |
|---|--------|--------|--------|--------|--------|--------|
| (EUR millions Dec YE) | 2016A | 2017E | 2018E | 2019E | 2020E | 2021E |
| Operating Income | (11.4) | (17.5) | (23.6) | (27.4) | (18.0) | (8.7) |
| Depreciation and Amortisation | 0.2 | 0.2 | 0.2 | 0.3 | 0.5 | 0.7 |
| EBITDA | (11.3) | (17.3) | (23.4) | (27.1) | (17.5) | (8.0) |
| Other Adjustments and Exceptionals | 0.4 | 0.9 | 1.2 | 1.3 | 1.4 | 1.5 |
| Decrease/(Increase) in Inventories | 0.0 | 0.0 | 0.0 | (0.3) | (0.2) | (1.0) |
| Decrease/(Increase) in Receivables | (0.1) | 0.0 | 0.0 | (0.6) | (2.1) | (5.3) |
| Increase/(Decrease) in Payables | (2.7) | 0.9 | 0.8 | 0.8 | 2.5 | 8.1 |
| Increase/(Decrease) in Deferred Income | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Change in WC | (2.8) | 0.9 | 0.8 | (0.2) | 0.1 | 1.8 |
| Taxation Paid | (0.0) | (0.0) | 0.0 | 0.0 | 0.0 | 0.0 |
| Interest Paid | (0.7) | (1.8) | (1.5) | (4.0) | (8.0) | (12.0) |
| Net Cash Flow from Operating Activities | (14.3) | (17.3) | (22.9) | (29.9) | (24.0) | (16.7) |
| Purchase of Tangible Fixed Assets | (0.3) | (0.1) | (0.7) | (1.0) | (1.5) | (2.4) |
| Proceeds from Sale of PP&E | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Purchase of Intangible Assets | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| (Purchase)/Sale of Investments | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| (Acquisitions)/Disposals of Subsidiaries | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Dividends Received from Associates | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Interest Received | 0.1 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 |
| Net Cash Flow from Investing Activities | (0.2) | 0.0 | (0.7) | (1.0) | (1.5) | (2.4) |
| Management of Liquid Resources | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Capital Changes | 1.6 | 23.4 | 0.0 | 0.0 | 0.0 | 0.0 |
| Debt Changes | (1.2) | 8.9 | (2.1) | 29.7 | 25.8 | 21.0 |
| Equity Dividends Paid | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Other Financing Cash Flows | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Net Cash Flow from Financing Activities | 0.4 | 32.3 | (2.1) | 29.7 | 25.8 | 21.0 |
| Effect of FX on Cash and Cash Equivalents | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Increase in Cash | (14.1) | 15.0 | (25.7) | (1.3) | 0.3 | 1.9 |
| Change in Net Debt | 13.0 | (6.0) | 23.6 | 30.9 | 25.5 | 19.1 |
| (Cash Burn) | (14.6) | (17.3) | (23.6) | (30.9) | (25.5) | (19.1) |

Source: Jefferies estimates; company data

Initiating Coverage

31 January 2018

Table 6: Kiadis Balance Sheet Model

| ie 6: Kiadis Baiance Sneet Model | | | | | | |
|--|--------|---------|---------|---------|---------|---------|
| (EUR millions Dec YE) | 2016A | 2017E | 2018E | 2019E | 2020E | 2021E |
| Non-current Assets | 14.1 | 14.0 | 14.5 | 15.2 | 16.2 | 18.0 |
| Intangible Assets | 13.5 | 13.5 | 13.5 | 13.5 | 13.5 | 13.5 |
| Property, Plant and Equipment | 0.5 | 0.5 | 0.9 | 1.6 | 2.7 | 4.4 |
| Investments | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Other Long-term Assets | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Current Assets | 15.1 | 30.1 | 4.4 | 4.0 | 6.6 | 14.8 |
| Inventories | 0.0 | 0.0 | 0.0 | 0.3 | 0.6 | 1.6 |
| Trade Accounts Receivable | 0.0 | 0.0 | 0.0 | 0.6 | 2.7 | 8.0 |
| Other Current Assets | 0.6 | 0.6 | 0.6 | 0.6 | 0.6 | 0.6 |
| Cash and Cash Equivalents | 14.6 | 29.5 | 3.8 | 2.5 | 2.8 | 4.7 |
| Total Assets | 29.2 | 44.1 | 18.8 | 19.2 | 22.8 | 32.7 |
| Current Liabilities | 4.2 | 3.5 | 4.3 | 39.9 | 73.9 | 108.6 |
| Trade Accounts Payable | 1.3 | 2.1 | 2.9 | 3.8 | 4.2 | 7.0 |
| Other Current Liabilities | 0.7 | 0.7 | 0.7 | 0.7 | 0.7 | 0.7 |
| Accrued Expenses | 0.7 | 0.7 | 0.7 | 0.6 | 2.7 | 8.0 |
| Deferred Income | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Short-term Debt | 1.6 | 0.0 | 0.0 | 34.9 | 66.4 | 92.9 |
| Leasing Obligations | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Dividends | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Non-current Liabilities | 15.6 | 26.5 | 25.6 | 30.8 | 25.3 | 20.2 |
| Long-term Debt | 15.6 | 26.5 | 25.6 | 30.8 | 25.3 | 20.2 |
| Leasing Obligations | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Deferred Tax Liabilities | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Deferred Income | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Long-term Provisions | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Total Shareholders' Equity | 9.4 | 14.0 | (11.1) | (51.5) | (76.5) | (96.0) |
| Share Capital | 1.4 | 1.4 | 1.4 | 1.4 | 1.4 | 1.4 |
| Share Premium Account | 103.2 | 126.1 | 125.0 | 114.6 | 114.2 | 113.9 |
| Other Reserves and Adjustments | 0.3 | 0.3 | 0.3 | 0.3 | 0.3 | 0.3 |
| Retained Earnings | (95.5) | (113.8) | (137.7) | (167.8) | (192.4) | (211.6) |
| Minority Interests | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Total Liabilities and Shareholders' Equity | 29.2 | 44.1 | 18.8 | 19.2 | 22.8 | 32.7 |

Source: Jefferies estimates; company data

KDS NA Initiating Coverage 31 January 2018

Company overview

Kiadis develops innovative cell therapies for safer and more effective bone marrow transplants. Its Allodepleted T cell ImmunotheRapeutics (ATIR) are based on the Theralux platform. Lead programme ATIR101 is filed in Europe for haploidentical haematopoietic stem cell transplants (HSCT) in patients with blood cancers. Kiadis is based in The Netherlands and listed on the Euronext Amsterdam in July 2015.

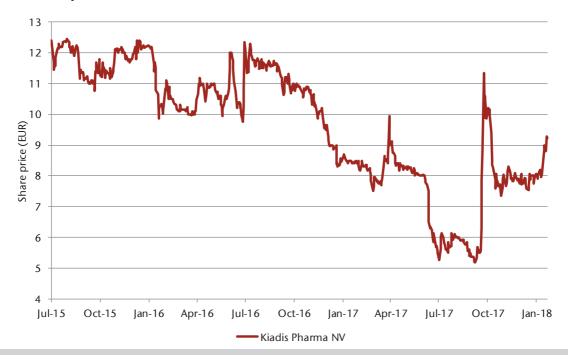
| | Title | Comments |
|----------------------|-----------------|---|
| Arthur Lahr | CEO | Joined Kiadis as COO and CEO designate on 1 January 2017, then transitioning to CEO on 1 April. Previously Chief Strategy Officer at Crucell from 2001 until its acquisition by JNJ in 2011, and before that a consultant at McKinsey & Co and engineer at Unilever. Mr. Lahr holds a Masters degree in Applied Physics from the University of Delft, The Netherlands, and an MBA from INSEAD, France. |
| Robbert van Heekeren | CFO | CFO since 1 May 2008 and a member of the Management Board since its incorporation on 12 June 2015. Prior to this he was Executive Director, Head Global Finance & Control at Organon, where Mr. van Heekeren worked for more than ten years in various positions. Mr. van Heekeren holds a Masters degree in Industrial Engineering & Management Science from Eindhoven University of Technology, The Netherlands. |
| Jan Feijen | COO | COO since joining Kiadis in March 2017 after perviously being Vice President of Manufacturing and Technical Operations, Platform Lead Vaccines and Advanced Therapies at Janssen (part of JNJ). Mr. Feijen has also held various positions at Crucell, Avebe, and Gist Brocades. He holds a Masters degree in Applied Physics from the University of Delft, The Netherlands. |
| Andrew Sandler | СМО | Appointed as Chief Medical Officer in September 2017. Prior to this Dr. Sandler was Senior Vice President, Medical Affairs, at Medivation, as well as Chief Medical Officer at Dendreon and Spectrum Pharma. He has also worked at Bayer Healthcare, Berlex, and Seattle Genetics. Dr. Sandle obtained his MD from Mount Sinai School of Medicine, New York and completed a fellowship in medical oncology at University of California San Francisco. |
| Karl Hard | Head of IR | After spending nearly 20 years at AstraZeneca, Mr. Hard joined Kiadis as Head of IR & Communications in September 2017. At AZN he worked within Investor Relations as well as being a Global Program Director and a Director in Biological Chemistry. He has published over 40 scientific articles in peer-reviewed journals. |
| Margot Hoppe | General Counsel | Appointed as General Counsel & Corporate Secretary in 2008 with over 20 years' experience in corporate legal affairs, including at Gist-Brocades and DSM. Ms. Hoppe has a Masters degree in Law and Political Science from the Erasmus University of Rotterdam, The Netherlands. |

Source: Jefferies research

Initiating Coverage

31 January 2018

Chart 11: Kiadis share price since its IPO



Source: FactSet; Jefferies research

| KDS NA |
|---------------------|
| Initiating Coverage |
| 31 January 2018 |

Company Description

Kiadis develops innovative cell therapies for safer and more effective bone marrow transplants. Its Allodepleted T-cell ImmunotheRapeutics (ATIR) are based on the Theralux platform. Lead programme ATIR101 is filed in Europe for haploidentical haematopoietic stem cell transplants (HSCT) in patients with blood cancers. Kiadis is based in The Netherlands and listed on the Europe than the Europe for haploidentical haematopoietic stem cell transplants (HSCT) in patients with blood cancers. Kiadis is based in The Netherlands and listed on the Europe for haploidentical haematopoietic stem cell transplants.

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(Article 3(1)e and Article 7 of MAR)

Recommendation Published , 22:22 ET. January 30, 2018 Recommendation Distributed , 00:00 ET. January 31, 2018

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| Initiating Coverage |
| 31 January 2018 |

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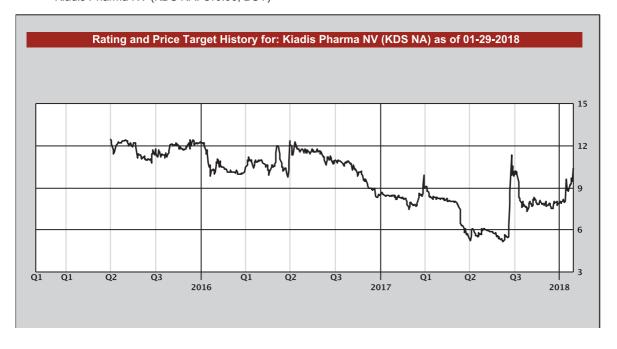
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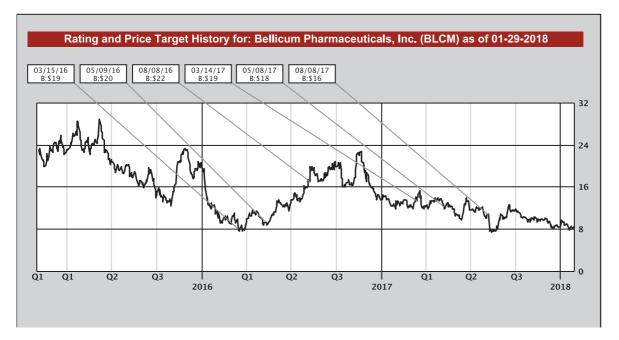
Other Companies Mentioned in This Report

- Bellicum Pharmaceuticals, Inc. (BLCM: \$8.20, BUY)
- Kiadis Pharma NV (KDS NA: €10.96, BUY)



Initiating Coverage

31 January 2018





Notes: Each box in the Rating and Price Target History chart above represents actions over the past three years in which an analyst initiated on a company, made a change to a rating or price target of a company or discontinued coverage of a company.

<u>Legend:</u>

- I: Initiating Coverage
- D: Dropped Coverage
- B: Buy
- H: Hold
- **UP: Underperform**

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KDS NA Initiating Coverage 31 January 2018

Distribution of Ratings

IB Serv./Past 12 Mos. JIL Mkt Serv./Past 12

| | | | | | | WOS. |
|--------------|-------|---------|-------|---------|-------|---------|
| Rating | Count | Percent | Count | Percent | Count | Percent |
| BUY | 1103 | 53.31% | 342 | 31.01% | 66 | 5.98% |
| HOLD | 822 | 39.73% | 161 | 19.59% | 23 | 2.80% |
| UNDERPERFORM | 144 | 6.96% | 20 | 13.89% | 3 | 2.08% |

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31 January 2018

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