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Stock Rating
Overweight

Industry View
In-Line

Initiation

Tesaro Inc.

Low-Risk Lead Asset and Experienced Management; Initiate at Overweight

Lead asset rolapitant for chemotherapy induced nausea/vomiting has a high probability of Phase III success in 2H13 and management's experience in this market is a key positive. We see little in the current valuation for the two other in-licensed cancer drugs.

Rolapitant, an NK-1 inhibitor for prevention of chemotherapy induced nausea and vomiting (CINV), has low clinical trial risk given: 1) clear efficacy in Phase II, 2) a validated class with positive Phase III trials for two other chemically similar agents, and 3) strong correlation between Phase II and III for NK-1 inhibitors. Our $23 bull case assumes rolapitant's longer duration of action leads to evidence of superior nausea control compared to competitor Merck's EMEND, lowering commercial risks and expanding the market.

Management credibility is fundamental to the story in an underdeveloped NK-1 inhibitor market. The $250-$300 MM US NK-1 market is <20% penetrated and we believe the market can double by 2020 from active promotion and rolapitant's differentiated profile with ~50% share to Tesaro in our base case. The stock appears to discount $250 MM in peak rolapitant sales, which is conservative vs. our $325 MM base case. That said, EMEND oral/IV generics in 2015/2019 vs. rolapitant's 2014/2015 launch are a concern but management's past success in CINV provides precedent that branded and generics can coexist.

Two cancer therapeutics assets early but attractive. We include a risk adjusted valuation contribution for niraparib, a PARP inhibitor, given proof of concept from a Phase I trial in a form of ovarian cancer. Near term clarity on the development plan is the next catalyst with the potential for a rapid path to Phase III. We find the second asset TSR-011, an ALK inhibitor for lung cancer, hard to value without greater competitive visibility.

Key Ratios and Statistics

Reuters: TSRO.O Bloomberg: TSRO US
Biotechnology / United States of America

Price target	**$19.00**
Shr price, close (Jul 20, 2012)	$13.50
Mkt cap, curr (mm)	$360
52-Week Range§	$14.18-12.82

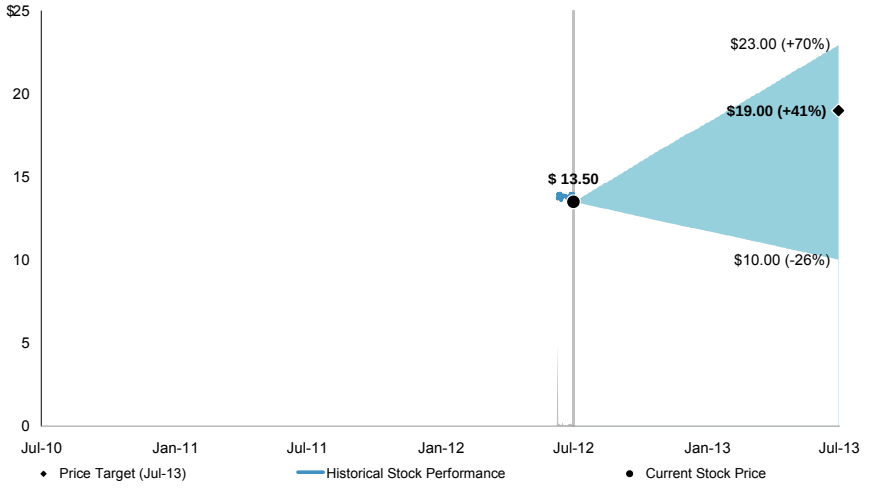
§ = Since IPO on June 28, 2012

Fiscal Year ending	12/11	12/12e	12/13e	12/14e
ModelWare EPS ($)	**(0.32)**	**(2.37)**	**(2.55)**	**(2.29)**
P/E	**NM**	**NM**	**NM**	**NM**
Consensus EPS ($)§	**-**	**-**	**-**	**-**
Div yld (%)	0.0	0.0	0.0	0.0

Unless otherwise noted, all metrics are based on Morgan Stanley ModelWare framework (please see explanation later in this note).
§ = Consensus data is provided by Thomson Reuters Estimates.
e = Morgan Stanley Research estimates

Risk-Reward Snapshot: Tesaro (TSRO, Overweight, $19 Price Target)

Risk-Reward View: Rolapitant the Focus but Early-Stage Pipeline Can Create Value



Source: Thomson Reuters, Morgan Stanley Research estimates

Price Target $19		Based on base case scenario. PT is derived from a DCF analysis, which assumes a discount rate of 17.5% and no terminal value.
Bull Case $23	DCF-based Valuation	**Rolapitant ~$500-600 MM in peak sales, Pipeline Delivers** **CINV Franchise:** Rolapitant achieves differentiated efficacy tripling the NK-1 market **Pipeline**: Same as base case
Base Case $19	DCF-based Valuation	**Rolapitant reaches ~$325 MM in sales, Pipeline a Toss-Up** **CINV Franchise:** Management succeeds in matching the success with Aloxi and converts Rolapitant into a $325MM drug with the NK-1 market doubling in size **Pipeline**: Niraparib succeeds in Phase II (included in our model) or ALK shows a favorable benefit/risk profile in Phase I
Bear Case $10	DCF-based Valuation	**Rolapitant Disappoints, No Pipeline Success** **CINV Franchise:** Rolapitant reaches only $150-200 MM in sales on just ~25% penetration of NK-1 inhibitor market and minimal expansion of the NK-1 market despite significant commercial efforts **Pipeline**: Both niraparib and ALK fail to deliver

Investment Thesis

- Lower-risk lead asset rolapitant has a high probability of meeting Phase III endpoints in 2H13
- NK-1 Inhibitors are a validated drug class for CINV with significant market growth potential
- Management team has deep experience in supportive care
- Cancer therapeutics pipeline is early but interesting

Key Value Drivers & Debates

- Degree of differentiation between Rolapitant and Merck's EMEND is a key question
- ALK program is early with a crowded development landscape
- Clarity on trials plan for niraparib

Key Risks

- Phase II for Rolapitant was only in HEC (Highly Emetogenic Chemotherapy) population
- Generic versions of EMEND and competition from Helsinn/Eisai
- IV Rolapitant is key commercial driver but will not launch until 2015
- NK-1 inhibitor market needs to grow
- Competitive landscape for pipeline

Key Catalysts

Niraparib development plan – 2H12
Eisai/Helsinn's netupitant Phase III in MEC – 2H12
IV rolapitant Phase I initiation – 4Q12
TSR-011 Phase I initiation – 4Q12
TSR-011 Phase I data – 2013 (MSe)
Niraparib Phase II data – 2013 (MSe)
Rolapitant Phase III data – 2H13

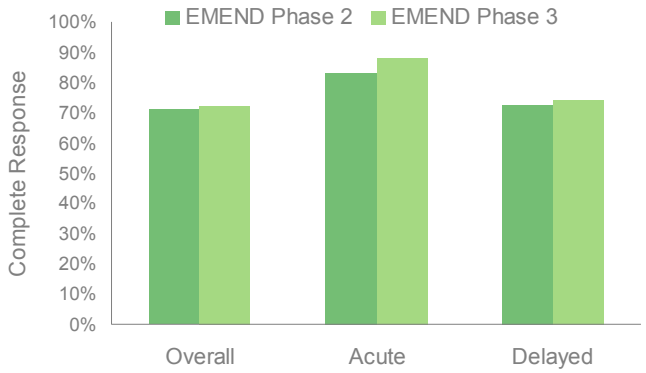
Investment Debates Summary

1. What clinical risk remains for rolapitant?

Market's view: Rolapitant's 2H13 Phase III trial readout carries low clinical risk.

Our view: Phase III looks relatively low risk given 1) clear evidence of activity in Phase II, 2) a validated class, 3) good correlation between Phase II and III historically, and 4) minimal Phase III trial design changes from Phase II. Our base case assumes rolapitant emerges from Phase III looking modestly differentiated from Merck's EMEND, the only NK-1 inhibitor on the market. However, rolapitant's longer half-life (allows >90% occupancy of brain NK-1 receptors for the entire 120 hour trial endpoint period) could lead to superior nausea control (our bull case).

Good Phase II Read-through to Phase III for NK-1 Inhibitors



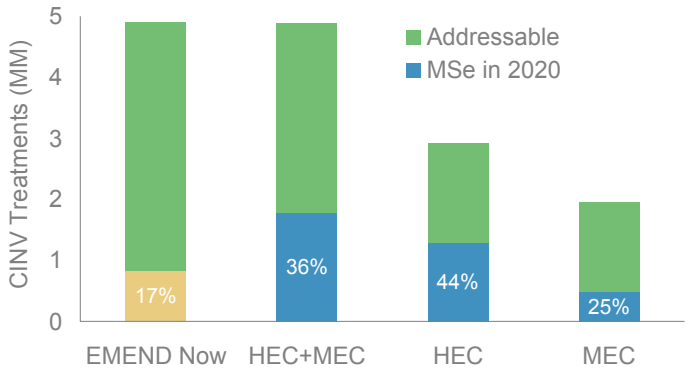
Source: Morgan Stanley Research; EMEND Phase II Data: Cancer 2003;97:2290–300; J Clin Oncol 29:1495-1501.

2. NK-1 inhibitor market expansion?

Market's view: The NK-1 inhibitor market is underpenetrated but growth potential is unclear.

Our view: The NK-1 inhibitor market is only 17% penetrated, and could double by 2020 from expansion of the class, promotion, and wider IV availability. Our base case for rolapitant relies on market growth more than share gains (50% share to rolapitant in our base case). Thus, the track record of Tesaro's management team in oncology supportive care is a key asset for the stock. Evidence of superior efficacy for rolapitant will expand the market and lower generic risk. The stock currently implies ~$250 MM in peak sales.

Room to Grow the NK-1 Inhibitor Market



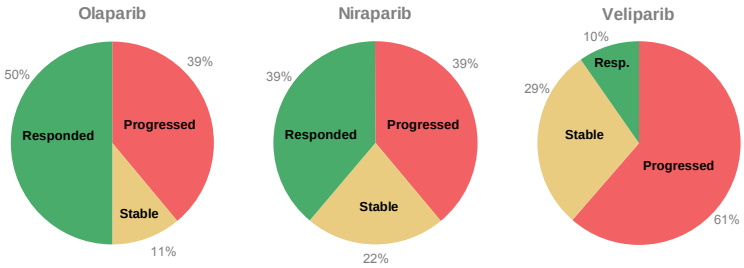
Source: Company Data, Morgan Stanley Research estimates

3. Looking for value beyond rolapitant?

Market's view: Limited visibility on early-stage pipeline assets caps conviction.

Our view: Tesaro in-licensed two oncology assets: 1) TSR-011, an ALK inhibitor from Amgen and 2) niraparib, a Phase I PARP inhibitor from Merck. We include niraparib in our base case after proof of concept in BRCA mutation positive ovarian cancer and a safety profile consistent with the class. Clarity on the development strategy in 2H12 is the next catalyst. Lacking patient data, we need more information on TSR-011 given multiple more advanced competitive agents.

Niraparib Proof-of-Concept in BRCA Mutation Positive Cancers In Line with PARP Inhibitor Class



Source: Morgan Stanley Research; N Engl J Med 361;2; 2012 ASCO Poster 3054; 2011 ASCO Poster 3102.

Investment Case

Initiating on TSRO with an Overweight rating and $19 price target. There are three parts to our thesis: **1)** a lower clinical risk late stage lead asset rolapitant, an NK-1 inhibitor for the nausea and vomiting often associated with chemotherapy with Phase III data in 2H13, **2)** an experienced management team with direct commercial experience in oncology supportive care provides the credibility to build the market for rolapitant despite near term competition from Merck and longer term from generics, and **3)** an early stage therapeutics pipeline with at least one interesting asset in niraparib, a PARP inhibitor, with proof of concept in genetically inherited ovarian cancer.

Exhibit 1
Tesaro Estimates at a Glance

($ in MM, except EPS)	2014E	2015E	2016E	2017E	2018E	2019E	2020E	16-'20 CAGR
Total Revenue	**$20**	**$45**	**$124**	**$271**	**$410**	**$456**	**$504**	*42%*
% growth	***700.4%***	***125.4%***	***175.2%***	***118.0%***	***51.6%***	***11.3%***	***10.5%***	
Rolapitant	$10	$40	$124	$213	$314	$294	$310	*26%*
Niraparib	$0	$0	$0	$57	$96	$162	$194	
COGS	$2	$7	$21	$51	$81	$70	$77	*38%*
R&D	$70	$45	$43	$59	$27	$22	$22	*-15%*
% of Revenue	350%	100%	35%	22%	7%	5%	4%	
SG&A Total	$26	$42	$51	$85	$122	$132	$138	*28%*
% of Revenue	130%	93%	41%	31%	30%	29%	27%	
Operating Margin	**NM**	**NM**	**9.2%**	**34.3%**	**54.9%**	**60.0%**	**62.6%**	
Basic EPS	($2.29)	($1.37)	$0.26	$1.29	$3.04	$3.83	$4.34	*101%*
% growth	NM	NM	NM	389%	135%	26%	13%	
Diluted EPS	($2.29)	($1.37)	$0.26	$1.80	$3.78	$4.21	$4.14	*99%*
% growth	NM	NM	NM	580%	110%	11%	-2%	

Source: Company Data, Morgan Stanley Research estimates

As brief introduction, Tesaro is a development stage oncology company that combines an experienced management team from MGI Pharmaceuticals (acquired by Eisai for $3.9B in cash in 2008) and a pipeline built through in-licensing rather than a technology platform. The lead asset rolapitant is in Phase III for chemotherapy induced nausea and vomiting (CINV) with data expected in 2H13. Rolapitant blocks certain nausea and vomiting signals in the brain. Importantly, the management team has direct experience in this market after successfully launching Aloxi, another agent for chemotherapy induced nausea and vomiting at MGI.

Filling out the pipeline is TSR-011, a preclinical ALK inhibitor from Amgen that will be developed in lung cancer among other areas. The drug will enter human testing in late 2012 with the potential for proof of concept data in 2013 or 2014. Most recently in May 2012, Tesaro added niraparib, a PARP inhibitor, from Merck for breast, ovarian, gastric, and lung cancer. Merck completed a Phase I and we expect Tesaro to start Phase II in late 2012 or early 2013.

Our $19 per share price target implies 41% upside from the current $13.50, which was also the IPO price. Our target is based on DCF with no terminal value using a relatively high 17.5% discount rate, which blends the risks around rolapitant (lower) and niraparib (higher). We assume peak sales for rolapitant of ~$300 MM to $325 MM in our base case, launching in late 2014 and $350 MM for niraparib, launching in 2017. Similar to other biotechnology business models, we believe Tesaro can achieve operating margins of >60% and expect the company to raise additional capital. Our price target implies 18x 2018e EPS ($2.89) and 3.5x 2018e revenue ($410 MM) discounted at 20%.

The "*if this drug is so great why didn't pharma want it*" question. In this era of thin large-pharma pipelines, "asset-light" business models like Tesaro inevitably beg the question of pharma's motivation to license *any* quality assets. While a natural question, we do not view this as a real issue. Tightening R&D budgets across pharma require *portfolio* development decisions where investors have little insight, which may have little or nothing to do with the quality of the asset in question. Second, relative scale differences cannot be overlooked as the risk-adjusted returns on a $250-350 MM drug may look different to Tesaro versus Merck or Amgen. Third, while the seller nearly always knows more than the buyer, development decisions that ultimately prove unwise stretch across biopharma, in our view. That said, we suspect this will remain a conceptual negative about Tesaro for some investors. Moreover, a derivative issue for asset-light models is that execution will be critical to value creation. Tesaro must grow from just ~20-25 employees today to encompass a full commercialization enterprise and the R&D and regulatory capabilities to support Phase III assets after being built on minimal infrastructure.

The lead asset rolapitant has a high probability of success in Phase III in 2H13. Several factors support rolapitant as a relatively low clinical risk asset: 1) clear efficacy in a placebo controlled Phase II trial, 2) NK-1 inhibitors are a validated class with positive Phase III data for two other chemically similar agents, 3) strong historical correlation between Phase II and Phase III for NK-1 inhibitors, 4) a Phase III program that largely recapitulates the positive Phase II trial, and 5) the large size of the Phase III program creates a wide statistical cushion to meet the primary endpoints with statistical significance. Of course, there are still questions with any Phase III program (discussed inside) but we see rolapitant as a relatively low risk asset.

Merck's EMEND is the only NK-1 inhibitor on the market with 2012 annualized sales (based on 1Q12) in the US of $252 MM and $156 MM ex-US. Our base case assumes that Phase III proves rolapitant more or less similar to EMEND with modest differentiation, including fewer interactions with other drugs, a single dose oral format compared to three oral doses for EMEND, and a longer half-life. However, our analysis shows that rolapitant is the only NK-1 inhibitor that maintains >90% blockade of brain NK-1 receptors (carry part of the nausea and vomiting signals caused by chemotherapy) for the entire critical 120 hour period following chemotherapy. This could lead to superior nausea control, the key unmet medical need in our diligence with oncologists, leading to our bull case revenue estimates for rolapitant of >$500 MM.

The NK-1 market has significant growth potential. Our thesis relies on market growth over share gains for rolapitant. The $250-300 MM US NK-1 market is underpenetrated today at just 17% of the total addressable market. Reasons include circumscribed commercial efforts (just ~75 sales people), limited discounting/contracting by Merck, and the relatively recent availability of an IV form of EMEND. Our base case assumes the market nearly doubles (in units) by 2020 with multiple companies promoting the class with total NK-1 inhibitor penetration still at <50%. Thus, our thesis relies on market growth rather than share gains. Our $300-325 MM peak sales base case assumption assumes ~50% share to rolapitant, and at current levels, we estimate the stock implies ~$250 MM peak sales, a relatively conservative commercial outcome.

But EMEND generics a major risk – management experience the key offset. A major risk to the story is that EMEND will go generic in 2015 for the oral form and 2019 for the IV forms which represent 40% and 60% of the market, respectively, but we see IV as 75% of the long term opportunity. Thus, Tesaro will contend with generic competition relatively quickly with oral rolapitant likely launching in late 2014 and IV rolapitant in 2015.

The management team's success at MGI Pharma with transforming Aloxi (~$400 MM in 2011 revenues), also for chemotherapy induced nausea and vomiting, into a market leader despite multiple generic competitors is a key dynamic for the stock and provides important precedent that branded drugs and generics can coexist in CINV. Thus, management credibility and execution is central to the Tesaro story providing critical credibility to build the NK-1 market despite generic risk. That said, our model builds in generic risk in two ways 1) we rely on market growth rather than share gains creating room for rolapitant and generics and 2) we model minimal growth following the availability of oral and IV generics with rolapitant holding its own to 2028 patent expiry. Finally, Helsinn and Eisai are developing a single dose oral combination of another NK-1 inhibitor netupitant with Aloxi (a 5-HT_3 inhibitor) with Phase III data in 2H12. We do not see significant risk as 1) this is oral only while IV represents 60% of the market and growing, 2) another player will help to grow the market, and 3) the product lowers longer-term risk from generic oral EMEND.

Therapeutics pipeline early and niraparib looks the most interesting. Tesaro's two earlier stage pipeline therapeutics assets TSR-011 and niraparib are interesting and have the potential to create value prior to Phase III and launch for rolapitant in late 2013 and 2014, respectively. However, competition remains an overhang for almost all targeted drugs, as any validated target inevitably has several agents in development, often by companies with greater resources. We include risk-adjusted revenue niraparib, but this could be seen as a broader valuation credit for pipeline option value.

Development plans the key near term catalyst for niraparib. Niraparib is a PARP inhibitor, which blocks part of the cell's ability to repair the DNA damage associated with cancer and many chemotherapeutic agents. Niraparib's Phase I trial shows proof of concept in ovarian cancer due to inherited mutations in the BRCA1/2 gene. Niraparib's ~40% response rates in BRCA+ patients and safety profile compare favorably with the class. Moreover, niraparib looks free of the formulation problems that have other hobbled PARP inhibitors.

Tesaro will meet with the FDA soon and should be able to provide clarity on development plans in 2H12. We see the potential for a rapid path to Phase III (potentially as soon as 2013) as monotherapy in women with inherited breast and ovarian cancer in addition to a broader Phase II program. That said, competition is a risk with several other PARP inhibitors in development. BioMarin will report Phase I data for BMN-763 in 2H12 and 1Q13, which has shown interesting early data.

Just too early for TSR-011. We do not include TSR-011 in our model. The pre-clinical data suggest that TRS-011 is an active ALK inhibitor and Pfizer's Xalkori validates the class. However, TSR-011 has yet to enter clinical trials and there are multiple second-generation agents (Ariad, Novartis, Chugai, and others) at least 1-2 years ahead. Thus, we are more comfortable waiting for the competitive landscape to develop (Ariad will report Phase I data in 3Q12 for example) and for some visibility on TSR-011's early safety and efficacy profile. More advanced competitors lend uncertainty to the path forward even with solid data for TSR-011 as the window for a rapid path to market could close.

Valuation

DCF supports $19 per share price target

Discounted cash flow is our primary valuation metric for TSRO. We view this as appropriate given still long timelines to Phase III data for rolapitant (2H13) and commercialization in late 2014 and late 2015 for the oral and IV forms respectively. Our model includes: 1) rolapitant (fundamental views discussed on pages 18 to 25) and 2) niraparib (fundamental views discussed on pages 28 to 31). We model each drug to their respective patent lives (including term extension) or 2030 for rolapitant and 2033 for niraparib. Licensing deal details on page 33.

We do not include any terminal value meaning R&D spend on TSR-011 (~$22 MM annually between 2019 and 2029) is modestly dilutive to our valuation. That said, we assume the rolapitant commercial infrastructure is largely leveragable for niraparib. We include an additional $7-$14 MM in SG&A in connection with the niraparib launch.

Exhibit 2

DCF Supports $19 per share in 12 Months

Discounted Cash Flow ($MM):	
WACC Applied (%)	17.5%
Discounted Net Cash Flow	**$339**
Terminal Value	$0
Firm Value	**$339**
Cash	**$156**
Equity Value	**$495**
Shares Outstanding (MM)	26.7
Equity Value per Share ($)	**$19**

Source: Company Data, Morgan Stanley Research estimates

We use a relatively high discount rate of 17.5% to reflect: 1) the commercial risks facing rolapitant including generic EMEND, 2) Phase III risks for the rolapitant, and 3) the earlier stage of the therapeutics pipeline including niraparib, which has only completed Phase I so significant risks remain.

Our model assumes Tesaro raises additional capital including $100 MM in 2013 following positive rolapitant data and $50 MM in 2015 to support niraparib.

Multiple based valuation is supportive

We tend to de-emphasize multiple based valuations on still distant revenue and/or earnings for stories like Tesaro. That said, they can still provide meaningful context and check on our valuation. For Tesaro, we model profitability in 2016 and the company is cash flow positive in 2017.

For EPS, we looked at 2018-2020, which captures the principal years of the rolapitant and potentially niraparib commercial launch. Our $19 per share valuation implies 18x/17x/18x on 2018/2019/2020e EPS discounted back to one year from now at 20%. The rationale for the high discount rate is similar to our DCF.

Exhibit 3

PE Multiple Based Valuation Supports Our Price Target

	Fully Taxed 2018 EPS	$2.89	
	WACC Applied		
P/E Multiple	18%	20%	22%
14x	$16	$15	$14
16x	$19	$17	$15
18x	$21	$19	$17
20x	$23	$21	$19
22x	$26	$23	$21

	Fully Taxed 2019 EPS	$3.65	
	WACC Applied		
P/E Multiple	18%	20%	22%
13x	$16	$15	$13
15x	$19	$17	$15
17x	$21	$19	$17
19x	$24	$21	$19
21x	$26	$23	$21

	Fully Taxed 2020 EPS	$4.14	
	WACC Applied		
P/E Multiple	18%	20%	22%
14x	$17	$15	$13
16x	$19	$17	$15
18x	$22	$19	$17
20x	$24	$21	$19
22x	$26	$23	$20

Source: Company Data, Morgan Stanley Research estimates

Looking at revenue multiples, our $19 per share target implies ~3.5-4x on 2018/2019/2020e revenue discounted back to one year from now at 20%.

Exhibit 4
Revenue Multiples Supportive of Our Price Target

P/E Multiple	2018 Revenues (MM) $410 — WACC Applied		
	18%	20%	22%
1.5x	$9	$8	$8
2.5x	$15	$14	$13
3.5x	$22	$20	$18
4.5x	$28	$25	$23
5.5x	$34	$31	$28

P/E Multiple	2019 Revenues (MM) $456 — WACC Applied		
	18%	20%	22%
1.5x	$9	$8	$7
2.5x	$15	$13	$12
3.5x	$20	$18	$16
4.5x	$26	$24	$21
5.5x	$32	$29	$26

P/E Multiple	2020 Revenues (MM) $504 — WACC Applied		
	18%	20%	22%
2.0x	$11	$10	$9
3.0x	$16	$14	$13
4.0x	$22	$19	$17
5.0x	$27	$24	$21
6.0x	$33	$29	$26

Source: Company Data, Morgan Stanley Research estimates

Stand-alone rolapitant valuation

Finally, given that rolapitant is central to the story, we also built a stand-alone asset valuation. We assume launch in 2014 reaching peak sales in 2019 with a run-rate of ~$330 MM. We include 100 sales people in the key launch years (2016-2020) at a cost of $25-30 MM and a marketing budget in the $40-50 MM range. R&D is significant over the next few years (cumulatively ~$240 MM between 2012 and 2015) but tapers off to $22 MM per year by 2019. We assume milestones of $5 MM and $15 MM to Opko upon filing and approval, respectively, with an on-going royalty of ~12% for realized gross margins in the mid-eighties.

In our stand-alone valuation for rolapitant we explore fully de-risked discount rate scenarios relative to our firm-wide blended discount rate of 17.5% that incorporates higher pipeline development risk for niraparib. Under a substantially de-risked 12-14% discount rate, we see value in the $15 to $19 per share range based on the rolapitant asset alone.

Our stand-alone valuation implies that at the current stock price of ~$13.50, and using a fully-de-risked discount rate of 10% typical of commercial drugs in our coverage universe, the current market valuation implies peak sales of only ~$250 MM, roughly 25% *below* our base case of ~$325 MM. In other words, one need only believe in a relatively narrow commercial opportunity for rolapitant to justify the current market valuation. The above reasoning assumes conservatively that all current share value is attributable to rolapitant (i.e., treating cash per share at zero).

Key Risks to Our Price Target

There are several risks to our valuation: 1) Generic versions of oral EMEND are expected in 2015 and oral rolapitant is launching just 1 year earlier, 2) IV rolapitant launch in 2015 is a key commercial driver for supportive care program and IV EMEND goes generic in 2019, 3) adoption of rolapitant among community physicians may be challenging since use of NK-1 inhibitors in this setting is more limited currently, 4) intense competitive landscape for cancer therapeutics assets with several programs more advanced in development by companies with greater resources, 5) Tesaro will likely require additional capital prior to reaching sustainable profitability.

Exhibit 5

Rolapitant Stand-Alone DCF Valuation

	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2028E	2029E
Rolapitant Revenues	**$0**	**$0**	**$10**	**$40**	**$124**	**$213**	**$314**	**$294**	**$310**	**$336**	**$48**
COGS	$0	$0	$2	$7	$21	$40	$64	$44	$46	$45	$7
Gross Profit	($52)	($44)	($47)	($27)	$34	$96	$175	$179	$196	$259	$19
Rolapitant SG&A	$6	$11	$36	$46	$57	$66	$66	$63	$63	$30	$21
Rolpatitant R&D	$46	$35	$30	$20	$12	$12	$10	$8	$5	$2	$2
EBIT	($52)	($44)	($47)	($27)	$34	$96	$175	$179	$196	$259	$19
Interest Income	$1	$1	$1	$0	$0	$0	$1	$2	$3	$13	$14
Taxes	$0	$0	$0	$0	$0	$5	$26	$45	$69	$95	$11
Rolapitant Cash Flows	**($51)**	**($43)**	**($47)**	**($27)**	**$35**	**$91**	**$149**	**$136**	**$129**	**$176**	**$21**
PV of Cash Flows in 12 Months	**($56)**	**($40)**	**($37)**	**($18)**	**$20**	**$44**	**$61**	**$48**	**$38**	**$14**	**$1**

DCF Details	
Discount Rate	17.5%
Discounted Cash Flow (2012 - 2029)	**$267**
Terminal Value	$0
Rolapitant DCF	**$267**
Shares Outstanding (MM)	26.7
Per Share Value	**$10**

DCF Sensitivity						
Discount Rate						
10%	12%	14%	16%	18%	20%	22%
$23	$19	$15	$12	$10	$8	$6

Source: Company Data, Morgan Stanley Research estimates

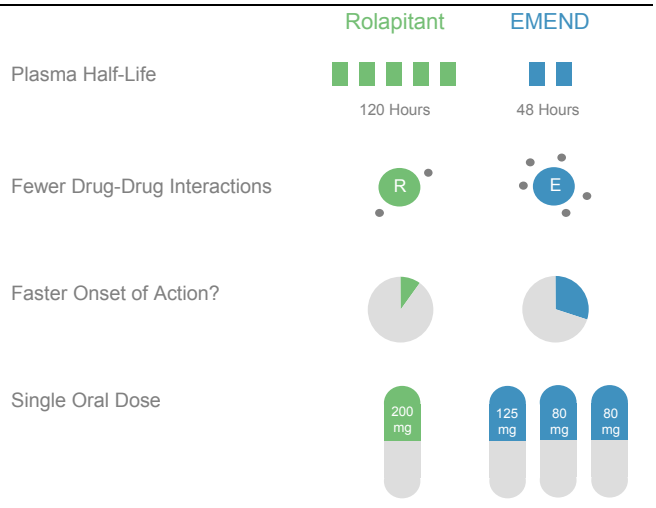
Debate #1: What Clinical Risk Remains for Rolapitant?

Market's view: Rolapitant's 2H13 Phase III trial readout carries low clinical risk.

Our view: Phase III looks relatively low risk given 1) clear evidence of activity in Phase II, 2) a validated class, 3) good correlation between Phase II and III historically, and 4) minimal Phase III trial design changes from Phase II. Our base case assumes rolapitant emerges from Phase III looking modestly differentiated from Merck's EMEND, the only NK-1 inhibitor on the market. However, rolapitant's longer half-life (allows >90% occupancy of brain NK-1 receptors for the entire 120 hour trial evaluation period) could lead to superior nausea control (our bull case).

Rolapitant's Phase III data in chemotherapy induced nausea and vomiting in 1H13 is the first major catalyst for the stock. In addition, the data will provide insight into the extent of clinical differentiation from Merck's EMEND, the major competitor. We see the trial as low risk to reach the primary endpoint of overall response rate (defined as no vomiting and no nausea) over placebo in the delayed phase with statistical significance. However, we have lower conviction that the Phase III data will support definitive clinical or efficacy differentiation from EMEND on nausea control alone, the major unmet medical need in management of CIINV. Thus, we include this outcome in our bull case valuation.

Exhibit 6
Rolapitant Points of Differentiation from EMEND



Source: Company Data, Morgan Stanley Research

Taking a broad perspective, we see four potential areas of clinical differentiation that may be borne out in the pivotal clinical trial program, including 1) the potential of rolapitant's longer plasma half life to improve nausea control, 2) a faster onset of action, which could provide better acute phase control, 3) fewer drug-drug interactions, and 4) a single oral dose compared to three oral doses or a single IV dose for EMEND. The first two are higher risk while the others are lower risk.

Background on CINV & NK-1 Inhibitors

There are two broad phases of CINV, acute and delayed. The acute phase occurs within the first 24 hours following chemotherapy whereas the delayed phase is defined as the second through fifth days post-chemotherapy or the 24-120 hour period. Without treatment, the incidence of CINV ranges from 20-90%. Current drugs for CINV provide good control of vomiting, however nausea still remains a challenge, especially in the delayed phase.

Responses for CINV therapies are gauged based on efficacy in controlling emesis (vomiting) and nausea, assessed independently in the acute and delayed phases as well as overall across 5-days post-chemotherapy. A frequently employed endpoint for CINV clinical trials is the complete response rate which reflects the percentage of patients with no emesis *and* no use of rescue medications, again measured in the acute, delayed and overall periods. A refinement on these definitions is the endpoint of no nausea and no *significant* nausea, which reflects an absence of nausea that interferes with normal daily activities. This measurement is ubiquitous in CINV trials and is a more meaningful assessment of nausea reduction, as completely eliminating even mild nausea not interfering with normal duties is a very high bar.

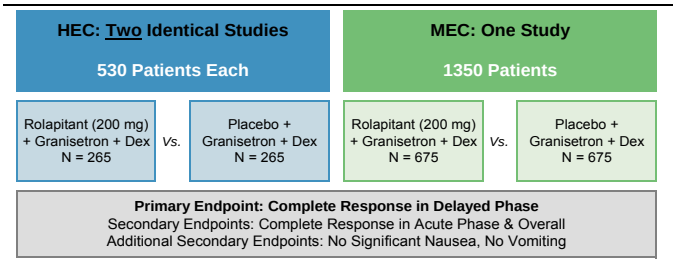
CINV is managed with a combination of two drugs called 5-HT_3 antagonists and a steroid called dexamethasone. NK-1 inhibitors like EMEND and rolapitant are added in certain cases where chemotherapies with high nausea and vomiting potential are employed and extra-protection is required. For this reason, CINV drug are generally tested in two populations, HEC or highly emetogenic chemotherapy and MEC, moderately emetogenic chemotherapy. The mechanism of NK-1 inhibitors relies on blocking the neurokinin-1 receptor in the brain, which functions to bind a neuropeptide (substance P) responsible for stimulating the vomiting center and nausea. Interestingly, substance P has been implicated in acute and *delayed* phase emesis, suggesting that NK-1 receptor inhibition could provide longer term protection than the 5-HT_3 receptor antagonists, another class of the CINV agents. Underlying this view is that while blocking serontonin is still necessary to prevent emesis in

the acute phase, delayed phase emesis has traditionally responded weakly to 5-HT_3 receptor inhibition alone.

Rolapitant is a low risk Phase III asset

The rolapitant Phase III program encompasses three trials 1) two identical trials in highly emetogenic chemotherapy (HEC) totaling 1,060 patients and 2) a study in moderately emetogenic chemotherapy (MEC) with 1,350 patients. The trials compare rolapitant at 200 mg combined with a 5-HT_3 inhibitor granesitron and dexamethasone to granesitron alone. The primary endpoint for all three trials is complete response in the delayed phase.

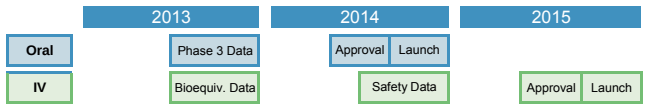
Exhibit 7
Rolapitant Phase III Program in a Nutshell



Source: Company Data, Morgan Stanley Research

Phase III timelines. The first Phase III studies are expected to read out in the second half of 2013. The IV formulation is approximately one-year behind with data at the end of 2014.

Exhibit 8
Rolapitant Development Timelines

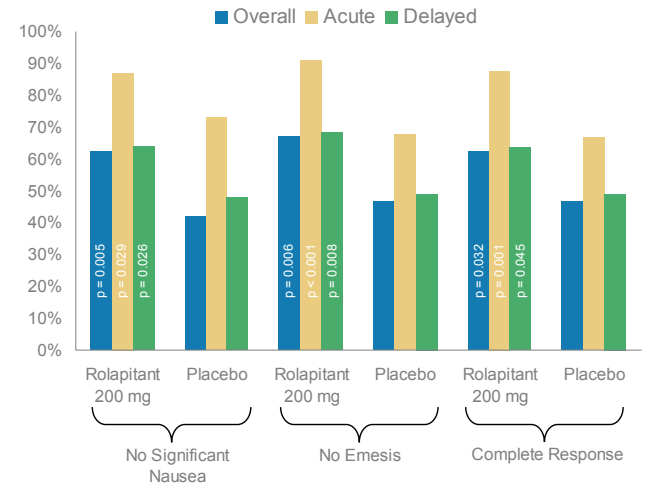


Source: Company Data, Morgan Stanley Research

There are six primary reasons that we see low clinical risk to Phase III meeting the primary endpoint: 1) rolapitant has shown a statistically significant benefit in a randomized placebo controlled Phase II trial, **2)** the NK-1 inhibitor class is validated by multiple agents showing efficacy in CINV providing important context for rolapitant, **3)** rolapitant efficacy compares well with other NK-1 inhibitors, **4)** the Phase III design largely recapitulates the positive rolapitant Phase II trial, **5)** the trial's size and powering provides significant efficacy cushion while still reaching statistical significance, and **6)** Phase II and III generally correlate for the other NK-1 inhibitors.

#1 Rolapitant has already shown clear efficacy in a placebo controlled Phase II trial. Key to our thesis of low clinical risk is that rolapitant, before it was in-licensed from Merck/Opko, completed a 454 patient Phase II dose ranging trial in HEC. The trial showed clear efficacy at the highest dose of 200 mg across all primary and secondary endpoints (see Exhibit 9). At the 200 mg Phase II dose, rolapitant showed a statistically significant benefit in the complete response rate for the overall, acute, and delayed phases of 63% vs 47%, 88% vs 67%, and 64% versus 49%, respectively.

Exhibit 9
Rolapitant Phase II Data Show Clear Efficacy



Source: Company Data, Morgan Stanley Research

Moreover, the safety profile looks solid in Phase II and we see low risk of a new safety issue emerging in Phase III. The Phase II safety data showed little that was concerning (Exhibit 10) further lowering Phase III risk.

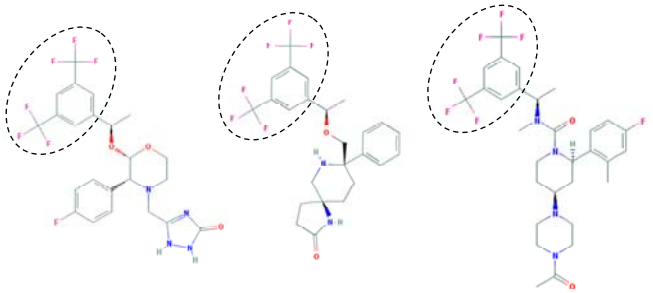
Exhibit 10
Summary of Rolapitant Phase II Safety Data

	Placebo		Rolapitant 200 mg	
	N	%	N	%
Any AE	**8**	**9%**	**12**	**13%**
Constipation	0	0%	1	1%
Headache	2	2%	0	0%
Fatigue	2	2%	4	4%
Dizziness	0	0%	1	1%
Distrubance in attention	2	2%	1	1%
Nausea	0	0%	1	1%
Anorexia	0	0%	2	2%
Vomiting	1	1%	0	0%
Hiccups	1	1%	1	1%
Adbominal Pain	0	0%	0	0%
Asthenia	0	0%	1	1%
Balance Disorder	0	0%	2	2%
Somnolence	0	0%	2	2%

Source: Company Data, Morgan Stanley Research

#2 and #3 NK-1 inhibitors are a validated class. Multiple NK-1 inhibitors have shown clear efficacy, including Merck's EMEND and GSK's casopitant with consistent efficacy across the response rate and no emesis endpoints. Therefore, rolapitant's efficacy in Phase II is supported by the broader landscape of efficacy across the NK-1 inhibitor class.

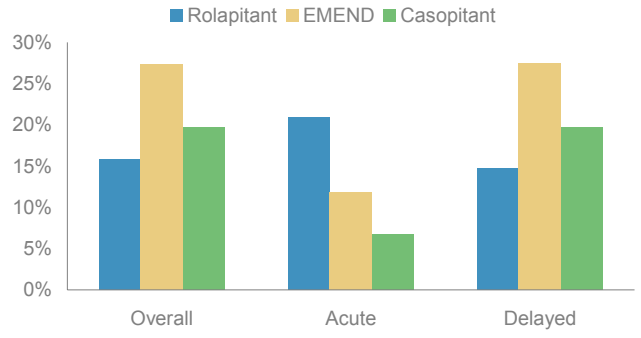
Exhibit 11
NK-1 Inhibitors Share Common Structures



Source: Morgan Stanley Research. PubChem.

This point is supported by an examination of the chemical structures of rolapitant, aprepitant and casopitant, which share a common scaffold (Exhibit 11, the trifluoromethylphenyl (TFMP) group) that is believed to enhance activity in vivo and improve metabolism. Additional common features on all NK-1 inhibitors drive affinity for NK-1 receptors, including a second aromatic ring in lower portion of the molecule, a centrally located nitrogen atom, and a methyl side chain adjoining the TFMP group.

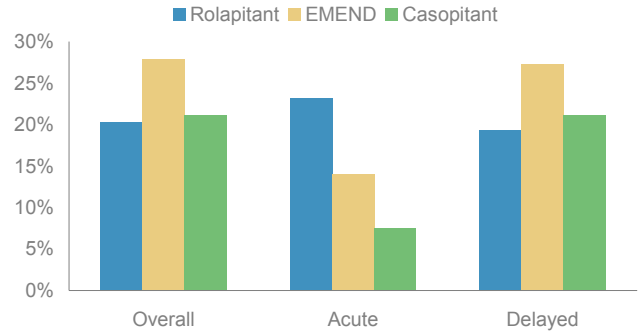
Exhibit 12
Complete Response Rates (placebo-adjusted) in HEC for NK-1 Inhibitors Comparable



Source: Company Data, Morgan Stanley Research. EMEND Phase II Data: Cancer 2003;97:2290–300; Casipotant Phase III Data: Lancet Oncol 2009; 10: 549–58.

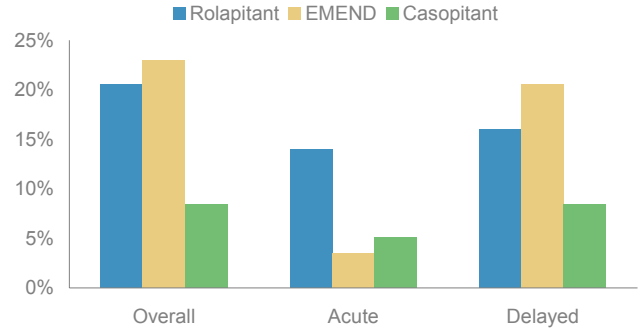
Cross trial comparisons are always fraught with risk. However, comparing rolapitant Phase II efficacy with Phase II trials for other NK1 inhibitors suggests that efficacy compares well across both emesis (vomiting) and nausea response rate endpoints (see Exhibit 13 and Exhibit 14).

Exhibit 13
No Emesis Response Rates (placebo adjusted) also Similar Across NK-1 Inhibitors in HEC



Source: Company Data, Morgan Stanley Research. EMEND Phase II Data: Cancer 2003;97:2290–300; Casipotant Phase III Data: Lancet Oncol 2009; 10: 549–58.

Exhibit 14
No Significant Nausea Response Rates (placebo adjusted) are More Variable Across NK-1 Inhibitors



Source: Company Data, Morgan Stanley Research. EMEND Phase II Data: Cancer 2003;97:2290–300; Casipotant Phase III Data: Lancet Oncol 2009; 10: 549–58.

#4 Rolapitant's Phase III design largely recapitulates the positive Phase II trial. Rolapitant's Phase III trial design is very similar to the positive Phase II trial discussed above, which lowers risk to the Phase III trial (Exhibit 15).

Exhibit 15

Rolapitant Phase III Design Recapitulates Phase II

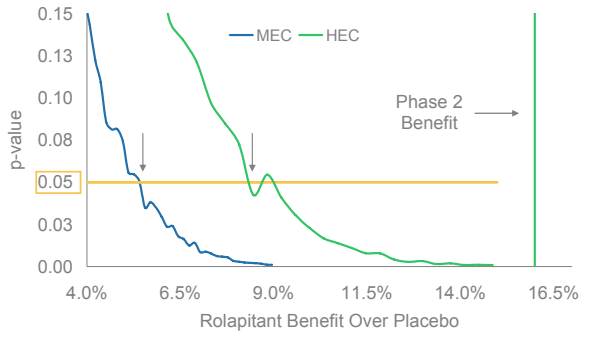
	Phase 2	Phase 3
Chemotherapy Class	**HEC**	**HEC & MEC**
5HT3 Inhibitor	**Ondansetron**	**Granisetron**
Total Subjects	450	1880
Rolapitant Doses	10, 25, 100, 200 mg	200 mg
Comparator	Placebo-Controlled	Placebo-Controlled
Masking	Double-Blind	Double-Blind
Allocation	1:1:1:1:1 Randomized	1:1 Randomized
Primary Endpoint	**Overall Complete Response (0-120 Hours)**	**Delayed Phase Complete Response (>24-120 Hours)**
Secondary Endpoint	Acute Phase Complete Response (0 - 24 Hours)	Acute Phase Complete Response (0 - 24 Hours)
Secondary Endpoint	Delayed Phase Complete Response (>24-120 Hours)	Overall Complete Response (0-120 Hours)
Secondary Endpoint	No Significant Nausea	No Significant Nausea
Secondary Endpoint	No Emesis	No Emesis

Source: Company Data, Morgan Stanley Research

#5 A low bar for statistical success in Phase III. Efficacy often erodes in the transition from Phase II to Phase III clinical trials. Therefore, we simulated the rolapitant Phase III trial in HEC and MEC to determine the statistical hurdle to meet the trial's primary endpoint with a p-value less than 0.05. Our analysis shows that the HEC Phase III trial will likely meet statistical significance at a difference over placebo as low as 8% to 9% on the primary endpoint. This is well below the ~16% benefit in the Phase II trial for rolapitant. Moreover, the HEC trials are designed with 90% power to detect a 15% benefit over placebo assuming a 50% response rate for placebo. Similarly, in MEC, we estimate the Phase III is powered at 90% to detect a smaller 9% benefit assuming a 50% response rate in the placebo arm. Therefore, we see a low risk to the Phase III trial reaching statistical significance on the primary endpoint.

Exhibit 16

A Low Statistical Bar for Success in Phase III

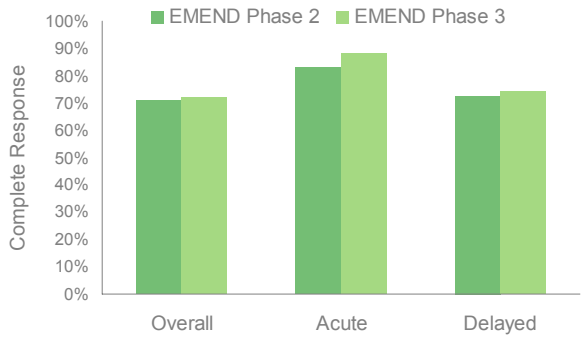


Source: Company Data, Morgan Stanley Research, XLSTAT

Importantly, our analysis shows that for other NK-1 inhibitors including Merck's EMEND that Phase II efficacy is largely preserved in Phase III for CINV (Exhibit 17). The same is also true in the MEC setting when examining data for GSK's casopitant (Exhibit 18).

Exhibit 17

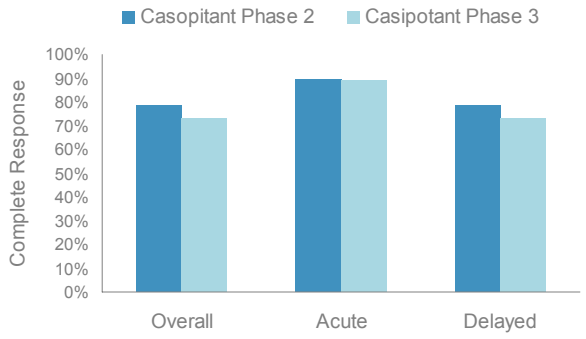
EMEND Phase II & Phase III Complete Responses (HEC) Highly Consistent



Source: Morgan Stanley Research. EMEND Phase II Data: Cancer 2003;97:2290–300; J Clin Oncol 29:1495-1501.

Exhibit 18

Casopitant Phase II & Phase III Complete Responses (MEC) also Consistent



Source: Morgan Stanley Research; J Clin Oncol 27:5363-5369; Cancer 2009;115:5807–16.

What about subsequent cycles of chemotherapy? Efficacy measures in CINV trials are based on the first cycle of chemotherapy. However, regulatory approval requires consistent performance in subsequent cycles. In Phase II, rolapitant showed consistent efficacy across cycles as did EMEND and casopitant in Phase III.

Will rolapitant show differentiated efficacy in Phase III?

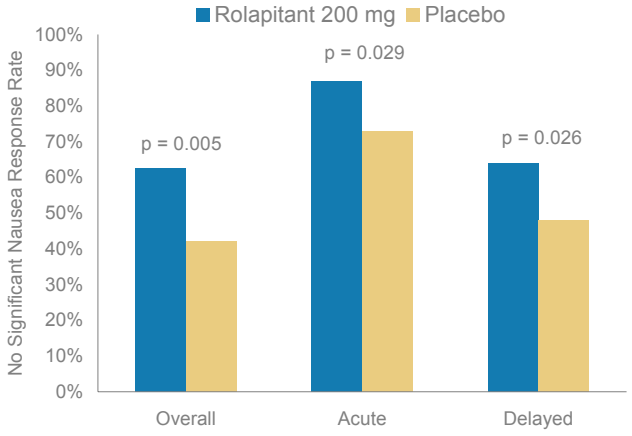
Differentiation on nausea would lead to significant value creation. Our discussion above focuses on low risk to rolapitant's Phase III data proving roughly similar to EMEND,

Merck's marketed NK-1 inhibitor. However, we see the potential for rolapitant to be differentiated on two fronts: 1) suppression of nausea, and 2) onset of action (e.g. working more rapidly). Our diligence with oncologists and oncology nurses consistently supports improved nausea control particular in the delayed phase (2-5 days following chemotherapy) as the major unmet medical need in CINV.

Intriguingly, rolapitant showed a statistically significant benefit on "no significant nausea" in Phase II … at the 200 mg Phase III dose with 63% for rolapitant compared to 42% for placebo for the overall timeframe (0 - 120 hours) (Exhibit 19).

Exhibit 19

Rolapitant Statistically Significant "No Significant Nausea" Benefit in Phase II



Source: Company Data, Morgan Stanley Research

… while EMEND failed to reach statistical significance on "no significant nausea" in Phase III creating an opportunity for rolapitant. Interestingly, in the US/EU Phase III trial for HEC, EMEND failed to show a statistically significant benefit on both the "no significant nausea" and no nausea endpoints at 73.2% vs 66.0% and 47.5% vs 44.2%, respectively, for EMEND and placebo for the overall timeframe.

In a parallel Phase III Latin America study, EMEND achieved significance for no nausea overall (49% vs 39%, $p < 0.05$), but on the more clinically relevant "no significant nausea" overall endpoint fell short of significance (71% vs 64%). For the delayed phase, both geographies failed to meet statistical significance for "no significant nausea", with the US/EU study yielding 75.3% vs 68.5% and the Latin America study yielding 73% vs 65% for EMEND and placebo, respectively.

These data also highlight the importance of a stable geographic mix across clinical trials for NK-1 inhibitors and Tesaro is making strong efforts to maintain comparable populations between Phase II and Phase III.

Rolapitant's longer half-life provides a potential mechanism for better nausea control. Rolapitant's half-life (a measure of how long a drug lasts in the blood) is 120 hours versus 48 hours for EMEND's single dose IV. Moreover, examination of EMEND's pharmacokinetics highlights the potential for inadequate coverage particularly in the critical delayed phase of 24 to 120 hours following chemotherapy when EMEND reaches very low levels leaving a window for suboptimal efficacy and an opportunity for rolapitant.

EMEND provides suboptimal NK-1 receptor occupancy during the 120 hour CINV evaluation period … Importantly, data for EMEND show that a blood level of 100 ng/mL is required for 90% occupancy for brain NK-1 receptors. However, we estimate that by roughly 48-72 hours the EMEND blood levels are below this threshold leaving suboptimal coverage for nearly 40% of the delayed phase potential limiting efficacy. That said, the likelihood of nausea events also declines with time so the effective coverage is still unclear.

Exhibit 20

Rolapitant Superior NK-1 Receptor Occupancy

Time (Hours)	NK-1 Receptor Occupancy		
	EMEND	Casopitant	Rolapitant
48	> 90%	> 90%	NA
72	~80%	~85%	NA
84	~75%	~75%	NA
120	~40%	< 10 %	> 90%

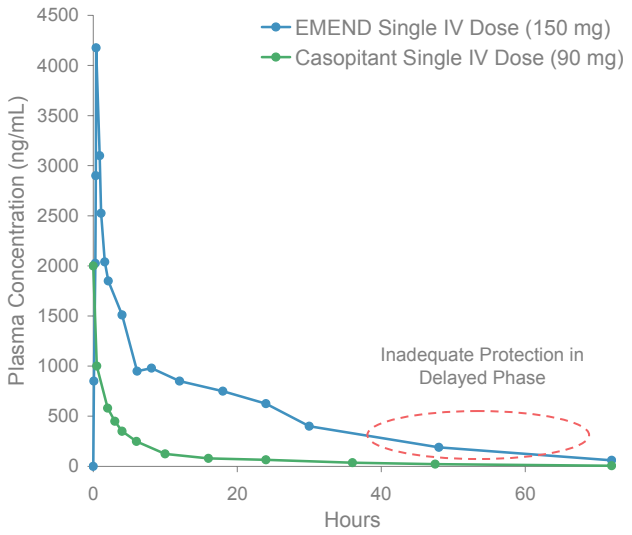
Source: Company Data, Morgan Stanley Research; Biol Psychiatry 2004;55:1007–1012; Eur J Nucl Med Mol Imaging (2012) 39:226–235; DMD 37:1635–1645, 2009.

… while rolapitant provides 100% coverage. By contrast, rolapitant requires blood levels of ~350 ng/mL to maintain 90% NK-1 receptor occupancy. The PK data are more encouraging than for EMEND with blood levels above this threshold for 100% of the 120 hour evaluation period, which provides a clear rationale for better delayed phase efficacy.

Casopitant's PK profile provides further evidence that previously developed NK-1 inhibitors appear to lack adequate delayed phase protection out to 120 hours. The PK curve for the IV single dose for casopitant (90 mg) falls more precipitously than EMEND's single dose IV (150 mg) PK profile (Exhibit 21) and our analysis of NK-1 receptor occupancy suggests occupancies <10% for casopitant by 120 hours (Exhibit 20).

Despite this, we only assume rolapitant shows a statistically and clinically significant, differentiated nausea benefit in our bull case for several reasons:

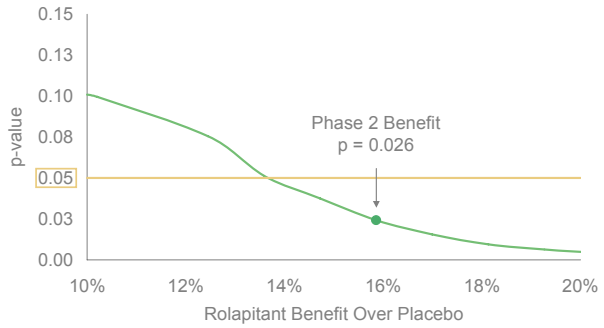
Exhibit 21
IV EMEND & Casopitant PK Profile Suggests Inadequate Delayed Phase Protection



Source: Morgan Stanley Research; Clinical Pharmacology in Drug Development July 2012 1: 93-101; DMD 37:1635–1645, 2009.

First, the Phase II "no significant nausea" benefit was statistically significant but far from robust. The p-value for the delayed phase nausea response rate was 0.026 for a 16% response rate benefit. Our sensitivity analysis (Exhibit 22) suggests that the impact would have been lost at a 13%-14% difference equivalent to only 2-3 fewer patients in the rolapitant arm with no significant nausea given the small size of the study. Therefore, given the number of secondary endpoints examined in Phase II, we still see a risk that the result was random.

Exhibit 22
Statistical Sensitivity of Delayed Phase Nausea Response Rate in Phase II



Source: Company Data, Morgan Stanley Research, XLSTAT

Second, the history of EMEND suggests caution is warranted. Merck's EMEND showed a statistically significant nausea benefit in Phase II for HEC but failed to do so in Phase III for HEC, which provides a cautionary note for rolapitant. Specifically in Phase II, for the overall timeframe (0-120 hours), EMEND showed a response of 81.7% vs 58.7% for placebo ($p < 0.01$) and 52.7% vs 34.1% for placebo ($p < 0.01$) for the "no significant nausea" and no nausea endpoints, respectively. On the other hand, in Phase III the difference was not significant at 73.2% vs 66.0% for no significant nausea (p value > 0.05) and 47.5% vs 44.2% for no nausea (p value > 0.05) for EMEND vs placebo, respectively, assessed in the overall timeframe. Additionally, when examining the delayed phase specifically where nausea control is most challenging, EMEND showed significance for nausea endpoints in Phase II but not in Phase III.

Third, the physiological mechanism of nausea is poorly understood. Our scientific analysis provides a rationale for better efficacy for rolapitant. However, this is only half the battle as our diligence supports a major psychological component to nausea particularly in the delayed phase. Of course, we have no reason to think that rolapitant would differentially impact the psychological aspects of nausea, the potential anti-depressive effects of NK-1 inhibitors aside.

Fourth, the bar for a nausea benefit in both HEC *and* MEC is high meaning the *magnitude* of the nausea benefit matters. Our analysis shows a reasonable probability that rolapitant shows a statistically significant nausea benefit in HEC. However, the bar in MEC is higher given a lower overall CINV intensity and we see a relatively lower probability of success in MEC. Differentiated efficacy in only one setting could be sufficient to drive the perception amongst providers that rolapitant is a superior drug. However, this dynamic

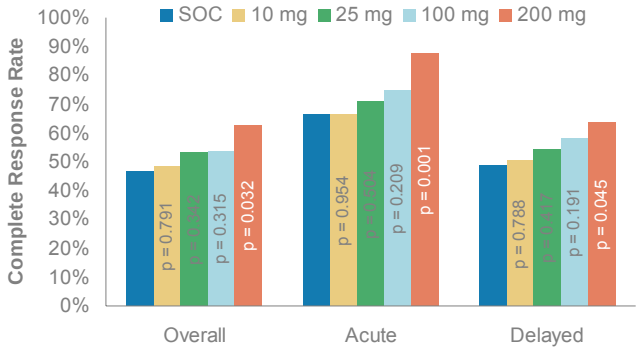
between HEC and MEC means that the *magnitude* of the nausea benefit will matter and we lack the conviction on this key point.

Our physician conversations suggest that a difference of ~15% would be considered clinically meaningful. The Phase II difference was 16% leaving little room for erosion in Phase III. Moreover, EMEND managed only a 3%/7% difference in Phase III on no nausea/no significant nausea while GSK's never-launched casopitant showed a difference of 8-10% that was statistically significant but of unclear clinical significance.

OK, so that's the bull case, there are still risks to Phase III

We view the overall risk in Phase III as low, but there are still risks. First, **we note that that the clinical dose response in the Phase II trial was non-linear** (Exhibit 23) with a convincing effect only at the highest 200 mg dose. Therefore, this pattern creates some question around the true efficacy and dose selection.

Exhibit 23
Rolapitant Phase II Dose Response Inconsistent



Source: Company Data, Morgan Stanley Research

This is a fair criticism. However, we are comfortable for several reasons: 1) the 200 mg dose alone already provides consistent >90% brain NK-1 receptor occupancy at 120 hours and it is thus questionable whether higher doses would further enhance clinical outcomes, 2) the Phase III trials in HEC and MEC are highly powered (90%) so the risk of not further exploring the dose-response curve in Phase II is mitigated, and 3) the broader NK-1 inhibitor class provides validation.

Second, given a placebo controlled trial and an already marketed competitor, cross trial comparisons of efficacy are inevitable as investors try to put the data in better context. Our base case is that the efficacy of rolapitant and EMEND is comparable. For example, in Phase II, the placebo adjusted "no significant nausea" response rate overall in HEC was 21% for rolapitant versus 23% for EMEND, while the placebo adjusted complete response rate overall in HEC was 16% vs 27% admittedly favoring EMEND, but still above the clinically relevant bar (~15%) for both agents. However, as second to market, rolapitant carries risk of enrolling a subtly different patient population. We see risk that physicians may hesitate to enroll patients at the highest risk for severe CINV in a placebo controlled trial *when EMEND is commercially available*. Combined with the natural improvements in overall supportive patient CINV case, a trial composed of lower risk patients could compress the benefit seen for rolapitant and/or increase the placebo response rate.

EMEND's Phase III program in HEC enrolled prior to 2003 and achieved a ~20% placebo adjusted increase in the overall complete response rate and a 52% placebo response rate to a 5-HT_3 inhibitor and dexamethasone alone. GSK's casopitant Phase III trial enrolled between November 2006 and October 2007 (EMEND was approved in 2003) and also showed a ~20% placebo-adjusted overall complete response rate benefit albeit with a higher 66% placebo response rate for ondansetron and dexamethasone. Rolapitant is enrolling 5-6 years later, which increases risk.

That said, we do not view this as a major issue for several reasons. Any cross trial comparisons may impact investor sentiment but are unlikely to be a fundamental driver of physicians' views of the drug. Second, our conversations suggest that a 15% benefit on the primary endpoint is clinically significant, which is an achievable fundamental bar for success. Third, rolapitant's longer half-life could provide additional efficacy to offset any patient population creep. Fourth, we expect much of the trial to enroll in non-US/EU5 geographies were EMEND availability is limited.

Third, there are minor differences in the trial design between Phase II and Phase III. Overall, the Phase III design recapitulates Phase II but with a few differences that deserve mention (Exhibit 24). **1)** The 5-HT_3 agent used in Phase III is granisetron but ondansetron was used in Phase II. Our diligence consistently supported no material difference between the two agents in clinical trials or from the perspective of oncologists and oncology nurses. In reality, this change is a positive given the recent FDA warning and label change for ondansetron. **2)** The primary endpoint is changed from overall response rate to the delayed phase complete response rate. We understand this was at the FDA's request and importantly, the Phase II trial showed statistically significant efficacy on the new primary endpoint at 63.6% vs 48.9% (p = 0.045) in favor of rolapitant.

Exhibit 24
Key Differences between Phase II & Phase III Trial Design for Rolapitant

	Phase 2	Phase 3
Chemotherapy Class	HEC	HEC & MEC
5HT3 Inhibitor	Ondansetron	Granisetron
Primary Endpoint	Overall CR	Delayed Phase CR

Source: Company Data, Morgan Stanley Research

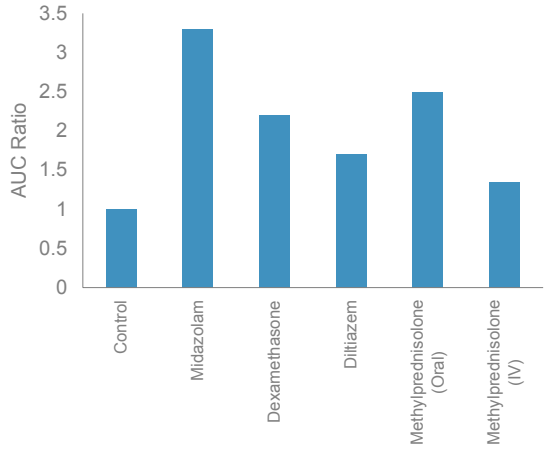
Fourth, rolapitant has no data in MEC. Tesaro proceeded to Phase III in HEC and MEC based on Phase II in HEC alone, which is a risk given that MEC is a more challenging indication with a smaller therapeutic benefit. We see the MEC data as low risk for multiple reasons, including **1)** the mechanism of action is the same in MEC and HEC, **2)** the correlation between positive HEC and MEC for EMEND and casopitant is perfect historically and we know of no instances where an NK1 inhibitor worked in HEC but failed in MEC, and **3)** the MEC trial is large at 1,350 patients which provides significant cushion around meeting the primary endpoint at differences over placebo as low as 5%-6%.

Some of rolapitant's areas of differentiation are low risk

Two of the four points of potential differentiation are low risk. First, rolapitant is a single oral dose (200 mg) compared to either three oral doses (125 / 80 / 80 mg), one IV dose (115 mg) followed by two 80 mg oral doses, or single 150 mg IV dose for EMEND. However, we assume 75% of the long-term market is single dose IV only lowering the importance of this dosing advantage. Second, rolapitant has significantly fewer drug-drug interactions (DDI) than EMEND. In vitro studies show that rolapitant does not inhibit CYP2C9, 2C19, 2D6, and 3A4 enzymes or p-glycoprotein.

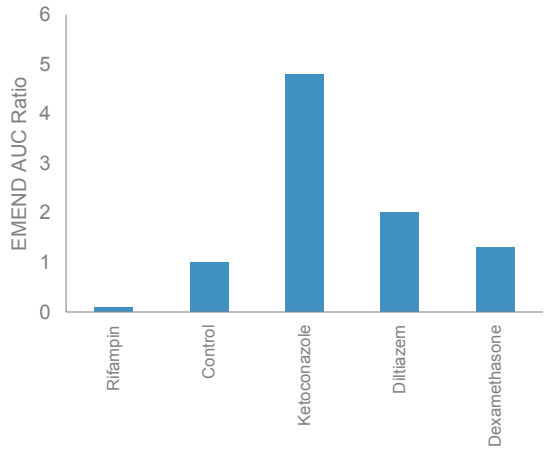
By contrast, EMEND is a moderate inhibitor of CYP3A4. EMEND is known to impact many drugs including corticosteroids dexamethasone and methlyprednisolone (Exhibit 25 and Exhibit 26). EMEND's DDI were not a major source of concern among physicians and awareness of the issue was low. Therefore, we see the commercial importance of fewer DDIs with rolapitant as low. However, all else being equal, we believe pharmacists will opt for the drug with fewer DDIs given the option.

Exhibit 25
EMEND has Multiple Drug-Drug Interactions



Source: GI Drug Advisory Committee Meeting, March 6 2003

Exhibit 26
More Examples

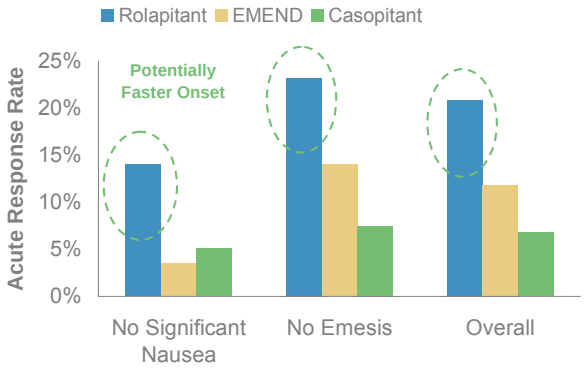


Source: GI Drug Advisory Committee Meeting, March 6 2003

Faster onset of action for rolapitant

The fourth major area of potential differentiation is a faster onset of action for rolapitant. We view this has higher risk for two reasons. First, the mechanism is unclear given that the PK curves suggest similar times to C_{max} and we have no data to suggest more rapid brain penetration for rolapitant relative to EMEND. Second, the clinical importance of a potentially faster onset of action is unclear given that the timing of NK-1 inhibitor administration relative to chemotherapy can be timed to mitigate this effect.

Exhibit 27
Acute Responses Favor Rolapitant across Endpoints

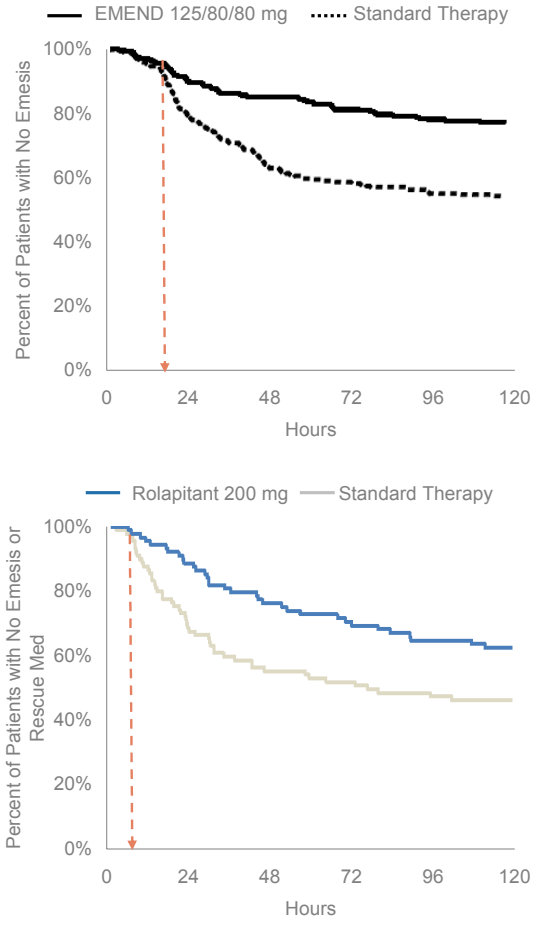


Source: Company Data, Morgan Stanley Research. EMEND Phase II Data: Cancer 2003;97:2290–300; Casipotant Phase III Data: Lancet Oncol 2009; 10: 549–58.

Comparing across the Phase II data, there is a trend toward better placebo-adjusted efficacy for rolapitant at the earliest time points across several relevant endpoints (Exhibit 27). Additionally, when examining the time to onset of emesis for EMEND in Phase III versus rolapitant in Phase II, one observes an apparent earlier separation of the Kaplan Meier curves relative to standard therapy for rolapitant (~ 8 hours) versus EMEND (~16 hours) (Exhibit 28). Admittedly, the comparison is imprecise, as the EMEND analysis included no rescue medications.

Still, literature evidence supports the view that aprepitant provides significantly weaker control of emesis in the acute phase relative to the 5-HT_3 class, with a 0-8 hour no-emesis rate of 36.7% for EMEND versus 82.6% for ondansetron. While these observations directionally favor rolapitant, lacking head-to-head data with EMEND, the Phase II data cannot provide definitive proof of faster onset.

Exhibit 28
Time to Emesis Curves Suggest Faster Onset of Action for Rolapitant



Source: Company Data, Morgan Stanley Research, EMEND FDA label.

The IV development path looks low risk

A unique dynamic for Tesaro is that the oral version of rolapitant will generate the bulk of the clinical data while the IV version will generate the great majority of sales (75% in our model). Therefore, the IV development path deserves discussion.

For timelines, the IV is roughly a year behind the oral version. The current plan assumes that the IV version enters a healthy volunteer Phase I comparative PK study in 4Q12 with data in roughly 2Q13 we estimate. Tesaro then plans to meet with the FDA to discuss next steps. The current timeline implies that the FDA requests a roughly ~200 patient CINV safety study that would begin in 2H13 with data in 2014 and a filing by late 2014 to support a late 2015 launch. Given this pattern, we see

Tesaro's IV rolapitant timeline as low risk with the potential for a faster path to market.

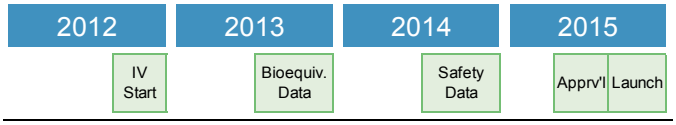
Tesaro has disclosed little about the specifics behind the IV formulation. However, we understand Merck/Schering did all of the formulation and animal work with the product ready to enter human testing. Importantly, our conversations suggest that the animal PK data suggests that matching the oral PK curve is achievable risk and that the formulation is relatively straightforward. Thus while we have not seen any data for the IV formulation and much hinges on its success, diligence supports low risk to the development path.

Rolapitant's oral to IV transition looks much lower risk than EMEND's. EMEND had a two year delay between the oral and IV approvals but we do not see this happening to rolapitant. First, our diligence revealed that EMEND was very difficult to formulate and ultimately required the use of a pro-drug (fosaprepitant) and early formulations were saddled with high pH causing injection site reactions. In addition, EMEND required the transition from a three dose oral to a single dose IV effectively eliminating a straightforward PK-matching bioequivalence study to support approval. Thus, given the pro-drug nature of the IV and different dosing schedule, Merck was forced to run a full head-to-head Phase III trial in HEC comparing the IV and oral formulations.

We expect a much simpler, more rapid path to market for IV rolapitant given the single dose to single dose comparison and the ease of formulation. In fact, in a bull case, if the FDA demands only PK bioequivalence data, the launch could be accelerated by 6 months. With longer-term risk from generic EMEND, this would a material positive for the stock. While not our base case, we understand that all the major excipient of the IV formulation are on the FDA's GRAS (Generally Regarded as Safe) list or have been used previously meaning that a more rapid path to market for IV rolapitant is certainly possible.

Exhibit 29

IV Development Timelines



Source: Company Data, Morgan Stanley Research

Debate #2: Rolapitant and the CINV Market

Market's view: The NK-1 inhibitor market is underpenetrated but growth potential is unclear.

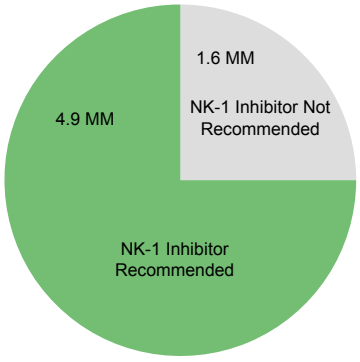
Our view: The NK-1 inhibitor market is only 17% penetrated, which could double by 2020 from expansion of the class, promotion, and wider IV availability. Our base case for rolapitant relies on market growth more than share gains. Thus, the track-record of Tesaro's management team in oncology supportive care is a key asset for the stock. Evidence of superior efficacy for rolapitant will expand the market and lower generic risk.

No free lunch: Commercial risk compensates for rolapitant's lower clinical trial risk. In this section, we outline: 1) the current state of the NK-1 market and the major commercial drivers, 2) our major modeling assumptions for rolapitant including why we are optimistic on rolapitant's share gains and Tesaro's ability to grow the market, and 3) how we think about risk from generic EMEND. Our discussion focuses solely on the US opportunity and does not include any contribution from ex-US licensing royalties. Merck's EMEND is currently annualizing at ~$156 MM ex-US.

The NK-1 antagonist market today

The NK-1 receptor antagonist market is significantly underpenetrated. The best approach to defining the total *potential* NK-1 antagonist market is the addressable portion of the current CINV market (5-HT_3 receptor antagonist units). The current CINV market is roughly 6.6 MM units growing in the low single digits. Using National Comprehensive Cancer Center (NCCN) and the American Society of Clinical Oncology guidelines, Tesaro estimates NK-1 inhibitors can be used in roughly 70-80% of cases or roughly ~5 MM units (Exhibit 30).

Exhibit 30
75% of CINV Market Addressable by NK-1 Inhibitors



Source: Company Data, Morgan Stanley Research

In particular, the guidelines specify the use of an NK-1 inhibitor for all forms of HEC while for MEC the use of an NK-1 inhibitor is recommended under certain conditions (Exhibit 31). The guidelines note that carboplatin ≥ 300 mg/m^2, cyclophosphamide ≥ 600 - 1000 mg/m^2, doxorubicin ≥ 50 mg/m^2 reflect situations where NK-1 use in MEC is associated with category 1 data. However, in our diligence other regimens beyond those cited by NCCN could also be appropriate for NK-1 application in MEC. Note that for oral chemotherapy, NK-1 inhibitors are not recommended.

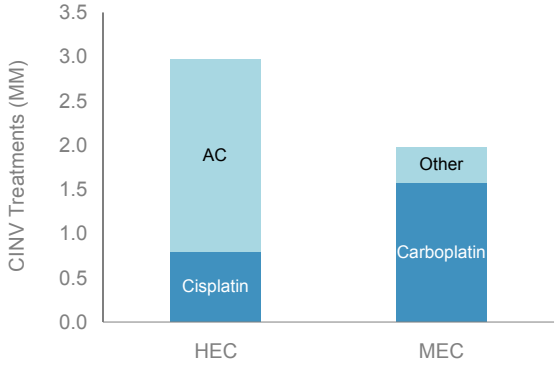
Exhibit 31
What the guidelines say about NK-1 utilization

	HEC	MEC
Intravenous Chemotherapy	Serontonin 5-HT3 Inhibitor	Serontonin 5-HT3 Inhibitor
	+	+
	Steroid	Steroid
	+	+/-
	NK-1 Inhibitor	NK-1 Inhibitor "for selected patients, where appropriate" Carboplatin ≥ 300 mg/m^2, cyclophosphamide ≥ 600 - 1000 mg/m^2, doxorubicin ≥ 50 mg/m^2
Oral Chemotherapy	Serontonin 5-HT3 Inhibitor	Serontonin 5-HT3 Inhibitor

Source: Morgan Stanley Research; NCCN Guidelines Version 1.2012 Antiemesis.

Currently the NK-1 market is significantly underpenetrated. We estimate the NK-1 market with EMEND today is roughly 0.9 MM units or only 18% penetrated leaving obvious room for growth. One caveat is that some physicians in our diligence utilize NK-1 inhibitors *only* for patients that fail 5-HT3 inhibitor + steroid treatment for CINV during the first cycle. Therefore, the addressable market may need to be adjusted lower to take into account the first cycle failure approach. While we have no market insight to size the impact, we would be surprised if this represented more than 10-20% of the total addressable market potential. We do not see this as material relative to the ~17% penetration currently.

Exhibit 32
NK-1 Inhibitor Use Today



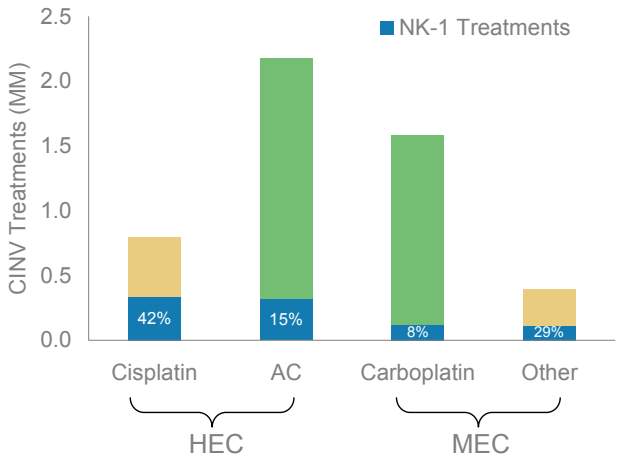
Source: Company Data, Morgan Stanley Research

Going into a deeper level of detail, the CINV market is broken down into to two major segments HEC or Highly Emetogenic Chemotherapy meaning chemotherapy agents that have a ~90% chance of causing nausea and vomiting. HEC is 60% of the NK1 opportunity. The second major segment is called MEC or Moderate Emetogenic Chemotherapy or patients that have a ~30-90% chance of experiencing nausea and vomiting. MEC is 40% of the market.

Drilling down into HEC, there are two major segments: cisplatin based regimens at ~27% and AC or adriamycin-cyclophosphamide for breast cancer at ~73%. NK-1 penetration of these two segments is 42% and 15%, respectively. As expected, penetration is highest in cisplatin-based chemotherapy where nausea and vomiting is nearly universal with the least growth opportunity here. Therefore, the AC market is the key focus for growth in the HEC market.

Taking a closer look at MEC, the major sub-segments include carboplatin-based regimens at 80% and others at 20%. NK-1 use in MEC is just 12% including 8% of carboplatin-treated patients and 29% of all other MEC regimens. Thus, MEC is a broad growth opportunity but especially the carboplatin segment.

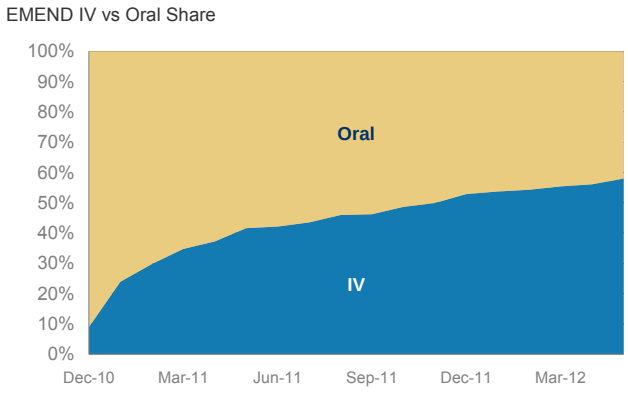
Exhibit 33
Detailed NK-1 inhibitor use patterns



Source: Company Data, Morgan Stanley Research

IMS data show IV is taking over. At YE11, oral EMEND was 47% of sales and 53% was IV. The latest IMS data show that oral EMEND has moved another 500 bps lower at 42% of the market vs IV at roughly 58%. The mix has been rapidly shifting since the introduction of the single dose IV EMEND in November 2010. Given a largely IV based treatment paradigm in most oncology clinics, we see these trends continuing (Exhibit 34).

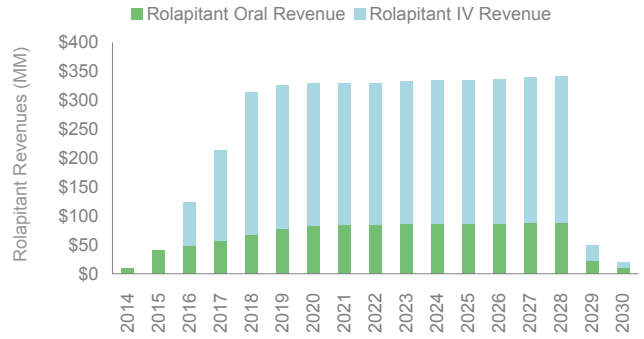
Exhibit 34
IMS data show IV becoming dominant form



Source: IMS Health, Morgan Stanley Research

Our model assumes IV rolapitant accounts for the majority of sales in our model. IV accounts for 73% and oral 27% in our model on an aggregate revenue basis from launch through patent expiry (Exhibit 35). Therefore, we assume that current market trends continue.

Exhibit 35
Rolapitant Oral vs. IV Revenue Mix (MSe)

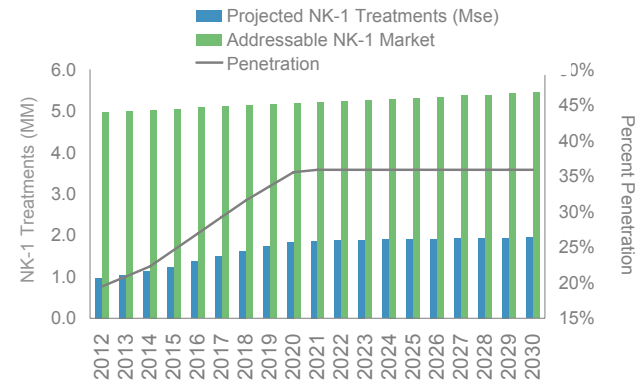


Source: Company Data, Morgan Stanley Research estimates

How we see the NK-1 market developing

A positive thesis on Tesaro requires belief in the growth potential of the NK-1 inhibitor market. Thus, our model envisions significant market growth with rolapitant attaining approximately 55% of the market (Exhibit 36 and Exhibit 37). We assume unit growth of NK-1 treatments to ~1.8 MM from ~0.9 MM currently by 2020, implying an 8% CAGR, meaning penetration of addressable market goes from 18% today to 36% at peak.

Exhibit 36
NK-1 Treatments Can Double by 2020 (MSe)



Source: Company Data, Morgan Stanley Research estimates

Thus, our model is driven by market growth rather than share capture from EMEND. Put another way, even assuming rolapitant takes 100% of the current market would account for ~90% of our estimated rolapitant unit sales in 2020 of ~1 MM and only ~50% of NK-1 unit sales overall. A sensitivity analysis of rolapitant share and market growth to reach our 2020 unit assumption of ~1 MM treatments highlights this dynamic (Exhibit 37). A market growth dominant thesis is more convincing in our view given relatively near-term EMEND generics for oral in 2015 and IV in 2019. Therefore, a larger market allows greater room for generic and branded drug coexistence as in the 5-HT_3 receptor antagonist market.

Exhibit 37
Share and NK-1 Market Growth Sensitivity Analysis to Reach ~1.0 MM Rolapitant Treatments by 2020

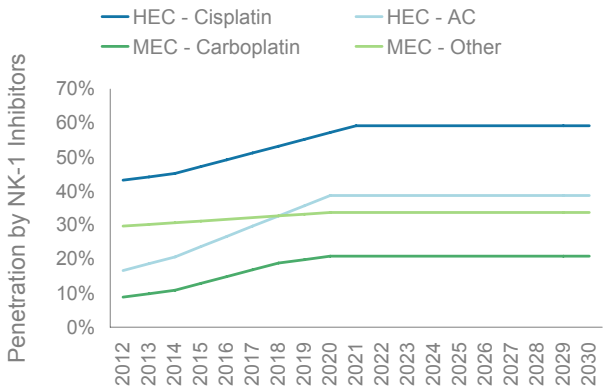
Rolapitant Share of NK-1 Treatments by 2020

Market Growth of NK-1 Treatments by 2020 from Current 0.9 MM	35%	40%	45%	50%	**55%**	60%	65%	70%	75%
50%	0.5	0.5	0.6	0.7	0.7	0.8	0.9	0.9	1.0
60%	0.5	0.6	0.6	0.7	0.8	0.9	0.9	1.0	1.1
70%	0.5	0.6	0.7	0.8	0.8	0.9	1.0	1.1	1.1
80%	0.6	0.6	0.7	0.8	0.9	1.0	1.0	1.1	1.2
90%	0.6	0.7	0.8	0.8	0.9	1.0	1.1	1.2	1.3
100%	0.6	0.7	0.8	0.9	1.0	1.1	1.2	1.3	1.3
110%	0.7	0.8	0.8	0.9	1.0	1.1	1.2	1.3	1.4
120%	0.7	0.8	0.9	1.0	1.1	1.2	1.3	1.4	1.5
130%	0.7	0.8	0.9	1.0	1.1	1.2	1.3	1.4	1.5
140%	0.8	0.9	1.0	1.1	1.2	1.3	1.4	1.5	1.6
150%	0.8	0.9	1.0	1.1	1.2	1.3	1.5	1.6	1.7

Source: Company Data, Morgan Stanley Research estimates

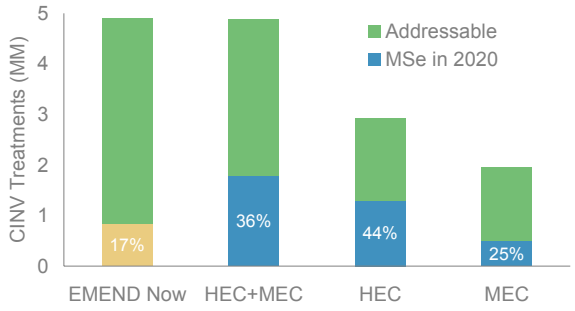
Specifically, our market model assumes broad penetration including: 1) HEC cisplatin from ~45% to ~60%, 2) HEC AC from ~15% to ~40%, 3) MEC carboplatin from ~10% to ~20%, and 4) MEC other from ~30% to ~35%. Therefore, in total, HEC accounts for 75% of total NK-1 market growth and MEC for 25%. HEC-cisplatin accounts for 16% growth, HEC-AC 58%, MEC-carboplatin 23%, and MEC-other just 3%.

Exhibit 38
Projected NK-1 Penetration by Chemotherapy Class



Source: Company Data, Morgan Stanley Research estimates

Exhibit 39
Plenty of Room to Grow the NK-1 Market



Source: Company Data, Morgan Stanley Research estimates

More competition coming to market but the impact is limited. Helsinn and Eisai are developing a single-dose oral combination pill of netupitant, another NK-1 inhibitor, and Aloxi, a 5-HT_3 inhibitor. The partners are running 1) a 1,460 patient Phase III trial in MEC comparing the combination of netupitant and Aloxi to Aloxi alone in MEC with data expected in July 2012 (according to clinicaltrials.gov) and 2) a 400 patient safety study comparing the combination to oral EMEND plus Aloxi in a mixed HEC and MEC population (data expected in September 2012 according to clinicaltrials.gov).

We do not see the netupitant combo pill as a significant risk for several reasons. First, the product is oral only in a market that is ~60% IV and growing today. Second, it will be limited to the segment of the 5-HT_3 market that uses branded Aloxi. Third, the Phase III trial is in MEC *only* and it is not clear if this is a viable regulatory path lending uncertainty to the timelines. Fourth, a third player should grow the market. Fifth, another single dose oral will further take share and minimize risk from the genericization of oral EMEND in 2015/2016. Finally, we know very little about netupitant's activity profile relative to EMEND and rolapitant.

What are the major drivers of market growth in our model?

1. **Promotional intensity**. Our diligence suggests that Merck has a field force of just 75 today. Our model assumes Tesaro will add another ~90-100 sales people. This will allow market growth especially beyond large academic centers where EMEND is well penetrated today.

2. **More aggressive contracting**. We understand Merck does not discount or provide large contracts for EMEND. Therefore, we expect Tesaro will aggressively pursue this opportunity for large players like US Oncology (25% of the market), large practice networks such as Florida Cancer Specialists, Tennessee Oncology, Georgia Cancer Specialists & others (collectively another 25% of the market) and large academic cancer centers (30% of the market).

3. **Broader IV availability**. A three dose oral form has limited market growth and we expect the IV form to continue to grow the market.

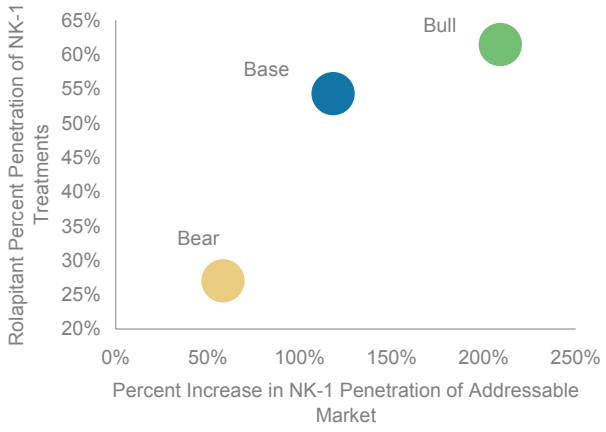
4. **Rolapitant's various differences** including fewer drug-drug interactions, faster onset, single dose oral, and a longer half life could help to grow the market.

5. **Tesaro management experience.** The Tesaro management team successfully grew another product in this market despite significant generic competition, which is critical to our view of the success of rolapitant.

Exploring the bull and bear cases for rolapitant

We discuss our base case for rolapitant above. In our bull case, we assume rolapitant achieves meaningful efficacy differentiation from EMEND with upside to market growth and share assumptions. Specifically, we assume market growth goes to ~210% from ~120% in our base case and share to ~60% from ~50% in our base case. In our bear case, rolapitant fails to achieve meaningful commercial traction and market growth sputters. Here, we assume market growth goes to ~60% from ~120% in our base case and share to ~25% from ~50% in our base case.

Exhibit 40
Bull to Bear Market Penetration Scenarios for NK-1 Class & Rolapitant



Source: Company Data, Morgan Stanley Research estimates

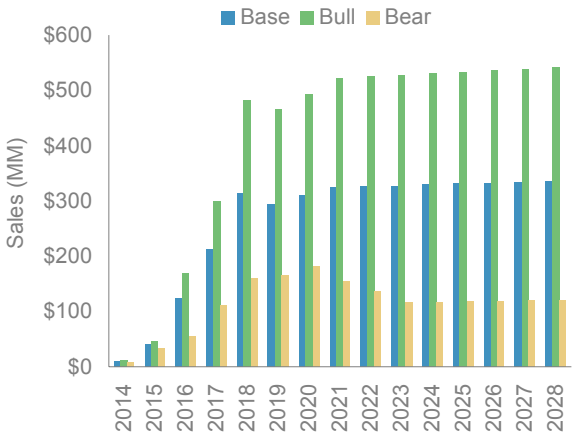
Exhibit 41

Rolapitant Market Model (Base case shown)

	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030
NK-1 Market Model																	
US Annual 5HT3 CINV Treatments (MM)	6.7	6.7	6.8	6.8	6.8	6.9	6.9	6.9	7.0	7.0	7.0	7.1	7.1	7.1	7.2	7.2	7.3
% of growth	*0.5%*	*0.5%*	*0.5%*	*0.5%*	*0.5%*	*0.5%*	*0.5%*	*0.5%*	*0.5%*	*0.5%*	*0.5%*	*0.5%*	*0.5%*	*0.5%*	*0.5%*	*0.5%*	*0.5%*
Addressable Market (ASCO/NCCN Guidelines):																	
Total Potential Market	**5.0**	**5.0**	**5.1**	**5.1**	**5.1**	**5.2**	**5.2**	**5.2**	**5.2**	**5.3**	**5.3**	**5.3**	**5.3**	**5.4**	**5.4**	**5.4**	**5.4**
Highly Emetogenic Chemotherapy (HEC)	3.0	3.0	3.0	3.1	3.1	3.1	3.1	3.1	3.1	3.2	3.2	3.2	3.2	3.2	3.2	3.2	3.3
Cisplatin	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.9	0.9	0.9	0.9	0.9
AC	2.2	2.2	2.2	2.2	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.3	2.4	2.4	2.4	2.4
MEC	2.0	2.0	2.0	2.0	2.1	2.1	2.1	2.1	2.1	2.1	2.1	2.1	2.1	2.1	2.2	2.2	2.2
Carboplatin	1.6	1.6	1.6	1.6	1.6	1.6	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7
Other	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4
NK1 Penetration of CINV																	
Highly Emetogenic Chemotherapy (HEC)	0.8	0.9	1.0	1.1	1.2	1.3	1.4	1.4	1.4	1.4	1.4	1.4	1.4	1.4	1.4	1.4	1.4
Cisplatin	0.4	0.4	0.4	0.4	0.4	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5
AC	0.46	0.53	0.60	0.67	0.74	0.81	0.88	0.89	0.89	0.89	0.90	0.90	0.91	0.91	0.92	0.92	0.93
Moderately Emetogenic Chemotherapy (MEC)	0.30	0.33	0.37	0.41	0.44	0.46	0.49	0.49	0.49	0.49	0.50	0.50	0.50	0.50	0.51	0.51	0.51
Carboplatin	0.2	0.2	0.2	0.3	0.3	0.3	0.3	0.3	0.3	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4
Other	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Total NK-1 Treatments	**1.1**	**1.2**	**1.4**	**1.5**	**1.6**	**1.7**	**1.8**	**1.9**	**1.9**	**1.9**	**1.9**	**1.9**	**1.9**	**1.9**	**1.9**	**1.9**	**2.0**
% of addressable	*22%*	*25%*	*27%*	*29%*	*32%*	*34%*	*36%*	*36%*	*36%*	*36%*	*36%*	*36%*	*36%*	*36%*	*36%*	*36%*	*36%*
% market growth	*7%*	*11%*	*10%*	*9%*	*8%*	*7%*	*6%*	*1%*	*1%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*1%*	*0%*
% market growth from current	*25%*	*39%*	*53%*	*67%*	*81%*	*93%*	*106%*	*109%*	*110%*	*111%*	*112%*	*113%*	*114%*	*115%*	*116%*	*117%*	*118%*
Rolapitant																	
Oral Share																	
Rolapitant Oral HEC Share	*3%*	*11%*	*12%*	*13%*	*14%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*7%*	*7%*
Rolapitant Oral MEC Share	*5%*	*15%*	*16%*	*17%*	*17%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*9%*	*8%*
IV Share																	
Rolapitant IV Share HEC	*0%*	*0%*	*18%*	*35%*	*50%*	*45%*	*42%*	*42%*	*42%*	*42%*	*42%*	*42%*	*42%*	*42%*	*42%*	*9%*	*8%*
Rolapitant IV Share MEC	*0%*	*0%*	*25%*	*40%*	*55%*	*60%*	*55%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*10%*	*0%*
Rolapitant Units																	
Rolapitant Oral Treatments HEC (MM)	0.0	0.10	0.12	0.14	0.16	0.18	0.20	0.20	0.20	0.20	0.20	0.20	0.20	0.21	0.21	0.10	0.10
Rolapitant Oral Treatments MEC (MM)	0.0	0.03	0.04	0.05	0.05	0.06	0.06	0.06	0.06	0.06	0.06	0.06	0.06	0.06	0.06	0.03	0.03
Total Oral Rolapitant Treatments	**0.03**	**0.13**	**0.16**	**0.18**	**0.21**	**0.24**	**0.26**	**0.26**	**0.26**	**0.26**	**0.26**	**0.27**	**0.27**	**0.27**	**0.27**	**0.14**	**0.13**
Rolapitant IV Treatments HEC (MM)	0	0	0.18	0.38	0.59	0.57	0.57	0.58	0.58	0.58	0.59	0.59	0.59	0.60	0.60	0.13	0.12
Rolapitant IV Treatments MEC (MM)	0	0	0.06	0.11	0.17	0.20	0.19	0.17	0.17	0.18	0.18	0.18	0.18	0.18	0.18	0.04	0.00
Total IV Rolapitant Treatments	**0.00**	**0.00**	**0.24**	**0.49**	**0.76**	**0.77**	**0.76**	**0.75**	**0.76**	**0.76**	**0.76**	**0.77**	**0.77**	**0.78**	**0.78**	**0.17**	**0.12**
Total Rolapitant Treatments (MM)	**0.03**	**0.13**	**0.40**	**0.67**	**0.97**	**1.01**	**1.02**	**1.01**	**1.02**	**1.02**	**1.03**	**1.03**	**1.04**	**1.04**	**1.05**	**0.30**	**0.25**
% total share	*3%*	*11%*	*29%*	*45%*	*60%*	*58%*	*55%*	*54%*	*54%*	*54%*	*54%*	*54%*	*54%*	*54%*	*54%*	*15%*	*13%*
% oral	*100%*	*100%*	*40%*	*27%*	*22%*	*24%*	*25%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*45%*	*53%*
% IV	*0%*	*0%*	*60%*	*73%*	*78%*	*76%*	*75%*	*74%*	*74%*	*74%*	*74%*	*74%*	*74%*	*74%*	*74%*	*55%*	*47%*
Market Share to Rolapitant																	
Total Oral Market (% of total)	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*	*75%*
Total IV Market (% of total)	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*	*25%*
Rolapitant Oral Market Share	*12%*	*42%*	*46%*	*49%*	*52%*	*56%*	*56%*	*56%*	*56%*	*56%*	*56%*	*56%*	*56%*	*56%*	*56%*	*28%*	*27%*
Rolapitant IV Market Share	*0%*	*0%*	*23%*	*44%*	*62%*	*59%*	*55%*	*54%*	*54%*	*54%*	*54%*	*54%*	*54%*	*54%*	*54%*	*11%*	*8%*
Rolapitant Revenue:																	
Price Per treatment	$300	$306	$312	$318	$325	$292	$305	$320	$320	$320	$320	$320	$320	$320	$320	$160	$80
% growth		*2%*	*2%*	*2%*	*2%*	*-10%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*-50%*	*-50%*
Oral Revenue	$10	$40	$49	$58	$69	$70	$78	$83	$84	$84	$85	$85	$86	$86	$86	$22	$10
IV Revenue	$0	$0	$75	$156	$246	$224	$232	$241	$242	$243	$245	$246	$247	$248	$249	$26	$9
Total Rolapitant Revenue	**$10**	**$40**	**$124**	**$213**	**$314**	**$294**	**$310**	**$324**	**$326**	**$328**	**$329**	**$331**	**$333**	**$334**	**$336**	**$48**	**$20**

Source: Company Data, Morgan Stanley Research estimates

Exhibit 42
Rolapitant Bull to Bear Case – Revenue Summary



Source: Company Data, Morgan Stanley Research estimates

Rolapitant stand-alone valuation

Tesaro's valuation blends rolapitant and the pipeline agents making the stand-alone value of the lead asset a key consideration. To model rolapitant alone, we strip out most R&D, a portion of G&A, and any sales and marketing expense related to other assets. We run the DCF to 2029 and use a relatively high WACC of 17.5% to account for the remaining clinical and commercial risks. Our analysis yield ~$10 per share in stand-alone value for rolapitant using our base case (Exhibit 43). Varying the discount rate from 10-20% yields a stand-alone valuation range of $24 to $8 per share.

Exhibit 43
Rolapitant Stand-Alone DCF Valuation

DCF Details	
Discount Rate	17.5%
Discounted Cash Flow (2012 - 2029)	$267
Terminal Value	$0
Rolapitant DCF	$267
Per Share Value	**$10**

Source: Company Data, Morgan Stanley Research estimates

A broader framework for valuation across various rolapitant commercial and clinical outcomes. We performed a wide-ranging sensitivity analysis using various market growth and share scenarios for both rolapitant revenue (Exhibit 44) and stand-alone DCF value (Exhibit 45). Our analysis shows that each 500 bps of share is worth ~$15 MM to peak sales and each 500 bps of market growth is worth ~$34 MM to peak sales.

Exhibit 44
Peak Revenues (MM) Share Sensitivity Analysis

% of Addressable Market \ Rolapitant Share of NK-1 Treatments	40%	45%	50%	55%	60%	65%	70%
17%	$115	$129	$144	$158	$172	$187	$201
22%	$149	$167	$186	$204	$223	$242	$260
27%	$183	$205	$228	$251	$274	$297	$319
32%	$216	$243	$270	$297	$324	$352	$379
37%	$250	$281	$313	$344	$375	$406	$438
42%	$284	$319	$355	$390	$426	$461	$497
47%	$318	$357	$397	$437	$477	$516	$556
52%	$352	$395	$439	$483	$527	$571	$615

Source: Company Data, Morgan Stanley Research estimates

Running this through our DCF yields a valuation range for rolapitant of $4 to $21 per share (Exhibit 45). Our rough DCF sensitivity shows that each 500 bps of share is worth ~$0.5-$2 per share and each 500 bps of market growth is worth $1-2 per share.

Exhibit 45
Rolapitant Stand-Alone DCF/Share Sensitivity Analysis

% of Addressable Market \ Rolapitant Share of NK-1 Treatments	40%	45%	50%	55%	60%	65%	70%
17%	$4	$4	$5	$5	$6	$6	$7
22%	$5	$6	$6	$7	$8	$8	$9
27%	$6	$7	$8	$9	$9	$10	$11
32%	$7	$8	$9	$10	$11	$12	$13
37%	$9	$10	$11	$12	$13	$14	$15
42%	$10	$11	$12	$13	$15	$16	$17
47%	$11	$12	$14	$15	$16	$18	$19
52%	$12	$14	$15	$17	$18	$20	$21

Source: Company Data, Morgan Stanley Research estimates. 17.5% WACC

Taking the EMEND generic risk head on

Perhaps the most challenging issue for the Tesaro story is that EMEND will go generic in the relatively near term (Exhibit 46). Oral EMEND will go generic in 2015 (February) only two years after the potential launch of oral rolapitant in late 2014. IV EMEND will go generic in 2019 (March) only three years after the potential launch of IV rolapitant in later 2015.

Exhibit 46
Rolapitant Must Run the Gauntlet of Generic EMEND

Source: Company Data, Morgan Stanley Research estimates

More importantly, the bulk of the stand-alone value for rolapitant lies beyond the launch of IV EMEND. We estimate that >85% of the stand-alone DCF value lies beyond 2019 in our model. Granted a steeper launch curve could shift this mix modestly, but we suspect the same will prove true of most investor models. Therefore, comfort that rolapitant can prosper *despite* generic EMEND is key to outperformance.

Exhibit 47

Majority of Rolapitant Value Lies Post-2019 (MSe)

DCF Pre & Post 2019 EMEND Patent Expiry			
	2012 - 2019	2019 - 2029	2012-2029
Total Rolapitant Revenues (B)	$996	$2,999	$3,995
Average Revenue Per Year*	$166	$273	$235
Revenue Range	$0 - $314	$48 - $336	$0 - $336
DCF (MM) *17.5% WACC*	**$23**	**$244**	**$267**

** Excludes pre-commerical years 2012 & 2013*

Source: Company Data, Morgan Stanley Research estimates

Fundamentally, combating generic alternatives for rolapitant will depend on three factors: 1) the extent of rolapitant's differentiation from EMEND following Phase III, 2) a history of generic branded co-existence in CINV, and 3) Tesaro management experience.

The more differentiation the better. As we discuss in detail on pages 21 to 23, our base case assumes modest differentiation from EMEND based on drug-drug interactions and any perceived benefits of a longer half-life. That said, the generic risk comes down dramatically if rolapitant proves differentiated efficacy in Phase III particularly for nausea in the delayed phase where our diligence reveals the greatest unmet medical need. Historically, part of Aloxi's success (the 5-HT_3 receptor antagonist that the Tesaro management team built despite generic risk) was the existence of head to head data with ondansetron suggesting better efficacy.

Branded rolapitant can live in peace with generic EMEND. The history of the 5-HT_3 market shows that branded drugs can succeed despite generic competition (Exhibit 49). Aloxi (still the only remaining branded 5-HT_3 antagonist) shows that while the launch of generic ondansetron impacted sales for three quarters the growth resumed subsequently and was sustained thereafter. Therefore, we suspect this was helped by contracting and an incentive system that still rewards IV branded drugs over generics. Moreover, given that rolapitant and EMEND are distinct chemical entities and the fail-generic first paradigm is not widespread in oncology care, we see room for rolapitant to grow despite generic EMEND.

A clear risk to the stock will be changing perceptions around the commercial and payor environment given the unique dynamics in supportive care. Granted, concerns will continue in an increasingly cost pressured environment and generic pressure could prove greater in three to five years when this become an issue than the current market environment might suggest. For that reason, we see a clear rationale for Tesaro to pursue a head to head study with EMEND following approval of the single dose IV to combat payor scrutiny when EMEND goes generic.

Perversely, Aloxi generics and G-CSF, EPO biosimilars could help by removing cost pressures elsewhere in supportive care market lowering incentives in the NK-1 market. Aloxi currently sells ~$400 MM in the US and will go generic in April 2015 while Amgen's Neupogen revenues in the US are annualizing at $950 MM for 2012 with generics launching as soon as late 2013 and Neulasta in the US is a $3.2 B drug with biosimilars in 2015+. Similarly, Amgen's Aranesp still sells ~$800 MM in oncology in the US with biosimilars in July 2014. With IV rolapitant likely launching in late 2015/2016, the timelines fit well with a lessening of cost pressures in oncology supportive care. This could spur oncologists to consider other branded IV agents like rolapitant assuming the ASP+ system survives.

Exhibit 48

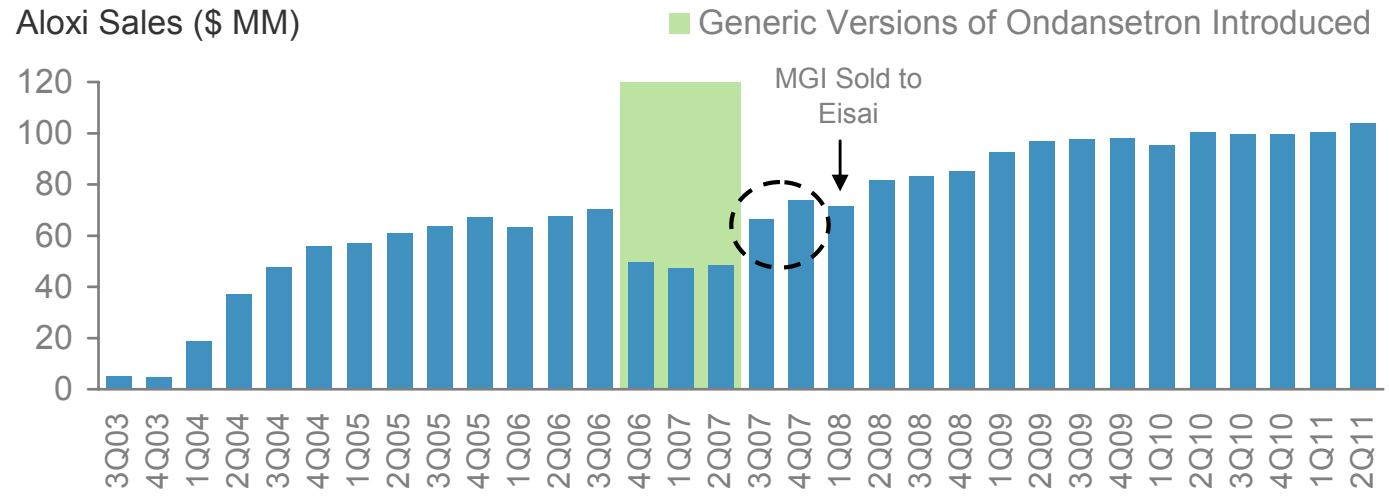
Tesaro Management Has Significant Commercial Experience with Oncology Products

	Lonnie Moulder, MBA	Mary Lynne Hedley, PhD	Rick Rodgers, MBA	Key Accomplishments
Tesaro	CEO	President & CSO	EVP & CFO	
MGI Pharma	President & CEO	EVP & CSO	SVP, Controller & CAO	Established 200+ oncology sales force - launched Aloxi & Dacogen
Eisai North America	EVP	EVP	NA	Developed oncology strategy including HALAVEN
Abraxis BioScience	President & CEO	EVP Operations & CSO	SVP & CFO	Expanded and directed a 150+ oncology sales force

Source: Company Data, Morgan Stanley Research

Rolapitant in the hands of a different management team is not the same asset. The direct experience of the core Tesaro management team in launching and succeeding with a CINV agent in the face of generic alternatives is a major driver of the risk-adjusted rolapitant asset valuation. This lends credibility to the product that may be harder to come by with other management teams. This is particularly true in terms of understand marketing and contracting dynamics, which are nearly as critical as the clinical data. However, visibility on this front will be limited until commercial truly begins in 2015 and 2016 for the IV.

Exhibit 49
Aloxi Sales Trajectory Reflects Management's Strengths in Commercial Execution

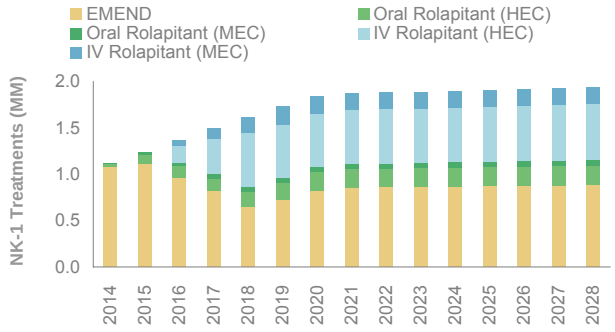


Source: Company Data, Morgan Stanley Research

Our model takes a middle road with respect to generic risk. At base, our model assumes that rolapitant does well despite generic EMEND. That said, we do not assume significant growth beyond the 2015 generic oral EMEND for oral rolapitant or the 2019 generic EMEND for IV rolapitant. Moreover, we assume flat price after generics enter the market and we do not model oral generic meaningfully impacting use of the IV. We believe oral will be a minority of the market by the time generics launch in 2016 as we estimate that are 40% currently down from 45% in 2011. Second, we see a clear rationale for IV CINV agents over orals.

However, our model does place heavy importance on a successful launch for rolapitant particularly for the IV. For example, our model sees IV rolapitant achieve ~90% of peak sales by the time IV EMEND goes generic in 2019 (Exhibit 50). This rapidity of the launch tracks well with the Aloxi experience, which achieved ~55% of peak sales prior to generic ondansetron by the sixth quarter (4Q04) following the launch in 3Q03.

Exhibit 50
Minimal Growth for Oral and IV Rolapitant Post Oral (2015) and IV (2019) EMEND Patents



Source: Company Data, Morgan Stanley Research estimates

Debate #3: Looking for Value Beyond Rolapitant?

Market's view: Limited visibility on early-stage pipeline assets caps conviction.

Our view: Tesaro in-licensed two oncology assets: 1) TSR-011, an ALK inhibitor from Amgen and 2) niraparib, a Phase I PARP inhibitor from Merck. We include niraparib in our base case valuation after proof of concept in BRCA mutation positive ovarian cancer and a safety profile consistent with the class. Clarity on the development strategy in 2H12 is the next catalyst. Lacking patient data, we need more information on TSR-011 given multiple more advanced competitive agents.

Exhibit 51
Pipeline Early But Attractive

	Niraparib	TSR-011
Indication(s)	Solid Tumors	Lung Cancer
Status	Phase 2 Development Plan Underway	Phase 1 Start 2H12
Data Expected	2013 / 2014	Safety 2013, Efficacy 1H14
Mechanism	PARP Inhibitor	ALK Inhibitor
In-Licensed From	Merck	Amgen
In Our Valuation?	Yes	No

Source: Company Data, Morgan Stanley Research

The Phase I/II pipeline for Tesaro is a developing part of the story and holds the potential for value creation in the interim given still protracted timelines for rolapitant Phase III data in late 2013 and potential launch in late 2014. Overall, we lack extensive data to evaluate these assets given that TSR-011 is pre-clinical and niraparib was recently in-licensed in May 2012. Our base case includes a valuation contribution from niraparib given that the asset has shown single agent proof of concept in a Phase I study. However, this could be viewed as representing a stand-in valuation credit for the pipeline and consistent with this, we use a high discount rate of 17.5%.

TSR-011 is still pre-clinical and will enter a crowded space with several second generation ALK-inhibitors that are one to two years ahead of Tesaro in development. By contrast, niraparib is new to Tesaro but we see 1) early single-agent response rates in BRCA-1/2 mutant ovarian cancer comparable to other PARP inhibitors like AstraZeneca's olaparib and 2) timelines broadly comparable to competitors.

TSR-011: Early but Interesting in a Crowded Corner of the World

TSR-011 is Tesaro's pre-clinical ALK inhibitor and we do not include a valuation contribution in our base case model. The drug was in-licensed from Amgen in March 2011. Details on the transaction are limited but included a $0.5 MM upfront payment and aggregate milestones (development and sales) of $138 MM. The royalty rate on future sales ranges from the mid-single to low double digits worldwide.

Patent: TSR-011 has loss of exclusivity in 2030, excluding possible extensions.

Timelines for TSR-011. TSR-011 will enter the clinic in 4Q12. Tesaro will accelerate through the first few levels in the dose escalation by using healthy volunteers before reverting to heavily pre-treated cancer patients for higher doses. We expect some early Phase I data and/or MTD during 2H13. Phase II or III could begin as soon as mid-2014 with a potentially rapid path to market as rapidly as 2018 or 2019 if a post-crizotinib indication or alternate tumor-type (neuroblastoma) indication remains open by the time TSR-011 completes Phase I/II.

Three things we need to see to put TSR-011 in our model: **1)** early signs of efficacy and tolerability in ALK+ lung cancer patients and/or crizotinib failures, **2)** a reasonable early safety profile, and **3)** competitive updates from Novartis and Ariad among others during 2H12 and 2013.

Exhibit 52
Competitive Landscape for ALK Inhibitors Intense

Company	Drug	Stage	Potency (IC50, nM) *Lower Numbers Are Better* Enzymatic	Cell-based	Clinical Efficacy Data
Pfizer	Crizotinib	FDA Approved	3.7	108	50% - 61% Response Rate in NSCLC
Novartis	LDK378	Phase 1	0.15	27	81% Response Rate in NSCLC Patients Failing Crizotinib
Chugai	CH5424802	Phase 1/2	1.9	3.0	100% response rate at doses of 240 mg twice daily or higher
Ariad	AP26113	Phase 1/2	0.62	5.3	First clinical data in 3Q12 (ESMO)
Astellas	ASP-3026	Phase 1	NA	NA	1H13 (Blood Cancers & Solid Tumors) 1H14 (Solid Tumors)
Xcovery	X-396	Phase 1	NA	NA	Mid-2013
GSK	GSK-1838705	Pre-clinical	NA	NA	NA
Nerviano	NMS-E628	Pre-clinical	NA	NA	NA
Tesaro	TSR-011	Pre-clinical	1.6	2	1H14

Source: Company Data, Morgan Stanley Research; Bioorg. Med. Chem. 20 (2012) 1271–1280; PNAS108 (18) 7535-7540, 2011.

What do we know about TSR-011? Amgen designed TSR-011 based on careful analysis of the crystal structure of the ALK kinase to optimize potency and specificity. This could be a differentiating factor given that competitors including Pfizer's Xalkori has significant MET activity and Ariad's AP2113 is a dual mutant EGFR and ALK inhibitor.

The pre-clinical basic science for the TSR-011 is solid with clear activity against a major ALK gatekeeper mutation L1196M in ALK that confers resistance to Xalkori (crizotinib) with an IC_{50} of 0.1 nM versus 19.2 nM for crizotinib. Similarly, TSR-011 was active against the ALK resistance mutation R1275Q with an IC_{50} of 0.5 nM versus 3.7 nM for crizotinib. Crizotinib is an active drug. However, the resistance barrier proved relatively low in the clinic with ALK amplification sufficient to cause resistance. TSR-011 has a reasonable IC_{50} of 10-11nM in cell based assays of ALK amplification.

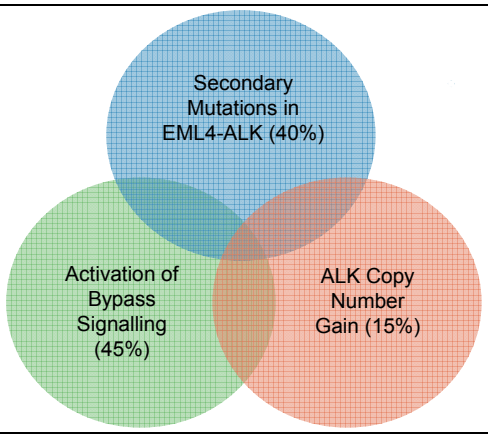
ALK is a validated target given the approval of Pfizer's crizotinib. So, why not more optimism about TSR-011? While early, TSR-011 looks like a potent ALK inhibitor, but we have major questions remaining about both the ALK development landscape and TSR-011.

First, toxicities and tolerability have varied widely to date for the class. Therefore, we need more information on TSR-011's activity *and* tolerability profile relative to competitive agents. For example, crizotinib's major toxicities include visual disturbances (~60% in Phase III), nausea/vomiting (57%/45%), and diarrhea (49%), Novartis' LDK378 showed nausea (45%), vomiting (36%), and diarrhea (29%) in Phase I, while Chugai's CH5424802 appears relatively well tolerated.

Second, the pipeline is crowded and we expect significant updates from Ariad on AP26113 in 3Q12 at ESMO and Novartis in 2013 among others. Therefore, we would like to get a better sense of the competitive environment before judging the potential of TSR-011. The more advanced agents from Novartis and Ariad could make it more difficult for Tesaro to leverage a more rapid path to market in crizotinib failures for example.

Exhibit 53

Multiple Resistance Mechanisms to ALK Inhibitors Suggest Fractured Market



Source: Morgan Stanley Research; Clin Cancer Res. 2012 Mar 1;18(5):1472-82.

Finally, the post crizotinib market opportunity is not yet clear in our view. Studies in patients failing crizotinib has found three major mechanisms of resistance including 1) ALK mutations (40%), 2) ALK amplification (15%), and 3) activation of by-pass signaling mechanisms like MET and EGFR (45%). While most second/third generation ALK inhibitors should be able to overcome ALK amplification, this is only 15% of the crizotinib failure market.

For the remaining 85% of the market, the breadth of the market addressable by each second/third generation drug is unclear. For the 40% of the patients failing crizotinib due to secondary mutations, early data shows variable activity of the second/third generation agents across various crizotinib-resistance mutations meaning newer agents may not prove useful in all patients failing crizotinib (Exhibit 54). This observation makes sense given that each of the newer ALK inhibitors binds to the protein in a different way. In addition, this could complicate clinical development in crizotinib failures.

That said, Novartis' LDK378 showed an 81% (21/26) response rate in patients failing crizotinib at ASCO 2012, which is encouraging. Moreover, a first line strategy is still possible but would likely require a head-to-head trial with crizotinib with longer timelines.

Exhibit 54
Differential Mutational Sensitivity across ALK Inhibitors Likely to Split Market Further

ALK Mutation	Location in ALK	Drug Resistance/Sensitivity Profiles TAE684 (Novartis)	Crizotinib (Pfizer)	CH5424802 (Chugai)	AP26113 (Ariad)	TSR-011 (Tesaro)
L1196M	Gatekeeper	Sensitive	Resistant	Sensitive	Sensitive	Sensitive
R1275Q	Kinase domain	Sensitive	Sensitive	Sensitive	No data	Sensitive
F1174L, F1174C*	Kinase domain	Sensitive	Resistant	Sensitive	Sensitive*	Sensitive
G1269A, G1269S*	Vicinity of DFG motif	Sensitive	Resistant	No data	Sensitive*	No data
L1198P	Hinge region	Resistant	Resistant	No data	No data	No data
D1203N	Solvent front	Resistant	Resistant	No data	No data	No data
G1202R	Solvent front	Resistant	Resistant	Resistant	No data	No data
1151Tins	Loop N-terminal of α-helix C	Resistant	Resistant	Resistant	No data	No data
C1156Y	Loop N-terminal of α-helix C	No data	Resistant	Sensitive	No data	No data
S1206Y, S1206R*	Solvent front	No data	Resistant	No data	Resistant*	No data

Source: Company Data, Morgan Stanley Research; Cancer Cell 2011, 19, 679–690; Sci Transl Med 4, 120ra17 (2012); Clin Cancer Res 2011, 17:7394-7401; Clin Cancer Res; 17(8); 1–6; N Engl J Med. 2010, 363:1734–9; AACR 2010 LB-298 Poster.

Finally, bypass signaling presents a difficult challenge for highly specific ALK inhibitors like TSR-011. Clearly, if other signaling pathways pick up the baton from ALK to drive a given cancer, TSR-011 has a lower probability of activity. For example, early data suggests activation of EGFR can result in crizotinib resistance, which could favor a drug like Ariad's AP26116, a dual inhibitor of ALK and EGFR.

As a final note, the market for ALK inhibitors could prove larger than expected given that ALK-translocations have been found in a variety of cancers including kidney and ovarian. In addition, the kinase ROS1 is also often sensitive to ALK inhibitors, which could provide another area for development. However, these findings are all early and we have time to see how these alternate markets develop.

Niraparib looks interesting

Niraparib is a PARP inhibitor in-licensed from Merck in May 2012. PARP inhibitors are a class of oncology drugs that block part of the cellular machinery that helps to repair damaged DNA. PARP inhibitors have gotten significant attention for their potential to sensitize cancer cells to chemotherapy, which of course function by damaging DNA either directly or indirectly.

The terms of the deal with Merck include a $7 MM upfront payment, a $57 MM development milestone for approval in the first indication and $29.5 MM for each additional indication, as well as sales milestones of $87.5 MM. The worldwide royalty rate is in the low teens.

Patent term: Based on current expectations, the composition of matter patent of niraparib extends to 2028 with the potential for 5 years of patent extension.

Timelines: Niraparib completed an 80 patient Phase I trial and is ready to enter Phase II. Tesaro will be meeting with FDA to discuss the Phase II development plan including any potential for a rapid path to market in the next few months. Thus, we currently have no solid timeline information. Our expectation is that Phase II could start in early 2013 and our model assumes a launch in 2017 assuming that monotherapy in heavily pre-treated women with BRCA mutant positive breast and ovarian cancer is a viable path to market. Merck is currently completing a Phase I trial combining niraparib with temozolomide focusing in part of glioblastoma (a form of brain cancer) and melanoma that is slated to complete in 1Q13 (according to clinicaltrials.gov). Tesaro has disclosed that full doses of temozolomide with niraparib were well tolerated.

The outcome of Tesaro's FDA meetings will be the most significant near term catalyst related to niraparib. We expect Tesaro to meet with the FDA in 2H12, and we await an update from the company on more defined timing and development plans. Tesaro has said little about the development plan except that potential tumor types include breast, gastric, lung, and ovarian cancer. We anticipate Tesaro will move into several Phase II trials in combination with chemotherapy. Most important will be if the FDA agrees to a Phase III trial in women with mutant BRACA1/2 with ovarian and/or breast cancer where the proof of concept is strongest for PARP inhibitors. We can envision multiple designs including a monotherapy study versus best supportive case in heavily pre-treated patients or a monotherapy study in comparison with chemotherapy in earlier line patients among other strategies.

What do we know about niraparib so far? Merck designed niraparib (originally MK-4827) by optimizing a well-trodden chemical core in PARP inhibitor drug design. Niraparib is a potent inhibitor of PARP 1 and 2 (IC_{50} of 3.8 nM and 2.1 nM, respectively) but not PARP 3, v-PARP, or TANK-1. Niraparib also showed high cytotoxicity in BRCA1/2 mutated cells lines (CC_{50} of 18 nM and 90 nM, respectively) but not BRCA wild-type, supporting the synthetic lethality hypothesis where PARP inhibition of DNA repair and faulty homologous recombination in BRCA mutated tumors synergize to promote cell death.

Merck performed a Phase I study of niraparib of 80 patients that was presented at ASCO 2011. In the dose escalation portion of the Phase I trial, the maximum tolerated dose for

niraparib was 300 mg daily with a dose limiting toxicity of thrombocytopenia (low platelets) consistent with the PARP inhibitor mechanism. Importantly, pharmacodynamic assays show that niraparib increased signs of DNA damaged in patients (H2AX foci) and PARP activity declined by 50%. Importantly, these markers of activity are consistent with the other PAPR inhibitors in development discussed below. Therefore, the Phase I data show consistent signs that niraparib is an active PARP inhibitor.

After identifying the correct dose, the trial enrolled 30 patients with ovarian cancer where PARP inhibitors have shown activity to date. Among 39 platinum resistant ovarian cancer patients in the Phase I trial, the response rate was 26% and clinical benefit rate was 46%. Most importantly, in the 19 women with BRCA1/2 mutations (the genes associated with hereditary breast and ovarian cancer) the response rate was 37%. Given the dose escalation design, these rates likely understate niraparib's efficacy.

Exhibit 55

Phase I Niraparib Responses Reveal Meaningful Efficacy in Ovarian Cancer

Response Type	Overall (Ovarian, Breast, Lung, Other)		Platinum Resistant Ovarian Cancer							
			BRCA Status						Total	
			Positive		Negative		Unknown			
	N	%	N	%	N	%	N	%	N	%
Progressed	50	63%	7	37%	2	40%	7	47%	16	41%
Stable ≥ 12 weeks	18	23%	4	21%	0	0%	4	27%	8	21%
Partial Response	12	15%	7	37%	0	0%	3	20%	10	26%
No data	0	0%	1	5%	3	60%	1	7%	5	13%

Source: Company Data, 2011 ASCO Poster 3102

Safety and tolerability appears to be consistent with other PARP inhibitors. Niraparib is still early in development but the safety profile looks generally consistent with other PARP inhibitors. Nausea and vomiting, fatigue, anemia and anorexia are common for this class of agents and niraparib is no exception (Exhibit 56). Niraparib appears to have higher rates of bone marrow toxicity than other agents in the class but we will wait for more data before making firm conclusions on the clinical profile.

Exhibit 56

Phase I Single-Agent Safety Summary and Comparison to Other PARP Inhibitors

	Niraparib (N = 80)		Olaparib (N = 60)		Veliparib (N = 69)	
	Gr. 1-2	Gr. 3-4	Gr. 1-2	Gr. 3-4	Gr. 1-2	Gr. 3-4
Nausea	50%	3%	28%	3%	48%	1%
Fatigue	48%	5%	28%	2%	36%	1%
Constipation	34%	1%	0%	0%	0%	0%
Vomiting	33%	6%	18%	2%	19%	0%
Anemia	33%	9%	3%	2%	25%	3%
Anorexia	26%	1%	12%	0%	19%	0%
Diarrhea	19%	3%	5%	0%	0%	0%
Thrombocytopenia	19%	15%	0%	0%	9%	0%
Neutropenia	18%	4%	0%	0%	0%	0%
Abdominal pain	0%	0%	0%	0%	12%	1%
Dyspepsia	0%	0%	7%	0%	0%	0%
Dysgeusia	0%	0%	13%	0%	0%	0%
Dizziness	0%	0%	3%	2%	9%	0%
Stomatitis	0%	0%	5%	0%	0%	0%
Lymphopenia	0%	0%	0%	5%	10%	13%
Insomnia	0%	0%	0%	0%	12%	0%
Headache	0%	0%	0%	0%	12%	0%
Hyperglycemia	0%	0%	0%	0%	23%	4%

Source: ASCO 2011 poster 3102; N Engl J Med 361;2; ASCO 2012 poster 3054; Side effects with >10% incidence are highlighted in light red.

Two major issues have limited PARP inhibitor development to date, but we believe these are poised to be solved. PARP inhibitors have been in development for several years but the highest profile programs have spent over three years in the Phase II for two major reasons 1) formulation challenges and 2) and an unclear development path. However, we believe both issues are addressable based on the totality of the data to date so that PARP development path can finally start to mature from here. While the space remains crowded and risk remains, Tesaro's niraparib looks like a competitive agent meriting inclusion in our model.

Formulation has been a challenge for several players. AstraZeneca's olaparib (from the 2005 Kudos acquisition) was the leading PARP inhibitor in development and established much of the proof of concept for the field especially in BRCA mutation positive breast and ovarian cancer. However, the active 400 mg dose required 16 capsules per day and AstraZeneca paused development to optimize tablet formulation. Astra presented data for the tablet at ASCO 2012 and while the program is making progress, toxicity issues remain and our diligence suggests there is still work to be done on the dosing schedule before moving forward. Timelines remain unclear and if Astra pursues more Phase II work, Tesaro is on a competitive if not more advanced timeline. Similarly, the Cephalon/Teva program CEP-9722 appears to have formulation issues with significant PK variability in data at ASCO 2012.

The second issue has been the development path for PARP inhibitors agents. Broadly, clinical development of PARP inhibitors in oncology has followed three major paths 1) single agent approaches in women with BRCA1/2 mutations and 2) combinations with chemotherapy (chemosensitization) in multiple tumor types, and 3) maintenance therapy following chemotherapy especially in ovarian cancer.

Moreover, we suspect that enthusiasm for BRCA mutation monotherapy as a rapid path to market has been tempered by visibility into subsequent larger markets for pharma sponsors. In addition, the regulatory uncertainty surrounding the requirement for a significant overall survival benefit in ovarian cancer has contributed to the slow pace of development for the class. Finally, the high profile failure of Sanofi/BiPar iniparib in triple negative breast cancer likely contributed to developmental hesitancy as well, however it was later discovered that iniparib is not a true inhibitor of PARP.

However, we see several viable paths forward for niraparib. First, in the context of multiple recent approvals for targeted agents as monotherapy in defined genetic patient subsets including Pfizer's Xalkori, Roche's Zelboraf, and Roche's Erivedge, a PARP monotherapy approach in BRCA1/2 mutation positive late-line breast and ovarian cancer should be both rapid and viable. The scientific rationale is clear given 1) synthetic lethality hypothesis, 2) multiple PARP inhibitors showed monotherapy activity in this setting and niraparib's profile is consistent with the PARP class (Exhibit 57), and 3) the toxicity profile of these agents is manageable as monotherapy. Admittedly, there are still questions about trial design and patient population, but we do not see them as insurmountable.

Exhibit 57

Summary of PARP Inhibitor Monotherapy Activity in BRCA 1/2 Mutation Positive Ovarian & Breast Cancer Subjects

Response Type	Olaparib		Niraparib		Veliparib	
	N	%	N	%	N	%
Progressed	7	39%	7	39%	19	61%
Stable Disease	2	11%	4	22%	9	29%
Partial or Complete Response	9	50%	7	39%	3	10%

Source: Morgan Stanley Research; N Engl J Med 361;2; 2012 ASCO Poster 3054; 2011 ASCO Poster 3102.

We are intrigued by maintenance therapy approaches for PARP but Phase II work remains. Olaparib has shown clear evidence of activity in ovarian cancer maintenance therapy following combination with chemotherapy (Exhibit 58). Admittedly, the development path is long and uncertain but PARP inhibitors appear to have clear activity and niraparib's once daily dosing fits well with a maintenance paradigm.

Exhibit 58

Olaparib Maintenance Prolongs PFS but not OS

Trial 1	Olaparib (400 mg BID)	Placebo
	N = 136	N = 129
PFS	**8.4**	**4.8**
HR	0.35	
95% CI	(0.25, 0.49)	
p-value	<0.001	
OS	**29.7**	**29.9**
HR	0.94	
p-value	0.75	
95% CI	(0.63, 1.39)	

Trial 2	Olaparib (400 mg BID)	Placebo
	N = 66	N = 55
PFS	**12.2**	**9.6**
HR	0.51	
95% CI	(0.34, 0.77)	
p-value	0.0012	
OS	**NA**	**NA**

Source: N Engl J Med 2012; 366:1382-1392; ASCO 2012 Poster 5001; Morgan Stanley Research.

Chemosensitization approaches are interesting but toxicity issues remain and visibility is limited. PARP inhibitors could have broad applicability in combination with chemotherapy especially DNA damaging platinums or alkylating agents like temozolomide. The on-going Merck trials in combination with temozolomide in 2013 could be informative for Tesaro.

Tesaro faces a crowded development landscape. Tesaro will have to contend with multiple PARP inhibitors in development. However, given the apparent lack of formulation issues and evidence of activity from Phase I, we see niraparib as a competitive agent in this space in terms of activity and timelines. Moreover, the management team has experience in PARP inhibitor development as Eisai's E-7016 is of MGI provenance.

BioMarin's BMN-673 will get significant attention over the next 6-12 months with 2H12 Phase I data in solid tumors and 1Q13 for Phase I data in hematologic cancers. BioMarin foresees a Phase III trial starting in 2H13. Early single patient data suggest this is an active agent with CA-125 responses in BRCA1/2 mutation positive ovarian cancers with doses in the 25-400 microgram (µg) range suggesting a highly potent agent, and initial activity observed at 100 µg. However, without significant toxicity so far, which seems exceptional for this class, the company is now enrolling a 900 µg cohort and has yet to reach an MTD. We expect all potent agents will have bone marrow related on-target toxicities. **Abbott's veliparib** has languished in Phase I/II for some time across a range of tumors. The NCI is supporting a broad Phase II program and the commercial development plans are unclear. However, single agent activity has been underwhelming in the data to date, but this agent is worth watching. Finally, **Clovis' rucaparib** (formerly Pfizer's PF-01367338) showed early

signs of activity in an IV formulation, but data are limited on the oral formulation that is currently in several Phase I/II trials. We expect updates on this agent over the next two years.

Exhibit 59

Competitive Landscape for Oral PARP Inhibitors

Company	Drug	Stage	Tumor Type(s)	Clinical Efficacy Data	Next Data Readout
Astra Zeneca	Olaparib	Phase 1/2	Recurrent Ovarian Cancer	Best ORR 31%, Best PDS 8.8 Months	Mid-2012
Abbott	Veliparib	Phase 2	Solid Tumors & Blood Cancers	Partial RR 20%, SD Rate 17%	2H13
Cephalon / Teva	CEP-9722	Phase 2	Advanced Solid Tumors	PARP inhibiton seen	Mid-2013
Biomarin	BMN-763	Phase 1	Solid Tumors & Blood Cancers	NA	2H12 (Solid Tumors) 1Q13 (Blood Cancers)
Eisai	E-7016	Phase 1/2	Solid Tumors & Melanoma	NA	Mid-2012 (Solid Tumors) 1H14 (Melanoma)
Pfizer / Clovis	Rucaparib	Phase 1/2	Breast, Ovarian	NA	1H14
Tesaro	Niraparib	Phase 2	Possibly Breast, Ovarian, Gastric, and/or Lung	NA	2013 / 2014

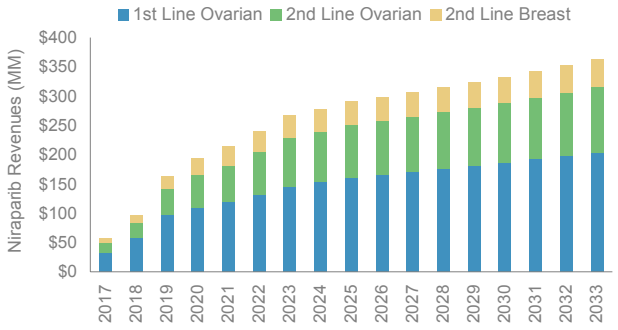
Source: Company Data, Morgan Stanley Research

What is in our model for niraparib? For the reasons discussed above, we include a risk-adjusted contribution from niraparib in our model. Briefly, we believe the Phase I data provide proof of concept in a validated class and timelines look competitive with the other agents in the development. At the same time niraparib's data are limited and competitive risks are still material.

Our model assumes that Tesaro is able to leverage a rapid path to market as monotherapy in first line and pre-treated (likely platinum insensitive) ovarian cancer and/or breast cancer. We assume Phase III in 2H13 with a launch in 2017. In total, peak sales reach $325 MM by 2033 (the final patent year) with the bulk of revenue coming from ovarian cancer (~85%) with a minority from breast cancer (~15%). Overall, we estimate niraparib contributes ~16% or $3 per share to our $19 base case valuation.

Exhibit 60

Niraparib Sales Projections (MSe)



Source: Company Data, Morgan Stanley Research estimates

Our major modeling assumptions for the BRAC mutation positive breast and ovarian cancer markets include: 1) 33% of all breast cancer patient are eligible for testing based on family history or other risk factors with a 9% positive test rate, and 2) in ovarian cancer, we assume 100% of patients are eligible for testing with a 18% positive test rate. Our model does not assume that a targeted agent loosens BRCA testing requirements to grow the market. Our model is risk adjusted for competition with <50% share to niraparib leaving room for competitive PARP inhibitors. As with nearly all targeted agents given as monotherapy, the duration of therapy is relative brief at ~9 months. Our initial pricing assumption in 2017 is $11,000 per month.

Exhibit 61

Niraparib Market Model in Breast & Ovarian Cancer

	2017	2018	2019	2020	2027	2028
Breast HER2+ 2nd Line	**26**	**26**	**27**	**27**	**29**	**29**
% eligible for BRACAnalysis	*33%*	*33%*	*33%*	*33%*	*33%*	*33%*
% mutant BRCA1/2	*9%*	*9%*	*9%*	*9%*	*9%*	*9%*
Addressable Market	**0.78**	**0.78**	**0.79**	**0.80**	**0.85**	**0.86**
% treated with Niriparib	*15%*	*20%*	*30%*	*35%*	*45%*	*45%*
2nd Line mBC revenue	**$8**	**$12**	**$21**	**$28**	**$41**	**$43**
Ovarian 1st Line	**19**	**19**	**19**	**19**	**21**	**21**
% eligible for BRACAnalysis	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*
% mutant BRCA1/2	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*
Addressable Market	**3.29**	**3.32**	**3.35**	**3.38**	**3.60**	**3.64**
% treated with Niriparib	*15%*	*20%*	*30%*	*33%*	*42%*	*42%*
First line ovarian revenue	**$33**	**$59**	**$97**	**$110**	**$171**	**$176**
Ovarian 2nd Line	**9**	**9**	**10**	**10**	**10**	**10**
% eligible for BRACAnalysis	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*
% mutant BRCA1/2	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*
Addressable Market	**1.69**	**1.71**	**1.72**	**1.74**	**1.85**	**1.87**
% treated with Niriparib	*15%*	*20%*	*30%*	*33%*	*45%*	*45%*
2nd Line ovarian revenue	**$17**	**$25**	**$44**	**$56**	**$94**	**$97**
Total Niraparib (MM)	**$57**	**$96**	**$162**	**$194**	**$306**	**$315**

Source: Company Data, Morgan Stanley Research estimates

Exhibit 62
Summary of Licensing Deal Terms

		Rolapitant	Niraparib	TSR-011 (ALK Inhibitor)
Upfront (MM)		$6	$7	$0.5 MM
All Milestones (MM)		$115	> $144.50	$138 MM
Development Milestones (MM)		$30	> $86.50	NA
First Indication (MM)		NA	$57	NA
Each Additional Indication (MM)		NA	$29.5	NA
Sales Milestones (MM)		$85	$87.50	NA
Royalties	US	Low teens to low twenties (effective range is low teens)	Low Teens	Mid-single digits to low double digits
	oUS & EU	Low double-digits		

Source: Company Data, Morgan Stanley Research

Exhibit 63

Income Statement

(Dollars in millions, except per share data)
Fiscal year ends Dec. 31

	2011A	1Q12A	2Q12E	3Q12E	4Q12E	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E
Rolapitant Revenue	$0	$0	$0	$0	$0	$0	$0	$10	$40	$124	$213	$314	$294	$310	$324	$326	$328	$329	$331	$333	$334	$336	$20
TSR-011 Revenue	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0
Niraparib Revenue	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$57	$96	$162	$194	$214	$240	$267	$279	$290	$298	$306	$315	$324
Other Revenue	$0	$0	$0	$0	$0	$0	$2.5	$10	$5	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0
Total Revenue	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$3**	**$20**	**$45**	**$124**	**$271**	**$410**	**$456**	**$504**	**$539**	**$566**	**$595**	**$608**	**$621**	**$631**	**$641**	**$651**	**$344**
% Growth y/y		*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*700.4%*	*125.4%*	*175.2%*	*118.0%*	*51.6%*	*11.3%*	*10.5%*	*6.8%*	*5.1%*	*5.1%*	*2.1%*	*2.2%*	*1.6%*	*1.6%*	*1.7%*	*-47.2%*
Cost of Sales																							
COGS	**$0**	0	0	0	0	**$0**	**$0**	**$2**	**$7**	**$21**	**$51**	**$81**	**$70**	**$77**	**$79**	**$82**	**$87**	**$89**	**$91**	**$93**	**$94**	**$96**	**$55**
% of revenue	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*17.0%*	*17.0%*	*17.0%*	*23.9%*	*25.7%*	*23.8%*	*25.0%*	*24.5%*	*25.3%*	*26.6%*	*27.0%*	*27.5%*	*27.8%*	*28.2%*	*28.5%*	*277.7%*
Gross Profit	**$0**	**0**	**0**	**0**	**0**	**$0**	**$3**	**$18**	**$38**	**$103**	**$219**	**$329**	**$386**	**$427**	**$459**	**$484**	**$508**	**$519**	**$530**	**$538**	**$547**	**$555**	**$289**
Gross Margin	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*91.5%*	*84.9%*	*83.0%*	*81.1%*	*80.3%*	*84.6%*	*84.6%*	*85.3%*	*85.4%*	*85.4%*	*85.4%*	*85.3%*	*85.3%*	*85.3%*	*85.3%*	*84.1%*
R&D	**$12**	8.2	15.3	16.0	19.8	**$59**	**$65**	**$70**	**$45**	**$43**	**$59**	**$27**	**$22**	**$22**	**$22**	**$22**	**$22**	**$22**	**$22**	**$22**	**$22**	**$22**	**$22**
% of revenue	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*2585%*	*350%*	*100%*	*35%*	*22%*	*7%*	*5%*	*4%*	*4%*	*4%*	*4%*	*4%*	*4%*	*3%*	*3%*	*3%*	*6%*
SG&A Total	**$3**	0	0	2	3	**$5**	**$10**	**$26**	**$42**	**$51**	**$85**	**$122**	**$132**	**$138**	**$134**	**$138**	**$106**	**$109**	**$91**	**$77**	**$78**	**$79**	**$67**
% of revenue	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*401%*	*130%*	*93%*	*41%*	*31%*	*30%*	*29%*	*27%*	*25%*	*24%*	*18%*	*18%*	*15%*	*12%*	*12%*	*12%*	*20%*
Sales & Marketing	$0	0	0	0	0	$0	$0	$15	$30	$38	$58	$81	$91	$92	$86	$87	$58	$67	$61	$52	$52	$53	$54
% of revenue	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*0%*	*75%*	*67%*	*31%*	*21%*	*20%*	*20%*	*18%*	*16%*	*15%*	*10%*	*11%*	*10%*	*8%*	*8%*	*8%*	*16%*
G&A	$3	0.0	0.0	2.2	2.5	$5	$10	$11	$12	$12	$27	$41	$41	$45	$48	$51	$48	$43	$30	$25	$26	$26	$14
% of revenue	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*401%*	*55.0%*	*26.6%*	*10.0%*	*10.0%*	*10.0%*	*9.0%*	*9.0%*	*9.0%*	*9.0%*	*8.0%*	*7.0%*	*4.8%*	*4.0%*	*4.0%*	*4.0%*	*12%*
Total Operating Expenses	**$15**	**8**	**15**	**18**	**22**	**$64**	**$75**	**$96**	**$87**	**$94**	**$144**	**$149**	**$154**	**$160**	**$156**	**$160**	**$128**	**$131**	**$113**	**$99**	**$100**	**$101**	**$89**
Operating Income (Loss)	**($15)**	**($8)**	**($15)**	**($18)**	**($22)**	**($64)**	**($72)**	**($78)**	**($49)**	**$10**	**$75**	**$181**	**$232**	**$267**	**$303**	**$324**	**$380**	**$387**	**$417**	**$439**	**$447**	**$454**	**$200**
Operating Margin	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*9.2%*	*34.3%*	*54.9%*	*60.0%*	*62.6%*	*66.0%*	*67.0%*	*74.9%*	*74.7%*	*78.7%*	*81.6%*	*81.7%*	*81.8%*	*69.1%*
Interest, Other Income	$0	0.02	0.10	0.36	0.31	$1	$1	$1	$0	$0	$0	$1	$2	$3	$3	$4	$6	$7	$8	$10	$11	$13	$14
Interest, Other Expense	$0	0	0	0	0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0
Other income (expense), net	($1)	0	0	0	0	$1	$1	$1	$0	$0	$0	$1	$2	$3	$3	$4	$6	$7	$8	$10	$11	$13	$14
Pretax Income (Loss)	**($16)**	**($8)**	**($15)**	**($18)**	**($22)**	**($63)**	**($71)**	**($77)**	**($48)**	**$10**	**$76**	**$181**	**$233**	**$270**	**$306**	**$329**	**$386**	**$394**	**$425**	**$449**	**$458**	**$467**	**$214**
Provision for Income Taxes	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$4	$27	$58	$94	$107	$115	$135	$138	$149	$157	$160	$163	$75
Effective Tax Rate	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*NM*	*5.0%*	*15.0%*	*25.0%*	*35.0%*	*35.0%*	*35.0%*	*35.0%*	*35.0%*	*35.0%*	*35.0%*	*35.0%*	*35.0%*	*35.0%*
Net Income	**($16.4)**	**($8.2)**	**($15.3)**	**($18.2)**	**($22.3)**	**($63.1)**	**($71.5)**	**($77.1)**	**($48.4)**	**$9.9**	**$71.9**	**$154.2**	**$175.1**	**$175.4**	**$199.1**	**$213.6**	**$250.9**	**$256.2**	**$276.2**	**$291.7**	**$297.4**	**$303.5**	**$138.9**
Net Income, Fully Taxed	**($16.4)**	**($8.2)**	**($15.3)**	**($18.2)**	**($22.3)**	**($63.1)**	**($71.5)**	**($77.1)**	**($48.4)**	**$9.9**	**$49.2**	**$117.9**	**$151.7**	**$175.4**	**$199.1**	**$213.6**	**$250.9**	**$256.2**	**$276.2**	**$291.7**	**$297.4**	**$303.5**	**$138.9**
Basic EPS					**($0.83)**	**($2.37)**	**($2.55)**	**($2.29)**	**($1.37)**	**$0.26**	**$1.29**	**$3.04**	**$3.83**	**$4.34**	**$4.83**	**$5.08**	**$5.85**	**$5.86**	**$6.19**	**$6.41**	**$6.41**	**$6.41**	**$2.88**
Diluted EPS					**($0.83)**	**($2.37)**	**($2.55)**	**($2.29)**	**($1.37)**	**$0.26**	**$1.80**	**$3.78**	**$4.21**	**$4.14**	**$4.60**	**$4.84**	**$5.57**	**$5.58**	**$5.90**	**$6.11**	**$6.10**	**$6.11**	**$2.74**
Diluted EPS, Fully Taxed					**($0.83)**	**($2.37)**	**($2.55)**	**($2.29)**	**($1.37)**	**$0.26**	**$1.23**	**$2.89**	**$3.65**	**$4.14**	**$4.60**	**$4.84**	**$5.57**	**$5.58**	**$5.90**	**$6.11**	**$6.10**	**$6.11**	**$2.74**

Source: Company Data, Morgan Stanley Research estimates

Exhibit 64

Balance Sheet

(Dollars in millions, except per share data)
Fiscal year ends Dec. 31

	2011A	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E
Assets																			
Cash & Marketable Securities	**$40**	**$121**	**$153**	**$81**	**$78**	**$69**	**$102**	**$231**	**$416**	**$586**	**$784**	**$999**	**$1,243**	**$1,500**	**$1,774**	**$2,064**	**$2,361**	**$2,665**	**$2,868**
Cash and cash equivalents	40	121	153	81	78	69	102	231	416	586	784	999	1,243	1,500	1,774	2,064	2,361	2,665	2,868
Marketable securities	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Inventory	0.0	0.0	0.0	0.7	3.6	9.9	16.2	20.5	22.8	25.2	24.2	22.6	23.8	24.3	24.8	25.2	25.6	26.1	13.8
Accounts receivable	0.0	0.0	0.2	1.4	4.5	12.4	27.1	36.9	41.1	45.4	48.5	51.0	53.5	54.7	55.9	56.8	57.7	58.6	30.9
Prepaid expenses and other current assets	2.6	3.0	4.0	4.5	4.5	11.2	18.9	24.6	27.4	30.3	32.3	34.0	35.7	36.5	37.3	37.8	38.4	39.1	20.6
Total current assets	**$42**	**$124**	**$158**	**$87**	**$90**	**$102**	**$164**	**$313**	**$507**	**$687**	**$889**	**$1,107**	**$1,356**	**$1,616**	**$1,892**	**$2,183**	**$2,483**	**$2,788**	**$2,933**
Restricted cash	0.2	0.2	0.2	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.0	0.0	0.0	0.0	0.0
Property and equipment, net	0.1	0.3	0.6	1.0	2.9	5.1	8.6	12.1	15.7	19.6	23.5	27.5	31.4	35.2	38.7	42.1	45.2	48.2	47.8
Other assets	0.1	0.2	0.3	0.4	0.4	1.0	1.9	2.9	3.2	3.5	3.8	4.0	4.2	4.3	4.3	4.4	4.5	4.6	2.4
Total assets	**$43**	**$124.6**	**$158.6**	**$88.9**	**$93.7**	**$108.6**	**$174.9**	**$327.9**	**$526.0**	**$710.0**	**$916.6**	**$1,138.5**	**$1,392.0**	**$1,655.3**	**$1,935.0**	**$2,229.9**	**$2,532.5**	**$2,841.2**	**$2,983.2**
Liabilities																			
Accounts payable	0.6	3.2	4.1	5.3	4.8	4.7	7.2	7.4	7.7	8.0	7.8	8.0	6.4	6.6	5.7	4.9	5.0	5.1	4.5
Accrued expenses	3.0	4.5	5.2	6.7	6.1	6.5	10.1	10.4	10.8	11.2	10.9	11.2	8.9	9.2	7.9	6.9	7.0	7.1	6.3
Loans payable, net of discount	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Deferred Taxes	0.0	0.0	0.0	0.0	0.0	0.0	3.8	31.0	68.9	68.9	68.9	68.9	68.9	68.9	68.9	68.9	68.9	68.9	68.9
Deferred revenue	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Deferred rent & other liabilities	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total current liabilities	**$4**	**$8**	**$9**	**$12**	**$11**	**$11**	**$21**	**$49**	**$87**	**$88**	**$88**	**$88**	**$84**	**$85**	**$82**	**$81**	**$81**	**$81**	**$80**
Loans payable, less current portion and discount	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Deferred revenue, net of current portion	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Deferred rent, net of current portion	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other long-term liabilities	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Warrants to purchase preferred stock	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total liabilities	**$4**	**$8**	**$9**	**$12**	**$11**	**$11**	**$21**	**$49**	**$87**	**$88**	**$88**	**$88**	**$84**	**$85**	**$82**	**$81**	**$81**	**$81**	**$80**
Preferred stock	64.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Stockholders' equity																			
Common stock	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Additional paid-in capital	0.3	205.4	309.1	313.9	368.3	373.0	380.2	387.6	395.3	403.3	411.1	419.1	425.5	432.1	437.7	442.7	447.7	452.7	457.2
Accumulated deficit / Retained Earnings	(25.4)	(88.5)	(159.9)	(237.0)	(285.5)	(275.6)	(226.4)	(108.5)	43.2	218.7	417.8	631.4	882.3	1,138.6	1,414.8	1,706.5	2,003.9	2,307.5	2,446.4
Accumulated other comprehensive income	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total stockholders' equity	**($25)**	**$117**	**$149**	**$77**	**$83**	**$97**	**$154**	**$279**	**$439**	**$622**	**$829**	**$1,051**	**$1,308**	**$1,571**	**$1,853**	**$2,149**	**$2,452**	**$2,760**	**$2,904**
Total liabilities and stockholder's equity	**$43**	**$125**	**$159**	**$89**	**$94**	**$109**	**$175**	**$328**	**$526**	**$710**	**$917**	**$1,139**	**$1,392**	**$1,655**	**$1,935**	**$2,230**	**$2,532**	**$2,841**	**$2,983**

Source: Company Data, Morgan Stanley Research estimates

Exhibit 65

Cash Flow Statement

(Dollars in millions, except per share data)
Fiscal year ends Dec. 31

	2011A	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E
CASH FLOWS FROM OPERATING ACTIVITIES:																			
Net (loss) Income	**($16.4)**	**($63.1)**	**($71.5)**	**($77.1)**	**($48.4)**	**$9.9**	**$49.2**	**$117.9**	**$151.7**	**$175.4**	**$199.1**	**$213.6**	**$250.9**	**$256.2**	**$276.2**	**$291.7**	**$297.4**	**$303.5**	**$138.9**
Depreciation and amortization	0.0	0.0	0.0	0.1	0.2	0.3	0.5	0.7	0.9	1.2	1.4	1.7	2.0	2.3	2.6	2.9	3.3	3.6	3.8
Stock-based compensation expense	0.3	1.3	3.7	4.8	4.4	4.7	7.2	7.4	7.7	8.0	7.8	8.0	6.4	6.6	5.7	4.9	5.0	5.1	4.5
Non-cash interest expense	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Loss on disposal of PPE	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Acquired in-process research and development	0.5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Increase in fair value of investor rights obligation	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tax Credit from NOL's	0.0	0.0	0.0	0.0	0.0	0.0	3.8	27.2	37.9	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Changes in working capital:	**0**	**4**	**0**	**0**	**(7)**	**(21)**	**(24)**	**(20)**	**(9)**	**(9)**	**(5)**	**(2)**	**(10)**	**(2)**	**(5)**	**(4)**	**(2)**	**(2)**	**59**
Accounts receivable	0.0	0.0	(0.2)	(1.2)	(3.1)	(7.9)	(14.6)	(9.9)	(4.2)	(4.3)	(3.1)	(2.5)	(2.6)	(1.2)	(1.2)	(0.9)	(0.9)	(1.0)	27.7
Prepaid expenses and other current assets	(2.7)	(0.4)	(1.0)	(0.5)	(0.0)	(6.7)	(7.8)	(5.7)	(2.8)	(2.9)	(2.1)	(1.7)	(1.7)	(0.8)	(0.8)	(0.6)	(0.6)	(0.6)	18.4
Other noncurrent assets	0.0	(0.1)	(0.1)	(0.1)	0.1	(0.6)	(0.9)	(1.0)	(0.3)	(0.3)	(0.2)	(0.2)	(0.2)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	2.2
Restricted cash	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Accounts payable	0.5	2.6	0.9	1.2	(0.5)	(0.1)	2.5	0.2	0.3	0.3	(0.2)	0.2	(1.6)	0.2	(0.9)	(0.7)	0.1	0.1	(0.6)
Accrued expenses	2.6	1.5	0.8	1.5	(0.6)	0.4	3.5	0.3	0.4	0.4	(0.2)	0.2	(2.2)	0.3	(1.3)	(1.0)	0.1	0.1	(0.8)
Deferred rent & other liabilities	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Deferred revenue	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Inventory	0.0	0.0	0.0	(0.7)	(2.9)	(6.3)	(6.3)	(4.3)	(2.3)	(2.4)	1.0	1.6	(1.2)	(0.5)	(0.5)	(0.4)	(0.4)	(0.4)	12.3
Net cash used in operating activities	**($14)**	**($58)**	**($67)**	**($72)**	**($51)**	**($6)**	**$37**	**$133**	**$189**	**$175**	**$204**	**$221**	**$250**	**$263**	**$280**	**$296**	**$304**	**$310**	**$206**
CASH FLOWS FROM INVESTING ACTIVITIES																			
Purchases of PPE	(0.1)	(0.2)	(0.3)	(0.5)	(2.0)	(2.5)	(4.1)	(4.1)	(4.6)	(5.0)	(5.4)	(5.7)	(5.9)	(6.1)	(6.2)	(6.3)	(6.4)	(6.5)	(3.4)
Acquisition of ALK license	(0.5)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Restricted Cash	(0.1)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Purchases of marketable securities	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Proceeds from maturities and sales of marketable securities	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Net cash used in investing activities	**($0.7)**	**($0.2)**	**($0.3)**	**($0.5)**	**($2.0)**	**($2.5)**	**($4.1)**	**($4.1)**	**($4.6)**	**($5.0)**	**($5.4)**	**($5.7)**	**($5.9)**	**($6.1)**	**($6.2)**	**($6.3)**	**($6.4)**	**($6.5)**	**($3.4)**
CASH FLOWS FROM FINANCING ACTIVITIES																			
Proceeds from issuance of convertible preferred stock, net of issuan	52.1	58.5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Proceeds from exercise of stock options and issuance of common an	0.0	81.0	100.0	0.0	50.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tax benefit from stock options	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Disbursements from repurchase of common stock	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Proceeds from issuance of loans payable	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Principal payments on loans payable	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Net cash provided by financing activities	**$52.1**	**$139.5**	**$100.0**	**$0.0**	**$50.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**
Increase in cash and cash equivalents	**37.3**	**81.1**	**32.4**	**(72.6)**	**(3.0)**	**(8.8)**	**33.1**	**128.9**	**184.8**	**170.2**	**198.2**	**215.3**	**243.9**	**257.0**	**273.5**	**289.7**	**297.5**	**303.7**	**202.9**
Restatement	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**
Cash and equivalents at beginning of year	2.5	39.8	120.9	153.3	80.8	77.7	68.9	102.0	230.9	415.7	585.9	784.1	999.4	1,243.3	1,500.3	1,773.8	2,063.5	2,361.0	2,664.7
Cash and equivalents at end of year	**$39.8**	**$120.9**	**$153.3**	**$80.8**	**$77.7**	**$68.9**	**$102.0**	**$230.9**	**$415.7**	**$585.9**	**$784.1**	**$999.4**	**$1,243.3**	**$1,500.3**	**$1,773.8**	**$2,063.5**	**$2,361.0**	**$2,664.7**	**$2,867.6**

Source: Company Data, Morgan Stanley Research estimates

MORGAN STANLEY ModelWare	**Morgan Stanley ModelWare is a proprietary analytic framework that helps clients uncover value, adjusting for distortions and ambiguities created by local accounting regulations.** For example, ModelWare EPS adjusts for one-time events, capitalizes operating leases (where their use is significant), and converts inventory from LIFO costing to a FIFO basis. ModelWare also emphasizes the separation of operating performance of a company from its financing for a more complete view of how a company generates earnings.

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(as of June 30, 2012)

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	Coverage Universe		Investment Banking Clients (IBC)		
Stock Rating Category	Count	% of Total	Count	% of Total IBC	% of Rating Category
Overweight/Buy	**1139**	**39%**	**474**	**43%**	**42%**
Equal-weight/Hold	**1252**	**42%**	**478**	**43%**	**38%**
Not-Rated/Hold	**108**	**4%**	**33**	**3%**	**31%**
Underweight/Sell	**458**	**15%**	**119**	**11%**	**26%**
Total	**2,957**		**1104**		

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Industry Coverage:Biotechnology

Company (Ticker)	Rating (as of)	Price* (07/20/2012)
David Friedman, M.D.		
AMAG Pharmaceuticals, Inc. (AMAG.O)	E (11/21/2011)	$15.36
Alexion Pharmaceuticals (ALXN.O)	O (09/07/2010)	$99.35
Amylin Pharmaceuticals (AMLN.O)	E (07/02/2012)	$30.79
Auxilium Pharmaceuticals (AUXL.O)	E (03/18/2011)	$26.19
Idenix Pharmaceuticals, Inc. (IDIX.O)	E (03/18/2011)	$10.35
Incyte Corporation (INCY.O)	E (02/16/2012)	$24.55
InterMune (ITMN.O)	E (09/07/2010)	$11.71
Ironwood Pharmaceuticals, Inc. (IRWD.O)	O (03/18/2011)	$13.17
Lexicon Pharmaceuticals, Inc. (LXRX.O)	E (09/07/2010)	$2.71
Synageva Biopharma Corp (GEVA.O)	O (04/20/2012)	$49.62
Theravance Inc (THRX.O)	U (01/31/2012)	$29.97
Vertex Pharmaceuticals (VRTX.O)	E (05/08/2012)	$51.27
XenoPort Inc (XNPT.O)	E (08/26/2011)	$6.24
Marshall Urist, M.D., Ph.D.		
Tesaro Inc. (TSRO.O)	O (07/23/2012)	$13.5
Amgen Inc. (AMGN.O)	E (02/09/2012)	$77.77
Aveo Pharmaceuticals (AVEO.O)	E (02/09/2012)	$13.21
Biogen Idec Inc. (BIIB.O)	O (02/09/2012)	$142.21
Celgene Corp (CELG.O)	O (02/09/2012)	$66.42
Elan Corporation PLC (ELN.N)	E (07/09/2012)	$13.72
Gilead Sciences Inc. (GILD.O)	O (02/09/2012)	$53.08
Hospira (HSP.N)	E (07/16/2009)	$35.45
Onyx Pharmaceuticals Inc. (ONXX.O)	E (06/21/2012)	$76.38
Sagent Pharmaceuticals Inc (SGNT.O)	O (05/31/2011)	$19.18

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