March 21, 2013

Tesaro

(TSRO-NASDAQ)

Stock Rating: **Outperform**
Industry Rating: **Outperform**

Biotechnology

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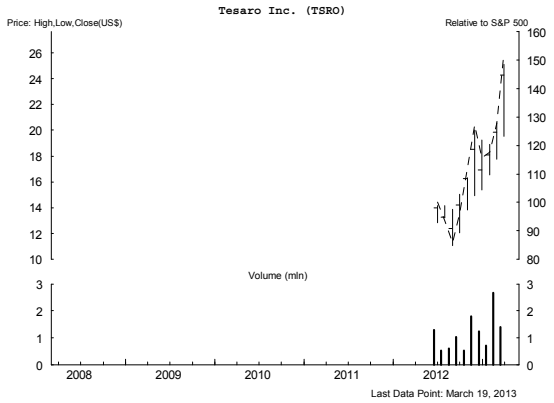
Considering Upside Opportunity from PARP Inhibitor Niraparib

Securities Info

Price (20-Mar)	**$23.24**	**Target Price**	**$36**
52-Wk High/Low	$25/$11	Dividend	--
Mkt Cap (mm)	$740	Yield	--
Shs O/S (mm, BASIC)	31.9	Float O/S (mm)	13.2
Options O/S (mm)	na	ADVol (30-day, 000s)	131

Price Performance



Event

Tesaro (TSRO) recently announced plans to initiate, by mid-2013, a phase 3 trial for PARP inhibitor niraparib as second-line maintenance therapy in patients with platinum-sensitive, serous ovarian cancer. The phase 3 trial is designed to randomize 360 patients 2:1 to niraparib maintenance versus placebo maintenance with a 180-patient BRCA+ cohort as well as a 180-patient BRCA- cohort being studied. The study design follows closely the design of a randomized phase 2 study for competing PARP inhibitor olaparib, which was published recently in *the New England Journal of Medicine* (NEJM) and demonstrated a statistically significant progression free survival (PFS) benefit.

Impact

We are reiterating our Outperform rating on shares of TSRO and increasing our price target to $36 in advance of phase 3 initiation for niraparib. Based on precedent data from olaparib where the hazard ratio for PFS was reduced by 65% (HR=0.35) in unselected relapsed/platinum-sensitive patients and by 90% (HR=0.10) in BRCA+ patients, we believe that the likelihood of success is very high for niraparib given similarities in activity. With olaparib requiring reformulation and closest competitor BMN-673 initiating phase 3 in 2H13, we expect niraparib to be first to market in a category with a $1.5B+ opportunity in the US alone for ovarian cancer maintenance therapy. We estimate peak US sales for niraparib of ~$730M with a probability-adjusted NPV of ~$14/share, assuming a 50% likelihood, and thus see significant upside potential on potential phase 3 success.

Forecasts

Our 2013 forecast is for a loss per share of $2.35.

Valuation

Our $36 target is based on 20x our 2017E EPS of $3.35 discounted 25%.

Recommendation

We rate Tesaro Outperform.

Valuation/Financial Data

(FY-Dec.)	2011A	2012A	2013E	2014E
EPS GAAP	-$31.90	-$4.51	-$2.35	-$2.57
P/E			nm	nm
First Call Cons.			*-$2.78*	*-$2.38*
FCF	-$14.24	-$47.73	-$66.00	na
P/FCF			nm	na
EBITDA ($mm)	-$15	-$62	-$65	-$81
EV/EBITDA			nm	nm
Rev. ($mm)	$0	$0	$0	na
EV/Rev			na	na

Quarterly EPS	**1Q**	**2Q**	**3Q**	**4Q**
2012A	-$0.70	-$21.31	-$0.52	-$0.70
2013E	-$0.53	-$0.58	-$0.60	-$0.65

Balance Sheet Data (31-Dec)

Net Debt ($mm)	-$125	TotalDebt/EBITDA	nm
Total Debt ($mm)	$0	EBITDA/IntExp	na
Net Debt/Cap.	nm	Price/Book	5.5x

Notes: Quarterly EPS may not sum due to share count. All values in US$.

Source: BMO Capital Markets estimates, Bloomberg, Thomson Reuters, and IHS Global Insight.

Changes

Target
$22.00 to $36.00

Please refer to pages 12 to 16 for Important Disclosures, including the Analyst's Certification.

Overview

Tesaro (TSRO) is a development stage biopharmaceutical company focused on novel therapies for patients with cancer. With three separate drug candidates under development, most focus has been on the most advanced program for NK-1 antagonist rolapitant for the prevention of chemotherapy-induced nausea and vomiting (CINV) in patients receiving highly emetogenic chemotherapy (HEC) or moderately emetogenic chemotherapy (MEC) and with phase 3 data expected in 2H13. With the recent initiation of a phase 3 program for the company's PARP inhibitor niraparib in patients with platinum-sensitive, serous ovarian cancer, we believe, however, that review of precedent data and consideration of the phase 3 design suggest significant incremental value that is not being recognized at present.

Review of Phase 3 Trial Design for PARP Inhibitor Niraparib

- TSRO has indicated that it will initiate enrollment in its niraparib phase 3 trial in patients with high-grade serous ovarian cancer by mid-2013.
- The phase 3 trial is designed to enroll ~360 women with advanced high-grade serous ovarian cancer, divided into two cohorts, germ-line BRCA+ and non-germ-line BRCA+.
- Within each phase 3 trial cohort patients with be randomized 2:1 to either niraparib or placebo (120 niraparib to 60 placebo) following second-line progression on a platinum-based therapy.
- Patients enrolled in the study will be defined as platinum sensitive, based on prior first-line treatment response to a platinum containing regimen lasting at least six months.
- Patients in the study will be stratified by depth of prior response (complete response vs. partial response) and by whether prior response was 6-12 months duration or > 12 months duration.
- The primary endpoint of the phase 3 trial across both cohorts of the study is progression free survival (PFS).
- Each cohort of the phase 3 has 90% power to detect a hazard ratio of 0.50 with $p<0.05$ assuming a median PFS of 4.5 months in the control group. In addition, either study could yield a statistically significant result with a hazard ratio of 0.70.
- Powering assumptions for the niraparib phase 3 trial in advanced high-grade serous ovarian cancer were based on phase 2 results published in the *New England Journal of Medicine* (NEJM) by Ledermann for competing PARP inhibitor olaparib.

Review of Precedent Data for Other PARP Inhibitors

- Precedent data for a PARP inhibitor as maintenance therapy following second-line platinum response in platinum-sensitive, high-grade serous ovarian cancer comes from a randomized phase 2 study of olaparib published in the *New England Journal of Medicine* (NEJM) in April 2012.
- The randomized phase 2 study enrolled 265 patients with platinum-sensitive, relapsed, high-grade serous ovarian cancer who had received 2 or more platinum based regimens with partial response (PR) or complete response (CR) to their most recent platinum regimen.
- Patients in the phase 2 study were randomized 1:1 to either olaparib 400mg or placebo and were dosed to progression as defined by RECIST criteria. In total 136 patients were randomized to olaparib and 129 were randomized to placebo.
- The olaparib phase 2 study was designed with 80% power to detect a 33% improvement in the primary endpoint of PFS at a hazard ratio=0.75 and with $p<0.20$.
- With respect to the primary endpoint of PFS, maintenance therapy with olaparib improved median PFS to 8.4 months versus 4.8 months with placebo and with a hazard ratio =0.35 ($p<0.001$, confidence interval, 0.25 to 0.49).
- Sub-group analysis demonstrated that the benefit of olaparib on PFS was maintained in all key sub-groups including patients with BRCA mutation and those with unknown status, older and younger patients, those with non-Jewish ancestry, those with prior CR or PR and those with platinum sensitivity of 6-12 months or >12 months.

Considering Commercial Opportunity for Niraparib in Ovarian Cancer

- We estimate peak sales for niraparib as second-line maintenance therapy in serous ovarian cancer of ~$740 million.
- Our peak sales estimate assumes 30% penetration of the overall second-line platinum sensitive market, and higher penetration of 50% of the BRCA+ second-line market.
- We estimate the size of the incident population of second-line, platinum-sensitive ovarian cancer patients at ~19,000 patients per year, and the BRCA1/2 sub-population at ~4,700 patients per year.
- Our peak sales estimates assume eight months of dosing at $10,000/month in second-line platinum sensitive patients and 12 months of dosing at $10,000/month in the subset of patients that are BRCA+.
- With estimates launch in 2017 and peak sales reached in 2020 we estimate an incremental probability adjusted NPV of $14/share.

Primer on PARP Inhibition and Niraparib

Niraparib was developed by Merck (MRK) where the product was known as MK-4827. It is an orally active PARP 1/2 inhibitor with IC_{50} for PARP1/2 ranging from 2-4nM versus >100nM potency against other PARP family members. Pre-clinical data show that niraparib has activity in BRCA deficient tumors with 50% cell kill (CC_{50}) concentrations ranging from 10-90nM. Niraparib activity is specific for BRCA deficient tumors as the CC_{50} for BRCA proficient tumors is >1500nM. Pre-clinical data also show that niraparib is somewhat effective in tumors that are BRCA proficient but have defects in other parts of the homologous recombination DNA repair pathway, displaying a so-called BRCA-ness phenotype. Niraparib CC_{50} concentrations range from 100nM to 800nM for tumor cell lines deficient in DNA repair enzymes such as PTEN or ATM.

Phase 1 data for daily oral niraparib were reported at the 2010 American Society of Clinical Oncology (ASCO) meeting. The phase 1 trial was enriched for patients with cancers likely to have homologous recombination (HR) DNA repair defects including patients with known BRCA mutations as well as tumors with a high likelihood of sporadic HR repair defects such as high grade serous ovarian tumors. The phase 1 trial evaluated once daily oral doses of niraparib ranging from 30mg to 400mg in 60 patients and was followed by dose expansion cohorts (300mg/day) in patients with platinum resistant ovarian cancer and castrate resistant prostate cancer (CRPC).

In the breast cancer dose escalation phase, 12 partial responses (PRs) were observed for an overall response rate (ORR) of 20% with the majority of activity (n=10) restricted to patients with BRCA mutations. An additional 24 patients showed prolonged stable disease (SD) including both BRCA and non-BRCA mutation carriers. Of the 39 evaluable patients with ovarian cancer, a 46% clinical benefit rate (CBR) was observed, increasing to 58% in patients with a BRCA mutations

Niraparib was well tolerated with grade 1 constitutional symptoms commonly observed including nausea, vomiting, diarrhea, fatigue and anorexia in 20%-45% of patients. Grade 3 non-hematologic events however were rare. Grade 3 hematologic toxicity was observed in the single digit range and only for thrombocytopenia were grade 4 events observed (6%). Thrombocytopenia was deemed dose limiting, with niraparib maximal tolerated dose (MTD) established at 300mg/day.

An analysis of niraparib pharmacokinetics (PK) show an accumulation of product over 21 days after which a steady state is observed with a terminal half life ranging from 37-42 hours. Pharmacodynamic (PD) studies of PARP activity in peripheral blood mononuclear cells (PBMC) suggest that at doses of 80mg/day and above significant PARP activity is inhibited. Accumulation of double strand breaks in DNA from pre and post niraparib tumor samples was observed using gamma H2AX, a member of the histone H2A family of proteins as a pharmacodynamic (PD) marker. A significant up-regulation of H2AX in tumor samples from a BRCA breast cancer patient suggests that niraparib inhibits homologous recombination DNA repair.

DNA Repair Primer

All cells are at risk for genetic instability, leading to DNA damage which is manifest in the production of non-functional proteins. Such proteins can have a deleterious effect on the control of cellular metabolism. While genetic instability can result randomly, exposure of cells to damaging agents such as excessive sunlight or genotoxic chemicals for example can lead to an increased rate of DNA damage. Perhaps not unsurprisingly, several detection and signaling pathways are activated by damaged DNA and initiate a cascade of pathways to repair damaged DNA. The two most common pathways lead to cell cycle arrest, to allow for DNA repair to occur or programmed cell death known as apoptosis.

Since one outcome of damaged DNA is to turn a normal cell into a cancer cell, the genes responsible for repairing damaged DNA can be considered to play a role in tumor suppression. Consequently, mutations in a gene involved in DNA repair would be expected to increase the capability of the cell to become cancer promoting. As noted earlier there are several DNA repair pathways and each pathway is specific for a particular type of DNA damage. Following is a brief list:

- Non-homologous end joining – repair double strand breaks and cross-linked DNA
- Homologous recombination – as above
- Base excision or single strand break repair – removes incorrect bases and repair
- Nucleotide excision repair – repair missing bases
- Mismatch repair – detects and repairs base mismatches, insertions and deletions

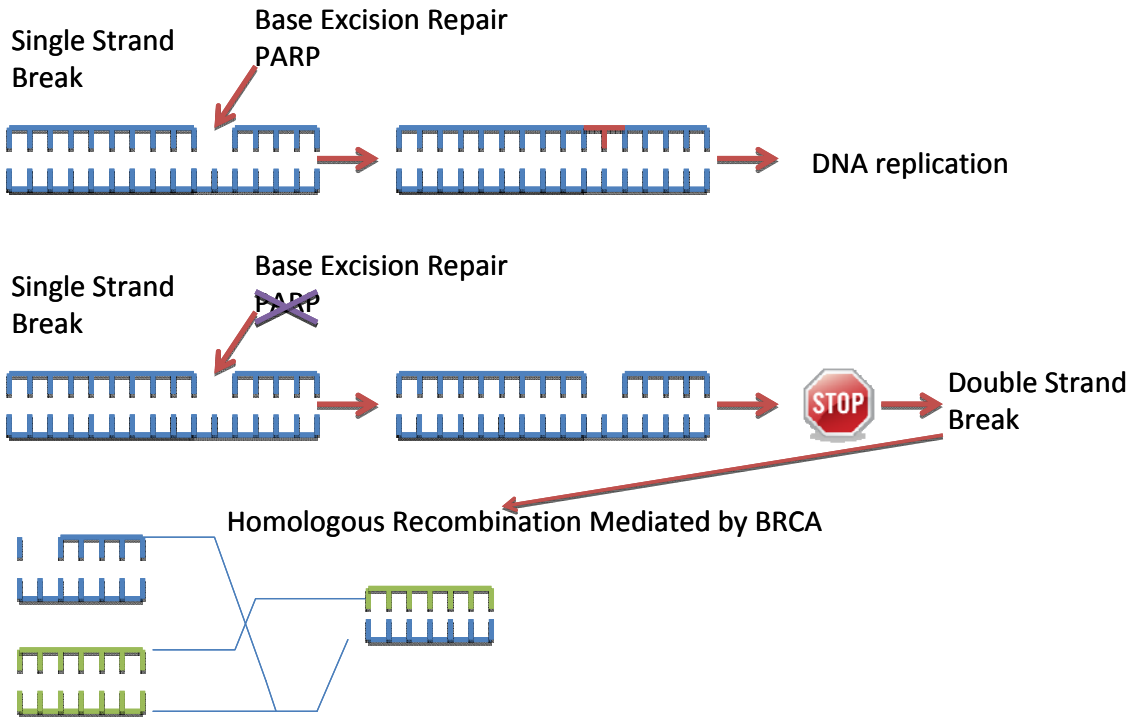
In cancer cells that have mutations to one or more components of a DNA repair pathway, cells can become overly reliant on the remaining pathway(s). This raises the possibility that targeting the dominant pathway could lead to cell death.

Targeting Base Excision and Single Strand Break

The best-known DNA repair genes associated with cancer are BRCA1 and BRCA2, which are involved in the repair of double-strand DNA breaks. Cells with germ line mutations in the BRCA genes become overly reliant on the base excision and single strand break repair mechanisms and thus targeting genes involved in this pathway becomes a viable strategy to target tumors with BRCA mutations. A critical enzyme in base excision or single strand break repair is poly (ADP-ribose) polymerase or PARP. At least 16 members of the PARP family have been identified, but only PARP-1 and PARP-2 appear to be involved in DNA repair.

In normal cells PARP-1 activity is low but is upregulated in response to DNA damage. PARP-1 over expression has also been documented in several cancers. PARP-1 is not essential to maintain cell viability as knockout mice are normal; however, knocking out PARP-1 and PARP-2 leads to embryonic lethality.

Exhibit 1: Cartoon of DNA Repair and the Role of PARP



Source: BMO Capital Markets

Clinical Development – The Concept of Synthetic Lethality

In classical cancer drug development, single agent activity is required to proceed from small proof-of-concept studies to a larger definitive one; however, because there are many pathways involved in DNA repair, it is unlikely that inhibiting a single pathway will lead to cell death. In the case of drugs targeting DNA repair pathways therefore, patients need to be selected that have an existing DNA repair defect, where the use of a drug to inhibit a second pathway may lead to cell death. The concept that two independent mechanisms alone do not affect cell viability but combined lead to cell death is referred to as synthetic lethality.

PARP inhibitors have been developed in three distinct areas:

- In patients with known non-base excision or single strand break repair DNA repair mutations
- With DNA damaging agents, where PARP inhibition serves to sensitize the tumor to a palatinate, for example.
- With radiation therapy where PARP also serves as a sensitizing agent.

A number of PARP inhibitors have entered the clinic and are at different stages of clinical development as noted in Exhibit 14.

Exhibit 2: PARP Inhibitor Development Summary

Sponsor	Drug	Phase
SNY	Iniparib	3
AZN	Olaparib	2
ABT	Velaparib	2
CLO*	Rucaparib	2
BMRN	BMN673	2
TEVA	CEP-9722	1
Eisai	E7016	1
Tesaro	Niraparib	3

Sources: clinicaltrials.gov and BMO Capital Markets Research; Licensed from Pfizer.

Sanofi's iniparib reached phase 3 testing following a successful phase 2 trial in patients with triple negative breast cancer (TNBC) that has been reported in the *New England Journal of Medicine* (NEJM), O'Shaughnessy et al., 20:364(3):205-14. While TNBC patients were not selected on the basis of a BRCA mutation, their physiology is considered to be BRCA-like based on the genetic profile of TNBC. However, the 520-patient phase 3 trial failed to confirm the benefit for iniparib shown in phase 2. Sanofi (SNY) has concluded that while iniparib does have effects on DNA repair, it does not have the activity profile of a typical PARP inhibitor.

This can be explained in part because iniparib targets the zinc finger domain of PARP rather than compete with NAD for the PARP catalytic site as is the case for all other PARP inhibitors in clinical development. While Sanofi is completing a phase 3 trial in NSCLC the development of iniparib will not be a template for other PARP inhibitors.

Behind Sanofi are several earlier stage inhibitors but for the most part published peer reviewed data are largely limited to AZN's olaparib. In comparison to iniparib, AstraZeneca has taken a more biomarker drive approach to developing the PARP inhibitor olaparib. In early phase 1 testing AZN enrolled 60 patients with advanced disease for a multiple ascending dose (MAD) trial at olaparib doses, ranging from 10mg/day to 600mg bid dosed 14 of 21 days. A dose of 200mg bid was selected for further evaluation in a cohort of 23 patients with BRCA mutations, comprising mostly breast, ovarian and pancreatic tumors. A partial response (PR) was observed in nine patients for a response rate of 39%. Following phase 1 testing, two multicenter randomized phase 2 trials were conducted in chemotherapy-refractory breast or ovarian cancer patients with BRCA mutations. Two doses were evaluated, including 400mg bid in the first cohort and 100mg bid in a second cohort. As noted in Exhibit 15, breast cancer patients receiving the higher dose had a greater chance of achieving a response (41% vs. 22%) and benefited from a longer time to tumor progression – 5.7 months versus 3.8 months. The ovarian cancer patients showed a similar dose response.

Exhibit 3: Phase 2 Olaparib Data in BRAC Mutation +ve Patients

	Breast		Ovarian
	ORR	TTP	ORR
400mg BID	41%	5.7 mo.	33%
100mg BID	22%	3.8 mo.	12.50%

Sources: Fong et al, JCO, 2010 and BMO Capital Markets Research.

Fong and colleagues published additional data from the ovarian cancer dose ranging expansion cohort in the *Journal of Clinical Oncology* (JCO), 2010, 28: 15, 2512-19, showing that responses to olaparib were higher in platinum-sensitive patients (61.5%) versus platinum-resistant patients (15.4%).

Additional evidence of olaparib activity in high-grade serous ovarian cancer was reported at the 2010 ASCO meeting by Gelmon and colleagues from a phase 1 Canadian trial. Of the 64 patients enrolled, ten patients were known BRCA mutation carriers and an overall response rate (ORR) of 40% was observed compared to 26% in the non-BRCA cohort.

In addition to monotherapy trials, data have been reported for a trial combining pegylated doxorubicin (PLD) with olaparib in patients with less than a 12-month PFS following platinate front-line therapy. While PLD had not been studied in BRCA+ve ovarian cancer the investigators assumed its activity would be similar to that observed in a general population, that is to say an ORR of ~ 20% and PFS of ~ four months.

In the Kaye trial, patients were randomized to PLD (n=33), PLD + olaparib 400mg BID (n=32) or olaparib 200mg BID (n=32). Overall, the treatments were well tolerated with one to three patients across the three arms discontinuing therapy owing to an adverse event (AE). As shown in Exhibit 16, an overall response rate (ORR) of ~ 20% was observed in the PLD arm increasing to 25%-30% in the olaparib arms; these increases did not achieve statistical significance.

Exhibit 4: Phase 2 Pegylated Doxorubicin /- Olaparib Data in BRAC Mutation +ve Ovarian Cancer

		Olaparib	
	PLD	PLD + 200mg bid	PLD + 400mg BID
ORR	18%	25%	31%
DoR - mo.	6	6.8	5.5
Deaths - n	13	9	11
%Δ ↓in target lesion size -%	14.3%	15.9%	24.6%
Patient with new lesions - %	45.5%	28.1%	34.4%

Sources: Kaye et al, JCO 2012 and BMO Capital Markets Research.

With 11 months of follow-up reported in the JCO manuscript deaths favored olaparib arms numerically, but perhaps more convincing of efficacy was a larger benefit in target lesion size reduction and a concomitant reduction in development of new lesions both favoring olaparib. But with ORRs limited to 20%-40%, clinical data suggest that olaparib has activity in an as yet to be defined subset of BRCA mutation +ve ovarian cancer patients. Further evidence for the subset benefit comes from patients crossing over from PLD to olaparib where 8/25 received long-term olaparib treatment.

In the Kaye, et al. trial, any safety and tolerability effects of olaparib 200mg/day or 400mg/day over and above those of PLD were hard to distinguish and thus it is not surprising that olaparib has been evaluated as maintenance therapy. At the recent ASCO meeting, Oza and colleagues presented data evaluating olaparib (400mg bid) or observation following carbo/taxol + olaparib (200mg bid) induction in patients with platinum-sensitive recurrent serous ovarian cancer. The trial enrolled 156 patients, 121 of whom entered a maintenance phase of observation or olaparib. A statistically significant increase in PFS was observed in favor of patients receiving olaparib maintenance therapy (12.2 vs. 9.6 months; p=0.0012). During maintenance, nausea and vomiting were more commonly observed in the olaparib arms at 50% vs. 6% and 29% vs. 7% respectively with 8% vs. 0% discontinuing owing to an adverse event (AE).

As reviewed earlier, Ledermann and colleagues published data for olaparib (400mg BID) maintenance in serous ovarian cancer patients with platinum sensitive disease, NEJM; 366: 1382-92. As mentioned previously, in this trial, 265 patients achieving a partial or complete remission in the second- or third-line setting were randomized to olaparib (n=136) or placebo (n=129) maintenance therapy. The trial was designed to show a 33% increase in PFS from 9-12

months. While this trial did not restrict enrollment to patients with BRCA mutations, it is estimated that about 50% of high-grade serous tumors have deficiencies in DNA repair, specifically homologous recombination due to mutations of BRCA, epigenetic silencing of BRCA or dysfunction in BRCA-related genes – so called BRCA-ness. Baseline demographics showed that of the 100 or so patients for which BRCA status was known about 60% had a BRCA mutation.

Following 153 progression events, the median PFS for the placebo group was 4.8 months increasing to 8.4 months in the olaparib group, a hazard ratio of 0.35. Using CA-125 as an additional marker of progression further widened the gap between the two cohorts. Following 21 months of follow up no differences in overall survival have been observed; however, 21 patients remain on olaparib compared to 4 on placebo.

Exhibit 5: Phase 2 Olaparib Maintenance Data in High-Grade Serous Ovarian Cancer

	Placebo	Olaparib
PFS - mo.	4.8	8.4
PFS/CA-125 mo.	3.7	8.3
ORR	4%	12%
Deaths n	49	52
OS - mo.	29.9	29.7

Sources: Ledermann et al, NEJM Mar12 and BMO Capital Markets Research.

As noted in the Oza trial, nausea and vomiting were commonly observed in the olaparib arm of the Ledermann trial and around one-quarter of patients either had a dose reduction or interruption.

Data for overall survival (OS) from both the Oza and Ledermann studies are not yet mature. Given relatively low ORRs for targeted therapy in selected patients, we are encouraged to see that AZN will conduct a biomarker analysis combining patient samples from the OZA and Ledermann studies, which in total will include over 350 patient biopsies. In addition to AZN identifying a subset of patients where a more profound benefit to olaparib can be expected, AZN needs to complete development of the tablet formulation. As noted by Oza at ASCO, the current capsule dose is unacceptable for continued development and a tablet formulation is required for further development.

With respect to breast cancer, Gelmon presented data at the 2010 ASCO meeting described data from a Canadian OL trial of olaparib in triple negative breast cancer (TNBC) or high-grade serous ovarian cancer. The TNBC cohort included 25 patients initially (15 with unknown BRCA status) with a plan to add 20 more patients with unknown BRCA status if a response was observed in the original cohort of 15. However, no objective responses were observed in either the known or unknown BRCA TNBC cohorts.

Exhibit 6: TSRO Income Statement 2012A-2017E

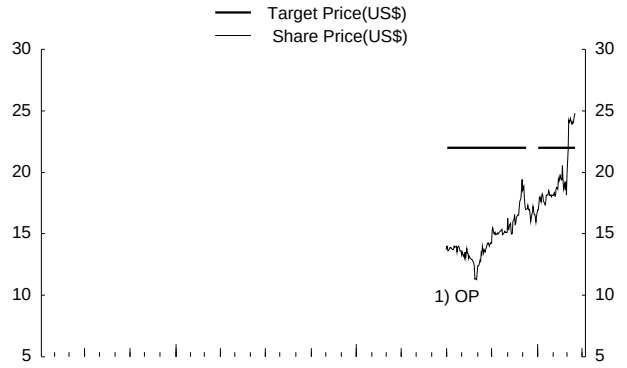
INCOME STATEMENT ($M)	2012A	1Q13E	2Q13E	3Q13E	4Q13E	2013E	2014E	2015E	2016E	2017E
REVENUES										
Product Revenues	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ 43.3	$ 129.9	$ 474.4
Collaborative Revenues/Milestones	-	-	-	-	-	-	-	-	-	-
Total Revenues	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ 43.3**	**$ 129.9**	**$ 474.4**
EXPENSES (GAAP)										
Cost of Goods Sold (COGS)	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ 0.2	$ 0.5	$ 1.7
R&D Expense	47.2	13.5	14.2	15.0	16.0	58.7	67.5	88.9	93.4	125.5
SG&A Expense	6.7	4.0	5.0	5.0	6.0	20.0	30.4	40.0	52.0	159.0
Acquired In-Process R&D	8.0	-	-	-	-	-	-	-	-	-
TOTAL EXPENSES	61.9	17.5	19.2	20.0	22.0	78.7	97.9	129.1	145.8	286.2
Operating Income	**(61.9)**	**(17.5)**	**(19.2)**	**(20.0)**	**(22.0)**	**(78.7)**	**(97.9)**	**(85.8)**	**(15.9)**	**188.2**
Depreciation and Amortization	-	-	-	-	-	-	-	-	-	-
EBIT	(61.9)	(17.5)	(19.2)	(20.0)	(22.0)	(78.7)	(97.9)	(85.8)	(15.9)	188.2
Interest Income	0.2	0.3	0.5	0.4	0.4	1.6	1.2	1.0	1.0	1.2
Interest Expense	-	-	-	-	-	-	-	-	-	-
Other Expense	-	-	-	-	-	-	-	-	-	-
Interest and Other (net)	$ 0.2	$ 0.3	$ 0.5	$ 0.4	$ 0.4	$ 1.6	$ 1.2	$ 1.0	$ 1.0	$ 1.2
Pre-Tax Income	(61.8)	(17.2)	(18.7)	(19.6)	(21.6)	(77.1)	(96.7)	(84.8)	(14.9)	189.4
Income Taxes	-	-	-	-	-	-	-	-	-	37.9
Net Income (GAAP)	**(61.8)**	**(17.2)**	**(18.7)**	**(19.6)**	**(21.6)**	**(77.1)**	**(96.7)**	**(84.8)**	**(14.9)**	**151.5**
EPS (GAAP) (basic)	$ (4.51)	$ (0.53)	$ (0.58)	$ (0.60)	$ (0.65)	$ (2.35)	$ (2.57)	$ (2.09)	$ (0.36)	$ 3.35
EPS (GAAP) (diluted)	**$ (4.51)**	**$ (0.53)**	**$ (0.58)**	**$ (0.60)**	**$ (0.65)**	**$ (2.35)**	**$ (2.57)**	**$ (2.09)**	**$ (0.36)**	**$ 3.35**
Total of Reconciliation Items	1.8	2.0	2.0	2.0	2.0	8.0	8.0	8.0	8.0	8.0
Net Income (Non-GAAP)	$ (60.0)	$ (15.2)	$ (16.7)	$ (17.6)	$ (19.6)	$ (69.1)	$ (88.7)	$ (76.8)	$ (6.9)	$ 159.5
Impact of Adjustments to EPS	0.44	0.06	0.06	0.06	0.06	0.24	0.21	0.20	0.19	0.18
EPS (Non-GAAP) (basic)	$ (22.30)	$ (0.47)	$ (0.51)	$ (0.53)	$ (0.59)	$ (2.11)	$ (2.35)	$ (1.89)	$ (0.17)	$ 3.52
EPS (Non-GAAP) (diluted)	$ (22.30)	$ (0.47)	$ (0.51)	$ (0.53)	$ (0.59)	$ (2.11)	$ (2.35)	$ (1.89)	$ (0.17)	$ 3.52
Weighted average shares outstanding (basic)	13.7	32.3	32.6	32.9	33.3	32.8	37.7	40.7	42.4	44.9
Weighted average shares outstanding (diluted)	13.7	32.3	32.6	32.9	33.3	32.8	37.7	40.7	42.4	44.9

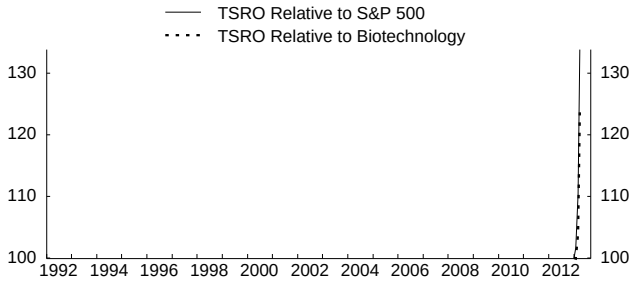
Source: Company reports and BMO Capital Markets

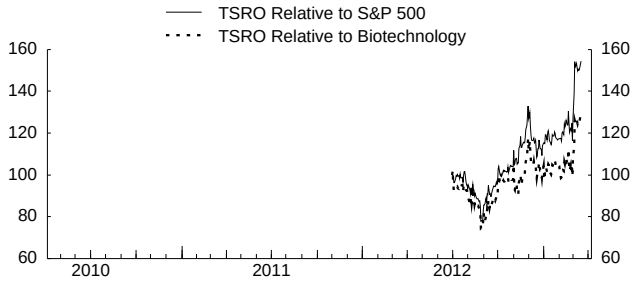
Other companies mentioned (priced as of the close on March 20, 2013):

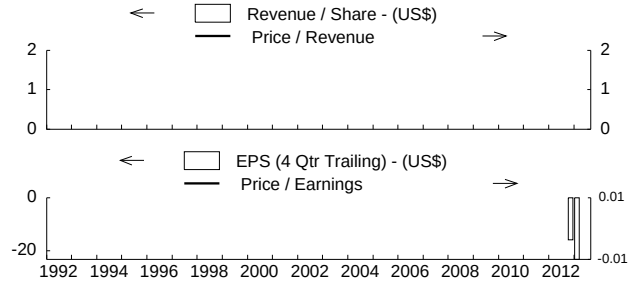
Merck (MRK, $44.12, Outperform by Alex Arfaei)
Sanofi (SNY, $50.71, Not Rated)

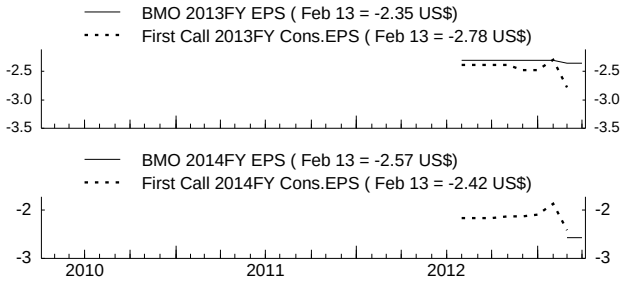
Tesaro Inc. (TSRO)











FYE (Dec.)	EPS US$	P/E Hi	P/E Lo	DPS US$	Yield% Hi	Yield% Lo	Payout %	BV US$	P/B Hi	P/B Lo	ROE %
2012	-4.51	na	na	0.00	0.0	0.0	0	2.6	7.8	4.3	
Range*:		na	na		0.0	0.0			7.8	4.3	
Current*	-23.23	NC		0.00	0.0		0	4.3	4.6		na

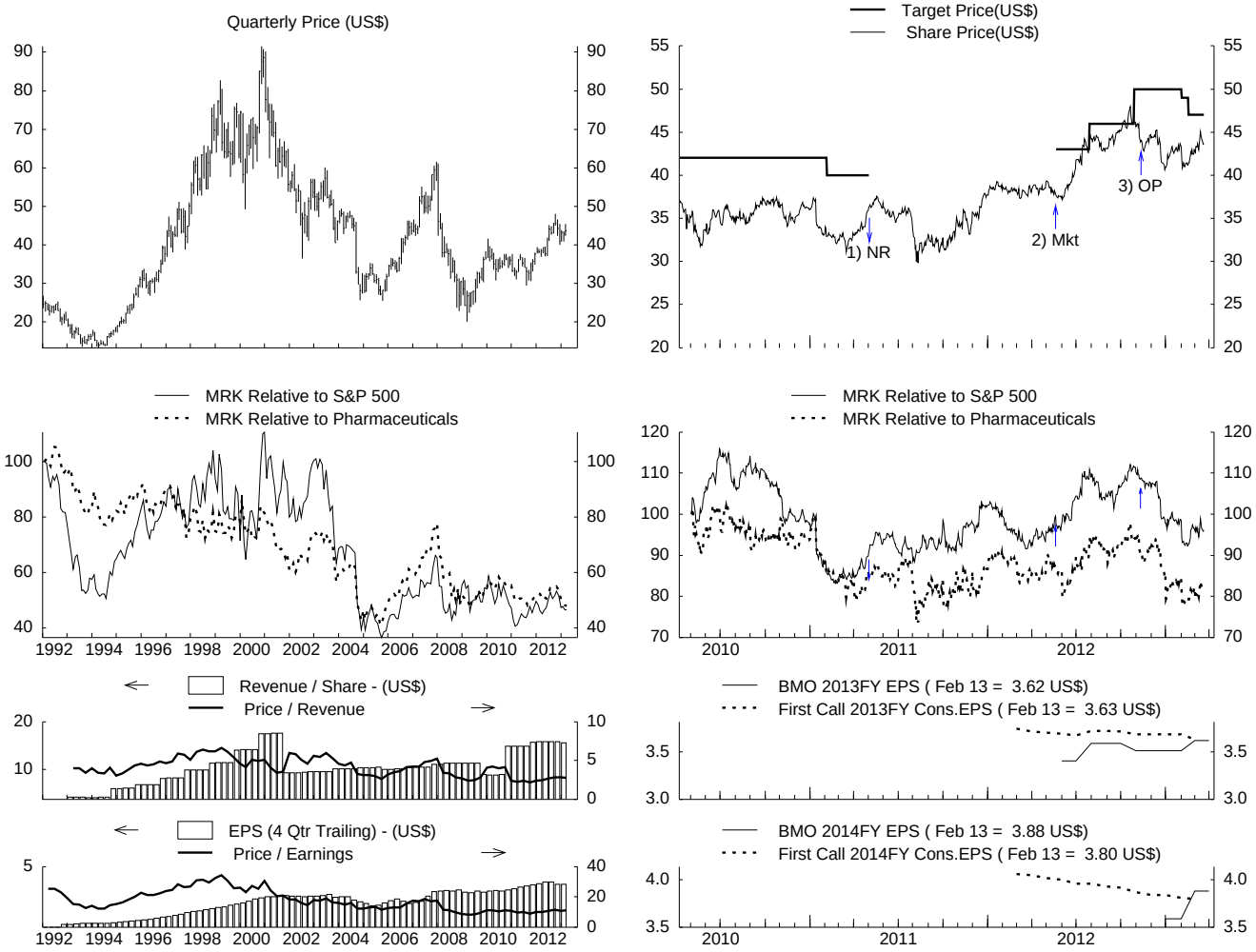
TSRO - Rating as of 23-Jul-12 = OP

	Date	Rating Change	Share Price
1	23-Jul-12	NR to OP	$13.90

* Current EPS is the 4 Quarter Trailing to Q4/2012.
* Valuation metrics are based on high and low for the fiscal year.
* Range indicates the valuation range for the period presented above.

Last Price (March 15, 2013): $24.83
Sources: IHS Global Insight, Thomson Reuters, BMO Capital Markets.

Merck & Co. Inc. (MRK)



FYE (Dec.)	EPS US$	P/E Hi	P/E Lo	DPS US$	Yield% Hi	Yield% Lo	Payout %	BV US$	P/B Hi	P/B Lo	ROE %
1992	1.06	25.3	18.1	0.45	2.4	1.7	43	2.2	12.2	8.8	
1993	1.17	19.3	11.6	0.53	3.9	2.3	45	4.0	5.6	3.4	38
1994	1.19	15.7	11.2	0.57	4.3	3.0	48	4.5	4.2	3.0	28
1995	1.35	23.6	12.8	0.64	3.7	2.0	48	4.8	6.7	3.6	29
1996	1.56	25.6	17.1	0.76	2.8	1.9	49	5.0	8.0	5.4	32
1997	1.87	27.4	19.0	0.85	2.4	1.7	46	5.3	9.7	6.7	37
1998	2.15	35.6	20.9	1.02	2.3	1.3	48	5.4	14.1	8.3	40
1999	2.45	33.8	23.5	1.10	1.9	1.3	45	5.7	14.5	10.1	44
2000	2.90	31.6	17.0	1.29	2.6	1.4	44	6.4	14.2	7.7	48
2001	3.04	29.9	17.7	1.33	2.5	1.5	44	7.1	12.9	7.6	45
2002	2.98	21.8	12.2	1.36	3.7	2.1	46	8.1	8.0	4.5	39
2003	2.92	20.6	13.9	1.48	3.6	2.5	51	7.0	8.6	5.8	39
2004	2.62	18.8	9.8	1.52	5.9	3.1	58	7.8	6.3	3.3	35
2005	2.53	14.0	10.1	1.52	6.0	4.3	60	8.2	4.3	3.1	32
2006	2.52	18.4	11.1	1.52	5.4	3.3	60	8.1	5.7	3.5	31
2007	3.31	18.6	12.8	1.52	3.6	2.5	46	8.4	7.4	5.1	40
2008	3.42	18.0	6.7	1.52	6.7	2.5	44	8.9	6.9	2.6	40
2009	3.26	11.8	6.2	1.52	7.6	4.0	47	19.0	2.0	1.1	23
2010	3.42	12.2	9.0	1.52	5.0	3.7	44	17.6	2.4	1.7	19
2011	3.77	10.1	7.8	1.68	5.7	4.4	45	17.9	2.1	1.6	21
2012	3.82	12.6	9.1	1.72	4.9	3.6	45	18.3	2.6	1.9	21
Range*:		35.6	6.2		7.6	1.3			14.5	1.1	
Current*	3.81	11.2		1.72	4.0		45	18.3	2.3		21
Growth(%):											
5 Year:	2.9			2.5				16.9			
10 Year:	2.5			2.4				8.5			
20 Year:	6.6			6.7				11.2			

MRK - Rating as of 8-Apr-10 = OP

	Date	Rating Change	Share Price
1	3-May-11	OP to NR	$36.41
2	18-May-12	NR to Mkt	$37.82
3	9-Nov-12	Mkt to OP	$44.05

* Current EPS is the 4 Quarter Trailing to Q4/2012.
* Valuation metrics are based on high and low for the fiscal year.
* Range indicates the valuation range for the period presented above.

Last Price (March 19, 2013): $43.70
Sources: IHS Global Insight, Thomson Reuters, BMO Capital Markets.

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Methodology and Risks to Our Price Target/Valuation
Methodology: Please fill in.
Risks: Please fill in.

Distribution of Ratings (December 31, 2012)

Rating Category	BMO Rating	BMOCM US Universe*	BMOCM US IB Clients**	BMOCM US IB Clients***	BMOCM Universe****	BMOCM IB Clients*****	Starmine Universe
Buy	Outperform	37.0%	17.7%	52.9%	38.5%	50.5%	54.0%
Hold	Market Perform	60.7%	9.6%	47.1%	57.3%	48.4%	40.6%
Sell	Underperform	2.4%	0.0%	0.0%	4.2%	1.0%	5.4%

* Reflects rating distribution of all companies covered by BMO Capital Markets Corp. equity research analysts.
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