January 21, 2020

OUTPERFORM

Reason for report:

PROPRIETARY INSIGHTS

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MERSANA THERAPEUTICS, INC.

Time 2b Great: Previewing 1H20 NaPi2b Data

• Bottom Line: In anticipation of the Phase I update of XMT-1536 in 1H20, we conducted a deeper dive to highlight our latest thoughts on XMT-1536. Overall, while there are risks associated with XMT-1536 at this early stage of development, we continue to view the risk/reward as attractive (Linked note To Be Or Not To Be: Could NaPi2b Be A Turning Point?). Lead program XMT-1536 is a NaPi2b-targeting antibody-drug conjugate (ADC) in Phase I development. Importantly, NaPi2b represents a potentially significant commercial opportunity with expression levels of >60% in large oncology indications such as lung and ovarian cancer. Initial Phase I data were presented at the American Society of Clinical Oncology (ASCO) 2019 that showed early signals of activity. However, MRSN continues to be a show me story as the XMT-1536 data disclosed to date remains early and in the context of historical platform concerns following discontinuation of XMT-1522 (HER2-targeting ADC) in early 2019. We see current levels as an attractive entry point for investors. MRSN was highlighted as one of our top picks for 2020 (Linked note: 2020 Outlook: Emerging Oncology - KURA, MRSN, and REPL Top Picks). While there are still risks associated with the story (including target risk associated with NaPi2b and past disappointments e.g., Roche's anti-NaPi2b ADC, lifastuzumab vedotin), there are reasons to believe that XMT-1536 could succeed (including improvements with MRSN's ADC construct and being at a higher effective dose than Roche). In this report, we review the data presented to date and highlight our latest thoughts ahead of the 1H20 update. We supplement our past work (linked above) with additional input from two MEDACorp KOLs (including one involved with the clinical development of lifastuzumab vedotin). MRSN is led by an experienced management team, and we remain optimistic on the potential for long-term appreciation as XMT-1536 and MRSN's platform become de-risked over future clinical updates. **Reiterate OP.**

(Continued inside...)

Key Stats:	**(NASDAQ: MRSN)**
Sector:	**Biopharma / Emerging Oncology**
S&P 600 Health Care Index:	**3,280.56**
Price:	**$8.05**
Price Target:	$11.00
Methodology:	50/50 blend of Discounted Sales Multiple Analysis, and DCF w/ 15% discount rate, 0% terminal growth rate
52 Week High:	$8.93
52 Week Low:	$1.32
Shares Outstanding (mil):	47.8
Market Capitalization (mil):	384.8
Cash Per Share:	$2.24
Dividend (ann):	$0.00
Dividend Yield:	0.0%

Completion: January 21, 2020, 6:15AM EDT.
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Dec Yr	1Q	2Q	3Q	4Q	FY Rev	1Q	2Q	3Q	4Q	FY EPS	P/E
2018A	$3.1	$4.2	$2.2	$1.2	$10.6	($0.54)	($0.54)	($0.74)	($0.97)	($2.79)	NM
2019E	$41.0A	$0.2A	$0.8A	$1.0	$43.1	$0.70A	($0.36)A	($0.35)A	($0.39)	($0.69)	NM
2020E	$0.6	$0.6	$0.6	$0.6	$2.5	($0.40)	($0.42)	($0.44)	($0.39)	($1.63)	NM
2021E	--	--	--	--	$2.1	--	--	--	--	($1.31)	NM

Source: Company Information and SVB Leerink LLC Research.
Revenues presented in $MM, EPS are GAAP.

Please refer to Pages 48 - 49 for Analyst Certification and important disclosures. Price charts and disclosures specific to covered companies and statements of valuation and risk are available at https://svbleerink.bluematrix.com/bluematrix/Disclosure2

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Situation Overview

- MRSN is developing XMT-1536, an antibody-drug conjugate targeting NaPi2b, in a Phase I dose escalation and expansion study. NaPi2b is a sodium-dependent phosphate transporter that is highly expressed in platinum-resistant ovarian cancer (prOC, >60% with assumptions for an expression level cutoff in future development) and non-small cell lung cancer (NSCLC, ~60-80%).
- A past attempt by Roche/Genentech to target NaPi2b with an ADC demonstrated promising early response data in prOC but ultimately failed to show a PFS benefit in a Phase II study.
- XMT-1536 data were last presented at the American Society of Clinical Oncology (ASCO) 2019, which demonstrated early but encouraging activity at likely sub-optimal dose levels.
- MRSN has guided to presenting updated data from the XMT-1536 dose escalation study in 1H20, initial data from dose expansion in 1H20, and more mature expansion cohort data in 2H20.

What we did in this report

- We review the data presented to date and highlight our latest thoughts ahead of the 1H20 update, with a focus on ovarian cancer where we expect to have the most data. We incorporate additional input from two MEDACorp KOLs (including one who was involved with the clinical development of the Roche/Genentech program).
- Our report includes:
  (1) A review of the clinical data presented at ASCO 2019
  (2) Our expectations for the Phase I data, including patient numbers and durability of treatment
  (3) Analysis of the bull and bear case for the data update
  (4) Additional input from MEDACorp KOLs on what would be considered a winning data scenario
  (5) Scenario analysis illustrating implications of the Phase I data update on our price target

Expected Data and Updates

- MRSN has not provided the forum for the data update, and reserved the possibility of a conference call with an investigator independently of a medical conference. We based our calculations on a presentation at ASCO 2020 and assume most of the patients will have ovarian cancer.
- For the dose escalation study, we speculate that ~7 new patients each from the 36 and 43 mg/m^2 cohorts would be evaluable for efficacy (≥2 scans) and ~3 patients from the 52 mg/m^2 cohort would have received ≥1 efficacy scan.
- For the dose expansion study, we speculate that ~14 patients from the 36 mg/m^2 cohort to be evaluable for efficacy (≥2 scans) and ~7 patients from the 43 mg/m^2 expansion cohort to have received ≥1 efficacy scan.

KOL Benchmarks

- For patients treated in the prOC dose-escalation study (median 5 priors), one KOL indicated a response rate of 30-35% and a PFS of ~4 months in the higher dose levels would be viewed as exciting in the sicker ovarian cancer patient population being evaluated. However, a response rate of 25% was viewed as sufficient for further development. The second KOL emphasized the difficulty with benchmarking dose-escalation data and instead highlighted that in a larger prOC dataset in the go-forward population of 1-3 priors, he would want to see a response rate where the 95% confidence interval doesn't overlap with 15%, which represents the historical benchmark.

Market Opportunity and Stock Impact

- Overall, we believe the market opportunity for XMT-1536 across indications is potentially ~$2B (probability-unadjusted), split ~$1.4B for prOC and ~$600M for NSCLC.
- In a positive data scenario, our PT increases to $15 (from $11) based on increasing our probability of success (POS) by 10% in prOC (from 30% to 40%). In a best-case data scenario, our PT increases to $19 at 50% POS. If we increase our POS to 100% in prOC, our PT goes to $37.
- In a disappointing data scenario, reducing our POS in prOC from 30% to 15% drops our price target to $6.

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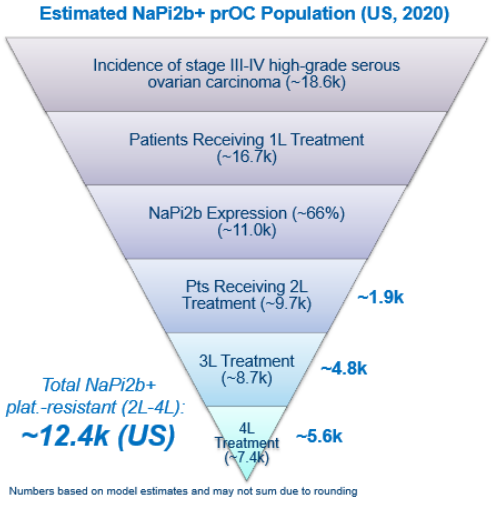
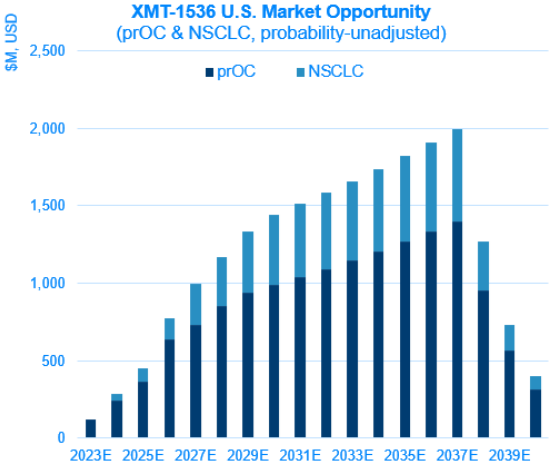
BACKGROUND AND SITUATION ASSESSMENT

MRSN is developing XMT-1536, an antibody-drug conjugate (ADC) targeting NaPi2b, in a Phase I study in platinum resistant ovarian cancer (prOC) and non-small cell lung cancer (NSCLC). NaPi2b is a sodium-dependent phosphate transporter that is highly expressed in approximately ~90% (>60% with assumptions for an expression level cutoff in future development) of prOC and ~60-80% of NSCLC (depending on histology). A previous attempt by Roche/Genentech to target NaPi2b with an ADC demonstrated promising early response data in prOC but ultimately failed to demonstrate a PFS benefit in a Phase II study. MRSN's approach with XMT-1536 uses an entirely different ADC construct, with a different antibody, linker, and payload than lifastuzumab vedotin. Specifically, XMT-1536 has a drug-antibody ratio (DAR) of 10-12, which is ~3x higher than lifastuzumab vedotin and which is designed to deliver higher drug payload at lower doses. It also contains a different active drug payload (auristatin-F vs. MMAE), which demonstrates controlled bystander-effect killing and is not a substrate of P-glycoprotein (a common resistance mechanism for ADCs). MRSN last presented data at the American Society of Clinical Oncology (ASCO) 2019 annual meeting (previewed HERE), which demonstrated early but encouraging efficacy at likely sub-optimal dose levels. MRSN has guided to presenting updated data from the XMT-1536 dose escalation study in 1H20, initial data from dose expansion in 1H20, and updated dose expansion data in 2H20.

Overview of XMT-1536 Market Opportunity





Source: SVB Leerink MRSN Model

REVIEW OF DATA PRESENTED TO DATE

American Society of Clinical Oncology (ASCO) 2019

Initial Phase I dose-escalation data were presented at ASCO 2019. Patients in the Phase I dose-escalation study were not selected for NaPi2b expression and were heavily-pretreated, with

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a median 4 prior therapies (median 5 for ovarian cancer patients). For patients treated at ≥ 20 mg/m^2, the ASCO data demonstrated an overall response rate (ORR) of 17% (3/18) patients across both platinum-resistant ovarian cancer (prOC) and non-small cell lung cancer (NSCLC) adenocarcinoma. Additionally, 9 patients treated at ≥ 20 mg/m^2 observed a treatment duration lasting beyond 16 weeks. Overall, we view these data as encouraging, particularly in the context of the heavily-pretreated patient population and that patients were unselected for NaPi2b expression.

Responses seen in heavily-pretreated prOC patients, with an early trend towards a dose-response relationship. The ASCO poster included response assessments for 19 prOC patients and 3 NSCLC patients who received XMT-1536 at any dose (data cut-off: 05/10/19). Of the 16 evaluable prOC patients treated with doses of 20 mg/m^2 or higher, the ORR was 19% and the disease control rate (DCR) was 57% (3 partial responses [PR]) and 6 stable disease [SD]). The responses appeared dose-dependent (albeit in a small sample size), as prOC patients treated with doses of 30 mg/m^2 or higher demonstrated a response rate of 28% and a DCR of 71% (2 PR and 3 SD of 7 evaluable patients). In NSCLC, 2 patients were treated at ≥ 20 mg/m^2 and both achieved SD.

Response Outcomes of Evaluable Patients Treated in Phase I Dose Escalation Study

Outcome Response Evaluable Population

Outcomes in Ovarian Cancer (OC) & Non-small Cell Lung Cancer (NSCLC)	All OC	All NSCLC	OC ≥20mg/m²	NSCLC ≥20 mg/m²	OC + NSCLC ≥20 mg/m²	OC ≥30 mg/m²
N	19	3	16	2	18	7
PR*	3 (16%)	0 (0%)	3 (19%)	0 (0%)	3 (17%)	2 (28%)
SD*	8 (42%)	2 (67%)	6 (38%)	2 (100%)	8 (44%)	3 (43%)
DCR (PR + SD)	11 (58%)	2 (67%)	9 (57%)	2 (100%)	11 (61%)	5 (71%)
Treatment duration >16 wk	8 (42%)	1 (33%)	8 (50%)	1 (50%)	9 (50%)	3 (43%)
PD*	8 (42%)	1 (33%)	7 (43%)	0 (0%)	7 (39%)	2 (28%)

*As measured by RECIST, version 1.1

- Based on objective responses and duration of treatment, clinical activity was observed at doses of 20 mg/m^2 and higher

As of 10 May 2019

Source: ASCO 2019 Poster #3010

Importantly, management provided additional detail indicating a possible early trend in activity in relationship to NaPi2b expression. The ASCO poster included NaPi2b expression levels for 34 available tumor samples from patients treated on the Phase I study (unselected for NaPi2b expression). MRSN provided separately that of the 3 ovarian cancer patients who had a PR, one patient had an H score of 120, one had H score of 295, and the third was not determined. Furthermore, of the 4 NSCLC patients on study, both patients who demonstrated SD were adenocarcinoma patients with >120 H score, while the other two patients did not have

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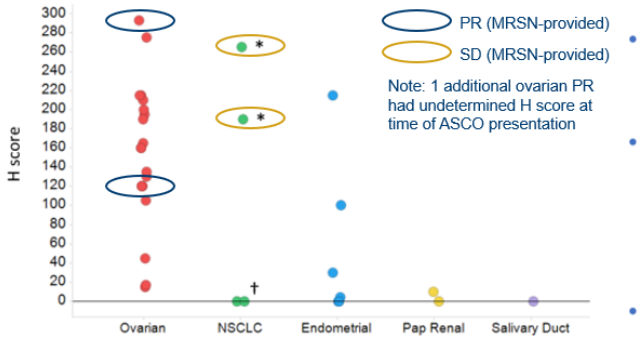
adenocarcinoma histology and had zero target expression. Preclinical experiments demonstrated XMT-1536 activity at H scores ≥70 (Triple Meeting 2017), but as of ASCO, MRSN was still working to determine what the best cutoff is in humans (although in a recent conversation with management they indicated that the clinical experience to date seems consistent with the experience in preclinical experiments). Overall, the expression data from the Phase I study continue to indicate that NaPi2b is broadly expressed on ovarian cancer cells (non-mucinous subtype), which is consistent with the literature that we have reviewed previously (LINK). MRSN also presented clinical expression analysis data from a large cohort of NSCLC patients that demonstrated a high level of NaPi2b expression in patients with adenocarcinoma histology at the AACR Triple Meeting 2019 (shown below).

NaPi2b Expression in Patients Treated With XMT-1536 in the Phase I Study

NaPi2b Protein Expression Measured by IHC from Archival Patient Tumor Samples

Detectable NaPi2b protein expression in all ovarian and lung adenocarcinoma samples



- Proprietary NaPi2b IHC assay formatted to show a range of NaPi2b expression in tumors
- H-Score measures number of positive tumor cells and staining intensity in NaPi2b IHC assay (H-score range 0-300)
- N=34 available tumor samples

*Confirmed lung adenocarcinoma
†One case of poorly differentiated neoplasm and one case with complex histology

Source: ASCO 2019 Poster #3010

AACR Triple Meeting 2019: NaPi2b Expression in NSCLC

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Table 1 NaPi2b Expression in a Cohort of Non-Small Cell Lung Carcinoma Cases

	NAPI2b		
Total (439)	H-score >50	H-score < 50	P value
Sex (%)			
M	82 (27.1%)	221 (72.9%)	<0.001
F	71 (52.2%)	65 (47.8%)	
Histology (%)			
Adenocarcinoma	132 (61.4%)	83 (38.6%)	<0.001
SqCC	12 (6.4%)	166 (93.6%)	
Other	9 (19.6%)	37 (80.4%)	
Smoking (%)			
No	20 (62.5%)	12 (37.5%)	0.001
Yes	133 (32.7%)	274 (67.3%)	
EGFR mutation (%)			
Mutant	18 (75.0%)	6 (25.0%)	<0.001
Wild Type	36 (22.2%)	126 (77.8%)	
KRAS mutation (%)			
Mutant	59 (71.1%)	24 (28.9%)	<0.001
Wild Type	35 (21.9%)	125 (78.1%)	

Source: AACR Triple Meeting 2019 (#A043)

Early durability data trending in the right direction. The swimmer's plot presented at ASCO demonstrated that of the 18 patients treated with ≥ 20 mg/m^2 (across prOC and NSCLC), 9 patients had treatment duration > 16 weeks, including 3 of 7 (43%) prOC patients treated at 30 mg/m^2 or higher. At time of the ASCO analysis, 3 patients remained on treatment, including 2 (both 20 mg/m^2) that had not achieved a PR but were still on treatment at ~36 weeks. The poster also provided that a significant majority of patients were discontinued for either progressive disease per RECIST (53%) or clinical progression (26%). Overall, we view these early durability data as trending positively given the early dose levels. We discuss the durability comparison between the two Phase I datasets in more detail below.

Duration of Treatment of Patients Treated with XMT-1536 in the Phase I Study

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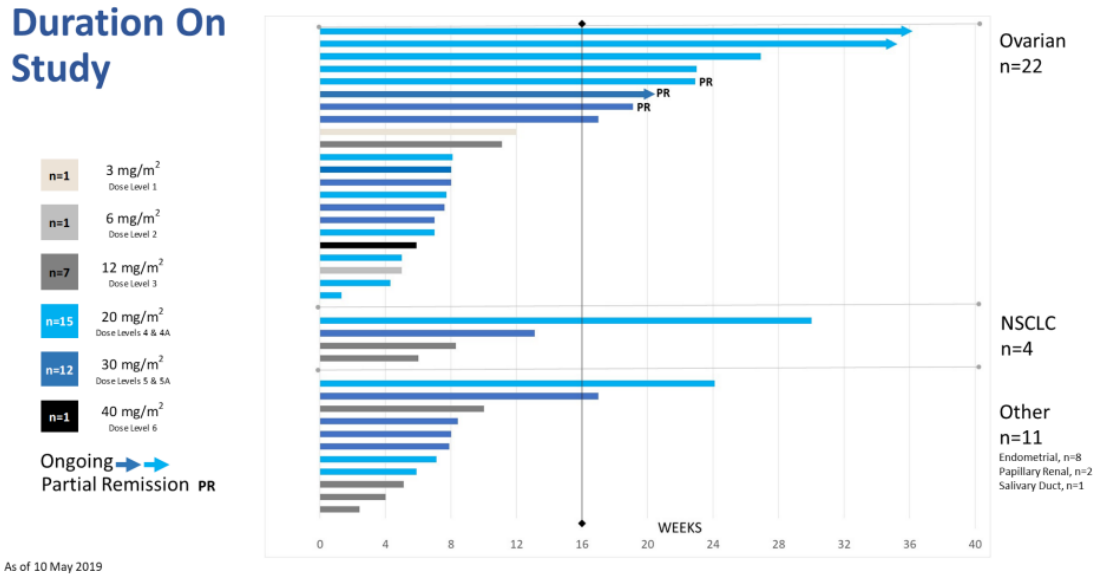

Source: ASCO 2019 Poster #3010

The PK profile of XMT-1536 showed a dose-proportional trend towards increased exposure with increasing dose. Overall, the early PK data in a small number of available patients showed a lower systemic exposure of free payload (AF-HPA) and its metabolite (AF) compared to the conjugated payload. The relationship between free AF-HPA (payload) and AF (metabolite) appeared consistent with preclinical models and suggested that the "locking" mechanism is working as designed. Recall that the DolaLock platform uses an auristatin F-hydroxypropylamide (AF-HPA) payload, which is freely cell permeable and capable of inducing a bystander killing effect. After the AF-HPA is internalized, it is naturally catabolized to auristatin-F (AF), which is not cell-permeable and becomes "locked" within the tumor cell. The Phase I PK data below showed that AF-HPA (payload) exposure slowly declined (dotted line) as AF (metabolite) exposure rose (solid line), indicating an accumulation of the "locked" payload. It is worth noting that the 12, 20, and 30 mg/m^2 doses were the only groups with more than one sample (orange, green, and blue lines respectively). Shown next to the clinical PK data is a preclinical experiment of XMT-1522 (MRSN's previously discontinued DolaLock HER2 ADC), which illustrates the trend more clearly.

Clinical PK Profile of XMT-1536 vs. Preclinical PK Profile of XMT-1522

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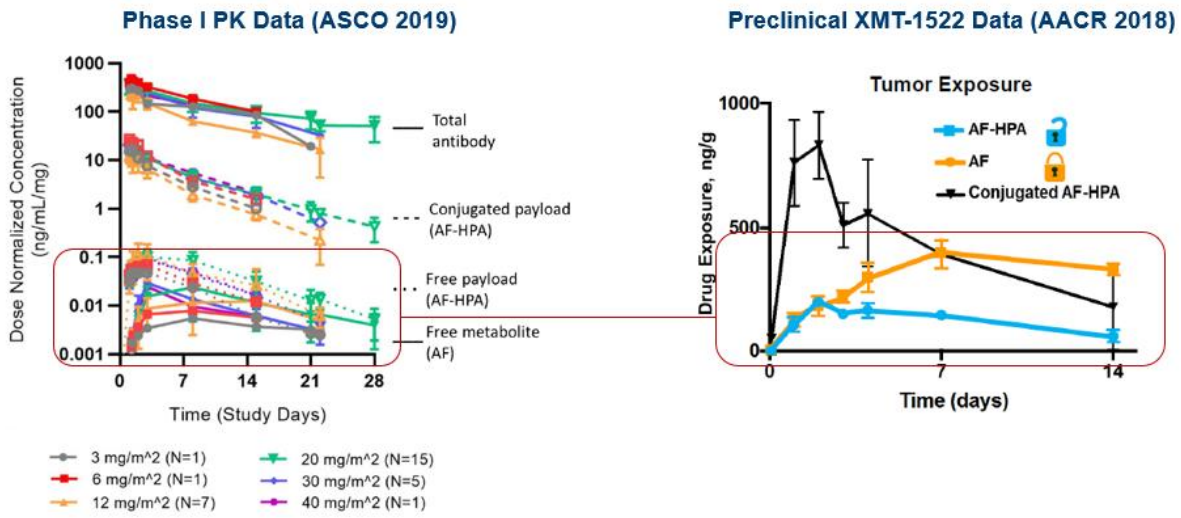

Source: ASCO 2019 Poster #3010, AACR 2018 Poster #754

XMT-1536 generally well-tolerated, with 2 dose-limiting toxicities (DLTs) and no maximum-tolerated dose (MTD) reached at ASCO. The most common treatment-related adverse events (TEAE) were nausea, fatigue, and headache, none of which were grade 3 or above. The most common grade 3 TEAE was increase in aspartate aminotransferase (AST), which was grade 3 in 11% of patients (24% all grades), and which induced 2 DLTs. Both DLTs occurred at Cycle 1, Day 8; one patient treated at 40 mg/m2 (Q3W) had grade 3 AST elevation that resolved to grade 1 within 21 days, the second patient was treated at 30 mg/m2 (Q4W) and had grade 3 AST elevation that resolved to grade 1 within 13 days. Serious adverse events (SAEs) occurred in 13 patients, two of which were considered treatment-related. One SAE of grade 2 pyrexia (considered probably-related) occurred at dose level 5 (30 mg/m2, Q3W) and one SAE of grade 3 cardiac failure congestive (considered possibly-related) occurred at dose level 4 (20 mg/m2, Q3W). There were no grade 4 or grade 5 TRAEs, and there was a low rate of neutropenia, ocular toxicity, and peripheral neuropathy, which are toxicities often associated with microtubule-targeting agents or ADCs.

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Treatment-Related Adverse Events Occurring in ≥10% of Patients

N = 37, data cutoff date 10 May 2019

Preferred Term	N (%) Grade 1	Grade 2	Grade 3	Total
Nausea	12 (32)	2 (5)	0	14 (38)
Fatigue	4 (11)	7 (19)	0	11 (30)
Headache	5 (14)	5 (14)	0	10 (27)
Aspartate aminotransferase (AST) increased	3 (8)	2 (5)	4 (11)	9 (24)
Decreased appetite	1 (3)	6 (16)	0	7 (19)
Blood alkaline phosphatase increased	6 (16)	0	0	6 (16)
Vomiting	4 (11)	1 (3)	0	5 (14)
Gamma-glutamyltransferase (GGT) increased	3 (8)	0	1 (3)	4 (11)
Myalgia	3 (8)	1 (3)	0	4 (11)
Pyrexia	3 (8)	1 (3)	0	4 (11)

Source: ASCO 2019 Poster #3010

Importantly, the ongoing dosing schedule was amended from every 3 weeks to every 4 weeks. The initial dosing of the XMT-1536 Phase I study was once every 3 weeks (Q3W), from dose levels 1-3. At dose level 4 (20 mg/m2), a once every 4 weeks (Q4W) dosing schedule was initiated alongside the Q3W schedule (schematic shown below). Notably, at dose levels above 20 mg/m2, the Q3W and Q4W dosing schedule each observed 1 DLT (of 2 total DLTs), and both of the SAEs were observed at the Q3W dosing schedule, despite more patients being treated at the Q4W dosing schedule (n=17 at Q4W vs. n=11 at Q3W). Overall, these observations suggest that the Q4W dosing schedule was better-tolerated at time of ASCO, and MRSN elected to continue dose escalating at the Q4W dosing schedule (discussed below).

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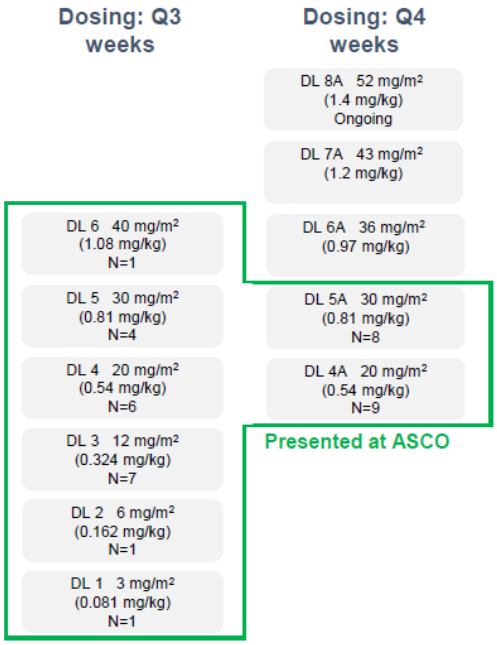
Ongoing Dose Escalation Schema



Source: MRSN January 2020 Corporate Slides

Post-ASCO 2019 Updates on Dose Escalation & Dose Expansion

Incrementally positive dose escalation updates provided post-ASCO 2019. At time of ASCO, dose escalation was ongoing at the 36 mg/m^2 (Q4W) dose. MRSN provided incremental updates on the dose escalation and expansion studies in the meantime:

- In August (2Q19 earnings call), MRSN provided that dose escalation remained ongoing and that a maximum-tolerated dose (MTD) had not yet been reached. At this time, management was deciding between the 30 mg/m^2 and 36 mg/m^2 doses (both Q4W) as the go-forward dose (LINK).
- Later in August, MRSN announced that they had initiated the dose expansion cohorts at 36 mg/m^2 (Q4W) dosing for both prOC and NSCLC. Notably, the prOC cohort of the dose expansion study enrolled patients with 1-3 prior lines of therapy, which contrasts with the dose escalation population where ovarian cancer patients had median of 5 prior lines of therapy. The prOC expansion cohort was later amended to include patients with 4 prior lines of treatment, regardless of platinum status (shown below). Regarding the dose escalation, MRSN also announced that the MTD had not yet been reached, and that dose escalation would continue (alongside the expansion cohorts) at 43 mg/m^2 (Q4W). At time of the update, MRSN believed that the 43 mg/m^2 (dose-escalation) cohort would enroll a very late-line patient population, and they expected investigators would reserve the less heavily-

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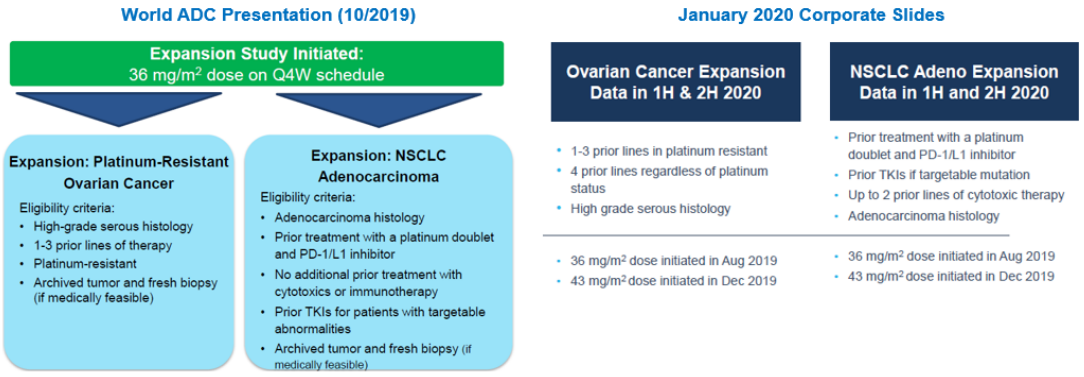
pretreated patients for the 36 mg/m^2 expansion cohort. At the time, MRSN also noted that they have evaluated a handful of additional patients at the 36 mg/m^2 dose (as well as some additional patients at the 30 mg/m^2 dose) and that a number of these patients are still on drug (LINK).

Dose Expansion Schema for prOC and NSCLC



Source: Presentation at World ADC Summit (Oct 2019) and Jan 2020 Corporate Slides

- **In November (3Q19 earnings call), MRSN provided another incrementally positive update.** The highlight from this update was that of the 3 patients treated at the 43 mg/m^2 dose-escalation dose, XMT-1536 continued to be well-tolerated and there were no DLTs observed to date. At the time, MRSN planned to continue enrollment and evaluation of additional patients at this dose level before deciding next steps for dose-escalation and expansion (LINK).
- **In January 2020, MRSN provided a positive safety update and continues to dose-escalate.** Importantly, MRSN provided that they had continued to dose 3-4 additional patients (6-7 total, later confirmed to be 7 total) at the 43 mg/m^2 Q4W dose, and had yet to see any dose-limiting toxicities at this dose. As a result, MRSN initiated dose level 8A (52 mg/m^2 Q4W). Also importantly, MRSN increased the dose from the dose expansion portion of the study (both cohorts) from 36 mg/m^2 (Q4W) to 43 mg/m^2 (Q4W). All newly recruited patients on the expansion study will receive the 43 mg/m^2 (Q4W) dose, and there will be no intra-patient dose escalation. In a discussion with management, MRSN provided that they believe this provides an opportunity to directly compare the 36 and 43 mg/m^2 doses in a larger and more homogeneous patient population. The January update also provided more detail on the cadence for 2020 data readouts, which we discuss further below.
- **Announcement of a second NaPi2b-targeting ADC (XMT-1592) helps to frame MRSN's strategy in lung cancer.** At the January 2020 update, MRSN also announced the development of XMT-1592, a second NaPi2b-targeting ADC that utilizes the company's Dolasynthen (vs. the Dolaflexin technology used in XMT-

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1536). MRSN noted that one of the biggest bottlenecks in bringing a new ADC forward is the cell line and antibody manufacturing. Thus, because MRSN already has the antibody, they can quickly move forward with this program and understand how the Dolasynthen technology translates in the clinic. Importantly, management believes that this study will not compete with patients for the XMT-1536 study, noting that they have enough sites up and running and are continuing to initiate new sites. They also believe that leveraging their knowledge and experience with NaPi2b will help them move through dose escalation pretty rapidly.

EXPECTATIONS FOR THE PHASE I DATASET

MRSN will provide updated dose escalation data and initial data from dose expansion in 1H20. MRSN also provided that they would present more mature expansion cohort data in 2H20. In terms of timing for the 1H20 update, MRSN has indicated to us the following possibilities: Society of Gynecologic Oncology (March 28-31), AACR 2020 (April 24-29), ASCO 2020 (May 29-June 2), or announce on a conference call with an investigator.

Amount of follow-up will vary, depending on when MRSN presents data. MRSN has not yet decided on a forum for the 1H20 data disclosure given the data are still emerging. This leaves a lot of variability in how much follow-up (i.e., how many tumor imaging scans) patients will have in the 1H20 data disclosure. Regardless, there are some known variables that can be used to estimate approximately how many scans we can see for each group of patients, and how many patients we can expect at each dose cohort. The known variables include:

Durability:

- Scan frequency for once-every-four-week dose: after a baseline scan, patients receive scans every 8 weeks (i.e., week 0, 8, 16, etc.). If at any given scan the patient has a PR (dubbed an unconfirmed PR), the patient will have a second scan ~3-4 weeks later (dubbed the confirmatory scan). For example, if a patient demonstrates a PR at their week 8 scan, they will have a confirmatory scan around week 12. If a patient at week 8 has SD, their next scan would be at week 16. Additionally, MRSN noted that their DLT evaluation period is 1 month.
- Approximate initiation dates for expansion cohort (both 36 mg/m^2 and 43 mg/m^2 dose)
- Approximate initiation dates for dose escalation cohorts (both 43 mg/m^2 dose and 52 mg/m^2 dose)
- Conference dates and MRSN's historically preferred data cut-off date for conferences
    - MRSN's 5/10/2019 data cut-off was 22 days prior (~3 weeks) to their 6/1/2019 ASCO 2019 presentation

Patient numbers:

- MRSN plans to have a minimum of 45 patients in each expansion cohort (ovarian and lung), though the study could end up over-enrolling depending on investigator interest,

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MRSN's desire for more NaPi2b expression data, or to enrich for other factors (i.e., a particular prior therapy).

- MRSN has provided that the FDA typically wants ~250-300 patients in the safety database for an accelerated approval (which is consistent with what we hear from KOLs). So if we assume the single-arm registrational study enrolls ~100 patients, we would expect to see about ~150 from the dose escalation and expansion studies.
- Single-patient dose cohorts for the first two levels of dose-escalation, followed by a standard "3+3" design with option for 4th patient at each dose level

By ASCO (if patients are still on drug), most patients in the 36 mg/m^2 expansion cohort should have 2 scans; for the 43 mg/m^2 expansion cohort, the last date to enroll a patient and see 1 scan by ASCO is approximately early March. Based on the above variables, some conclusions can be drawn about how many patients will be *efficacy-evaluable* in each cohort, and how much follow-up we can expect for each. If we assume a presentation at ASCO, the following conclusions can be made:

1. **36 mg/m^2 Expansion Cohort:** Given the expansion cohort is transitioning to enrollment of patients at 43 mg/m^2 between late December and early January, there should be few 36 mg/m^2 patients enrolled beyond the middle of January. Instead, most 36 mg/m^2 patients were likely enrolled in a period between late-August to late-December. As such, we expect that by the time of ASCO, most 36 mg/m^2 expansion cohort patients will have at least 2 scans if they remain on drug.
    a. During the same 4-month period (late August to late-December), MRSN enrolled 7 patients to complete the 43 mg/m^2 dose escalation cohort. However, the "3+3" design requires a 1-month safety evaluation at the end of the first "3" and the end of the second "3" before the decision to dose-escalate. Thus, the effective rate of enrollment was 7 patients in 2 months (3.5 pts / month). We apply this same rate of enrollment to the 36 mg/m^2 expansion cohort over the same period of time (late August-late December), and note that not all sites were immediately converted to the 43 mg/m^2 expansion cohort in December, so there could be some variability here. **Expectation: ~14 patients (we assume mostly ovarian)**

2. **43 mg/m^2 Expansion Cohort:** The amendment to dose escalate the expansion cohort to 43 mg/m^2 was submitted in late December. Effectively, this means the enrollment period for these patients began in early January. By our calculations, in order for a patient to have at least 1 efficacy scan by the time of ASCO a patient should be enrolled before early March 2020. However, this represents only ~2 months of recruitment at the 43 mg/m^2 dose. Therefore, if MRSN decides to present data from these patients in 1H20, we should expect the data to be early and for there to be a limited number of patients.
    a. If we assume the same enrollment rate as we do for (1), we conservatively estimate ~3.5 patients enrolled per month between early January and early March. **Expectation: ~7 patients (we assume mostly ovarian)**

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3. **36 mg/m^2 Dose Escalation Cohort:** Enrollment should have completed by latest mid-August, meaning this cohort largely complete (full safety assessment and multiple scans for each patient, provided they remained on drug).
    a. Dose escalation in the "3+3" (+1) design is complete. **Expectation: 7 patients (we assume mostly ovarian)**

4. **43 mg/m^2 Dose Escalation Cohort:** Enrollment should have completed by late-November, leaving this cohort also largely complete. Assuming a hypothetical last patient in on December 1st, that patient (and the rest of the cohort) should have 2 scans (week 8, 16) by the time of a hypothetical ASCO data cut-off.
    a. Dose escalation in the "3+3" (+1) design is complete. **Expectation: 7 patients (we assume mostly ovarian)**

5. **52 mg/m^2 Dose Escalation Cohort:** Similar to the 43 mg/m^2 expansion cohort, the enrollment period for these patients effectively began in early January. With a DLT evaluation period of 1 month, we believe it is highly likely that MRSN could have 3 patients worth of safety data to determine if 52 mg/m^2 represents the maximum tolerated dose (MTD). If MRSN does not hit DLTs at 52 mg/m^2, management mentioned that they would want to add another 3 patients to gain experience with the dose (similar to their 43 mg/m^2 strategy). Efficacy evaluations for this patient cohort would be the same as for the 43 mg/m^2 expansion cohort: in order for a patient to have at least 1 efficacy scan by the time of ASCO a patient should be enrolled before early March 2020.
    a. Assuming the same enrollment rate as we do for (1) and (2), we conservatively estimate ~3.5 patients enrolled per month between early January and early March. However, there is a 1-month DLT evaluation period after the last patient from the first "3" is dosed. **Expectation: ~3 patients (we assume mostly ovarian)**

As an additional data point, we looked at the mirvetuximab soravtansine enrollment rates in the Phase I expansion study. The expansion study initiated in August 2014 (*Moore, 2017*), and a data update was presented at ASCO 2015 (data cut-off: 04/30/15). If we assume mid-August start date we have approximately 8 months between September 2014 and April 2015. The ASCO data included 20 patients from the expansion cohort, for an enrollment rate of 2.5 patients per month. However, enrollment was restricted to patients with medium-high expression of FRα, while MRSN's expansion cohort enrolls all comers. IMGN assumed 60% of the overall population had medium-high FRα expression, so if we apply this factor to our enrollment rate: *2.5 patients per month / 60% of patients with FR*α expression = ***~4.2 patients per month.***

A comparison of the speed of trial site activation suggests that MRSN could enroll patients in the expansion cohort faster than IMGN did. For another additional data point, we compared the clinicaltrials.gov entries for XMT-1536 and mirvetuximab soravtansine to see how quickly the trial sites were activated in each study (following initiation of the expansion cohorts). Because we know the exact enrollment window for XMT-1536 (expansion study announced on 08/20/19), we tracked the number of new sites enrolled between August 2019 and today (latest entry: 01/08/20). We looked at the same enrollment window (5 months) for mirvetuximab following the initiation of

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the expansion study (09/03/2014). Both studies had 8 trial sites activated at the time that dose expansion was initiated, but the XMT-1536 trial sites appear to have come online faster (over the same period of time) compared to the mirvetuximab expansion study. We note that two of the recently activated sites from the XMT-1536 trial appear to have principal investigators who are lung specialists, so we removed those from the calculations below.

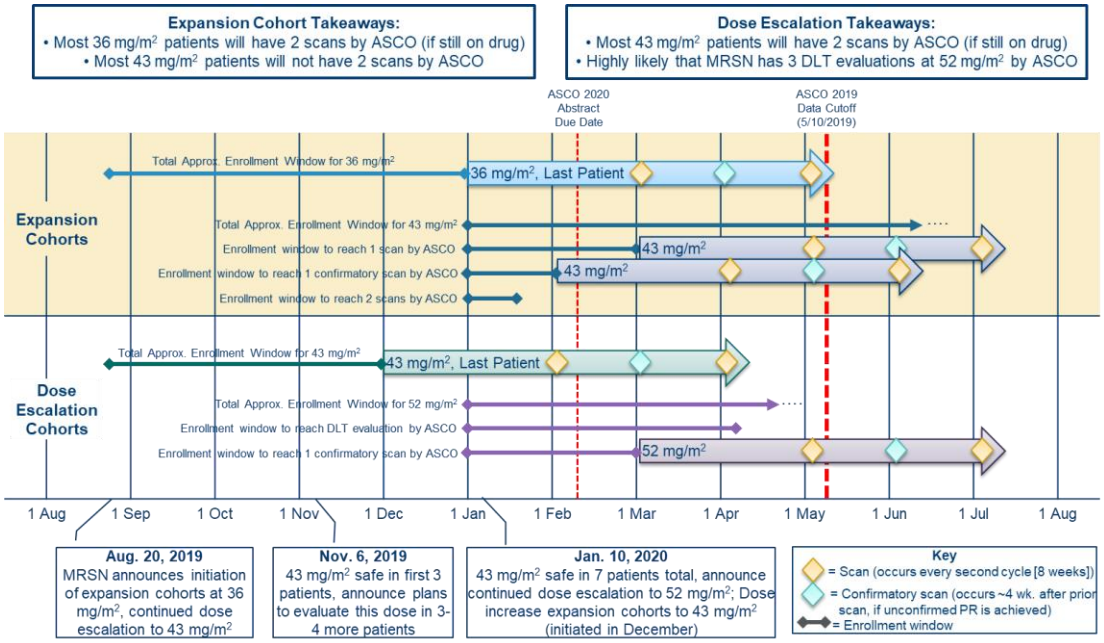
Comparison of Site Activation From XMT-1536 and Mirvetuximab Expansion Studies

	Study Start Date[1]	Enrollment Window *(for calc.)*[2]	Months	Sites at Start Date[1]	New Sites Enrolled[1]	Total Sites (at end date)[1]
XMT-1536	10/24/2017	08/15/2019-01/08/2020	5	8	11*	19* (01/08/20)
Mirvetuximab soravtansine	06/01/2012	09/03/2014-02/12/2015	5	8	2	10 (02/12/15)

* = Excludes two additional sites in which the principal investigator has a lung cancer specialty
(1) Per clinicaltrials.gov
(2) Mirvetuximab expansion study initiated August 2014, closest trial entry used (09/03/2014); XMT-1536 expansion study announced 08/20/19

Source: SVB Leerink Research; Clinical Trials Database, Accessed 01/16/20

Patient Follow-up Analysis: How Many Scans Will Patients Have By ASCO



Source: SVB Leerink Research

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Summary of Patient Estimates (Assuming ASCO 2020 Presentation)

Dose	Escalation	Expansion
36 mg/m^2	7	14
43 mg/m^2	7	~7
52 mg/m^2	~3	N/A
Total (New Pts)	**~17**	**~21**

Source: SVB Leerink Research

Benchmark prOC response rates: dose escalation (~5 prior therapies)

Response rates to chemotherapy in prOC are significantly diminished in later lines of therapy. According to a KOL we spoke with, there aren't a lot of data available in prOC patients with 3+ prior therapies, particularly that demonstrate response rates to a specific type of therapy. Two retrospective studies have been published analyzing response rates to chemotherapy by line of therapy in prOC. Overall, both studies demonstrate that the response rate to chemotherapy beyond the 3L setting is significantly diminished in subsequent lines of treatment, and that by the 5L-6L setting the response rate is in the single digits. However, we do note a few important caveats. Importantly, both studies defined a partial response as a reduction of 50% or more in the parameters of measurable lesions (vs. current standard RECIST 1.1 criteria of 30%, which is used in the MRSN Phase I study). Both studies are also dated and likely represent a different standard of care than current practice; the Bruchim study analyzed patients from 1995-2003 while the Hoskins study assessed patients diagnosed prior to 1999. Further, the Bruchim study assessed patients from the Meir Medical Center in Israel, and the Hoskins study assessed patients from the British Columbia Cancer Agency (BCCA) database. Ultimately, while it is difficult to compare these data and other early chemotherapy studies to the current standard of care, the studies provide evidence of diminished response rates by line of therapy in prOC.

ORR (≥50% tumor reduction) by Line of Therapy in prOC

Source	Input	2L	3L	4L	5L	6L
Hoskins, et al	N	*96*	*65*	*44*	*27*	*13*
	ORR	*34%*	*20%*	*20%*	*11%*	*8%*
Bruchim, et al	N	*44*	*63*	*36*	*24*	*14*
	ORR	*26%*	*12%*	*3%*	*5%*	*0%*
Weighted Avg.		**32%**	**16%**	**13%**	**8%**	**4%**

Source: Bruchim, Eur J Obstet Gynecol Reprod Biol. 2013 Jan;166(1):94-8 ; Hoskins, Gynecol Oncol. 2005 Jun;97(3):862-9.

Overall, these late-line response rates are largely in-line with KOL feedback and indicative of the standard of care for patients treated in the dose escalation study. While MRSN expects future development of XMT-1536 to focus on the 2L-4L population, the bar for efficacy in

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patients treated in the dose-escalation portion of the Phase I study is low, where the data suggest single digit response rates are associated with standard-of-care chemotherapy. One KOL we spoke to noted that the response rate drops toward 15% in the more heavily-pretreated patient population based on his clinical experience. According to a second KOL, Phase I (dose escalation) patients are typically not relevant to the broader population, because ovarian cancer is so heterogeneous, and patients are so heavily pre-treated that the false discovery rate here can be very high. This KOL's estimate for a response rate based on his clinical experience is ~5-10% in patients with 5 prior lines of therapy, but he noted that this number would not necessarily apply to a Phase I population.

Benchmark prOC response rates: dose expansion (1-3 prior therapies)

According to one KOL we spoke with, a response rate in prOC (1-3 priors) of ~20-25% and PFS of ~7 months would be viewed as a win. The KOL also added that the drug would need to demonstrate a manageable toxicity profile, which he believes XMT-1536 would certainly fit (based on the data to date).

A second KOL believes the historical experience in patients with 1-3 priors has been an ORR of ~15%, and his benchmark ORR would be any response rate with non-overlapping confidence intervals. According to this KOL, a point-estimate response rate in the dose expansion population (those with 1-3 priors) where a 95% confidence interval did not overlap with 15% would be considered the benchmark for new therapies.

The FDA's recent guidance to IMGN (MP) indicates a response rate of ~12% is considered the benchmark for single agent chemotherapy in prOC patients treated with 1-3 priors (2L-4L). IMGN is also developing an ADC, mirvetuximab soravtansine, which targets folate receptor alpha (FR-α) in prOC. IMGN's registrational strategy for mirvetuximab soravtansine involves a single-arm, pivotal study (SORAYA) to support accelerated approval, with a confirmatory Phase III study (MIRASOL) comparing mirvetuximab to investigator's choice chemotherapy. While MRSN had previously suggested that a single-arm study would support accelerated approval for XMT-1536, the FDA's decision to allow IMGN to pursue this pathway offers additional validation of this strategy. The SORAYA study will enroll ~100 patients and has a primary endpoint of ORR, and the FDA has provided to IMGN that the benchmark for single-agent chemotherapy in this setting would be 12% (IMGN Investor Call Transcript). IMGN provided that the 12% ORR was based on the Phase III AURELIA and CORAIL studies that included patients both naïve to and previously treated with bevacizumab. IMGN has also pointed to the response rate of 31.4% observed in 70 patients from the previously failed FORWARD-I study who met the eligibility criteria for SORAYA. The XMT-1536 Phase I expansion study in prOC is enrolling patients at the same line of therapy (1-3 priors) as the SORAYA study (as well as some patients with 4 priors, regardless of platinum-status). However, it is important (from an efficacy standpoint) to consider that these patients are not selected for NaPi2b expression (as they would likely be for an accelerated approval study).

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ORR in AURELIA Study in Overall Population and by Chemotherapy Cohort

	AURELIA				CORAIL
Regimen	Total Control Arm	PLD	Topotecan	Paclitaxel	PLD / Topotecan
# Priors	1-2	1-2	1-2	1-2	2 (prior chemo)
% 2 prior Tx	43%	33%	46%	51%	-
ORR*	**11.8%**	**7.8%**	**0.0%**	**30.2%**	**12.2%**
mPFS	3.4 mo.	3.5 mo.	2.1 mo.	3.9 mo.	3.6 mo.
mOS	13.3 mo.	14.1 mo.	13.3 mo.	13.2 mo.	11.1 mo. (interim)

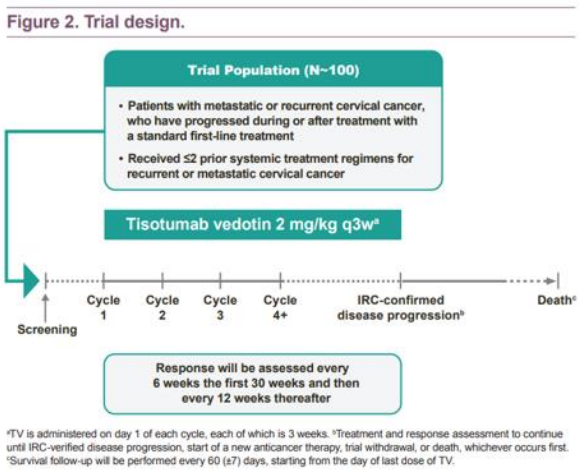
* By RECIST criteria (version 1.0 for AURELIA, version 1.1 for CORAIL)
PLD = Pegylated liposomal doxorubicin

Source: J Clin Oncol 32:1302-1308. 2014; J. Clin Oncol 33 3836-3838. 2015; Oaknin et al, ESMO Abstract 932O, Presented 10/2018

Both KOLs believe the path forward in prOC is through an accelerated approval trial with ORR as the primary endpoint. According to one KOL, the pivotal study to support accelerated approval would be in a highly-expressed population and would include ~100-150 patients. This KOL pointed to IMGN's SORAYA study as an example. The second KOL believes the pivotal study would need to have ~100 patients or more and pointed to the pivotal Phase II single-arm study (innovaTV 204) of tisotumab vedotin in cervical cancer. Overall, the KOL feedback is consistent with what MRSN has been hearing from investigators and appears in-line with the FDA's latest guidance to two other ADC programs.

innovaTV 204 and SORAYA Trial Designs (Both Registration-Directed)

innovaTV 204



SORAYA

SINGLE-ARM PIVOTAL TRIAL FOR MIRVETUXIMAB USING PS2+ SCORING IN FRα-HIGH, PLATINUM-RESISTANT OVARIAN CANCER

PRIMARY ENDPOINT
ORR by Investigator

SECONDARY ENDPOINT
DOR by Investigator

ENROLLMENT AND KEY ELIGIBILITY
~100 patients
Platinum-resistant disease (primary PFI >3 mos)
Prior bevacizumab required
Prior PARPi allowed
Patients with BRCA mutations allowed

Source: ASCO 2018 (Abstract TPS5601), Adapted from IMGN Corporate Slides (Jan 2020)

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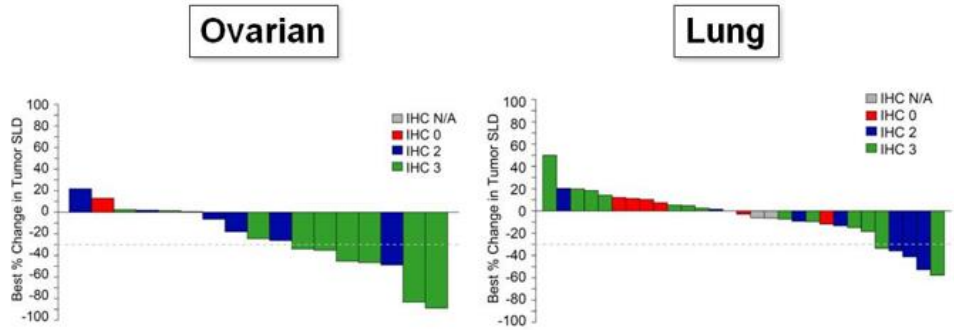
BULL CASE FOR THE PHASE I DATA READOUT

First, a brief review of lifastuzumab vedotin

RHHBY's (NR) lifastuzumab vedotin was the first ADC to target NaPi2b. A Phase I dose-escalation study was initiated in 2011 in NSCLC and prOC. In the Phase I study (which did not select patients for NaPi2b expression), promising response rates were observed in prOC patients with high levels of NaPi2b expression (IHC 2/3+), with 7/17 (41%) of patients treated at the recommended Phase II dose (RP2D, 2.4 mg/kg) achieving a partial response (PR). In NSCLC, the ORR was 10% (2/21) in patients with IHC 2/3+ treated at the RP2D or higher. The maximum tolerated dose (MTD) was not reached, and based on the maximum administered dose (2.8 mg/kg), the RP2D was determined to be 2.4 mg/kg (Q3W). Following the data presented at ASCO 2014, Roche/Genentech initiated a Phase II study in prOC evaluating lifastuzumab vedotin (LIFA) vs. PLD control (PEGylated liposomal doxorubicin).

Phase I Response Assessments of Lifastuzumab Vedotin



Response Assessment at RP2D (2.4 mg/kg)	Ovarian IHC 0	Ovarian IHC 2/3+	Lung IHC 0	Lung IHC 2/3+
Confirmed RECIST Response Rate	0% (0/1)	*41%* (7/17)	0% (0/5)	10% (2/21)
Clinical Benefit Rate (cPR or SD ≥ 3 months)	0% (0/1)	*53%* (9/17)	20% (1/5)	48% (10/21)

None of the patients with tissue unevaluable for NaPi2b staining demonstrated response to treatment

Source: Burris, et al, Presented ASCO 2014

Phase II study did not meet primary endpoint of PFS. The Phase II open-label study enrolled 95 patients with prOC (median 2 prior systemic therapies) randomized 1:1 to receive either LIFA (2.4 mg/kg, Q3W) or PLD (40 mg/m^2, Q4W). Like the Phase I, patients were not selected for NaPi2b expression. The ORR for LIFA was largely consistent with the Phase I data and significantly better than PLD control in both the ITT population (34% vs. 15% control, p=0.03) and the NaPi2b-high (IHC 2/3+) group (36% vs. 14% control, p=0.02). However, the study did not meet the primary endpoint of mPFS in the ITT, with LIFA demonstrating mPFS of 5.3 months vs. 3.1 months PLD (Hazard ratio [HR]: 0.78, 95% CI: 0.46-1.31, p=0.34). In the NaPi2b-high patients, the mPFS was 5.3 months LIFA vs. 3.4 months PLD (HR: 0.71, 95% CI: 0.40-1.26, p=0.24).

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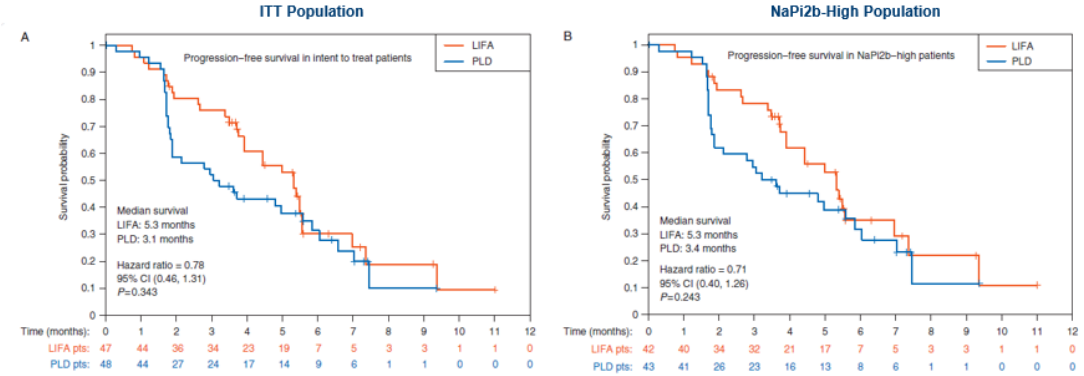
Following the failure of the Phase II study, Roche/Genentech discontinued the lifastuzumab program in 2014.

A KOL involved with the clinical development of lifastuzumab vedotin provided some insight on why the program was discontinued. According to this KOL, the entire Roche/Genentech experience with lifastuzumab vedotin was problematic for a number of reasons. While some of these problems were related to their ADC, the KOL believes there were problems across the entire development pipeline, and the company had a number of ADCs that didn't pan out. According to the KOL, there was a global decision made at one point to discontinue the program, and for what it's worth, the KOL believes that this does not make NaPi2b any less of a reasonable target for an ADC.

Phase II PFS of Lifastuzumab Vedotin vs. PLD Control



Source: Ann Oncol. 2018 Apr 1;29(4):917-923

XMT-1536 is a substantially different ADC construct from lifastuzumab vedotin. Lifastuzumab vedotin is a monoclonal antibody (targeting NaPi2b) conjugated to monomethyl auristatin E (MMAE) payload via a protease-cleavable peptide (valine-citrulline) linker. In preclinical development, the average drug-to-antibody ratio (DAR) was 3.5 for the anti-NaPi2b conjugate and other control conjugates (*Lin, Clin Can Res, 2015*). Alternatively, XMT-1536 uses a different NaPi2b-targeting antibody, a different payload (auristatin-F vs. MMAE) and a different linker (ester vs. vc). Importantly, XMT-1536 has a Fleximer polymer technology that produces a higher drug-antibody ratio of ~10-12. The use of the AF-HPA payload provides a controlled bystander killing effect that is designed to limit exposure of the anti-mitotic payload to normal tissue. And unlike MMAE, the AF payload is not a substrate of p-glycoprotein, which is a common resistance mechanism associated with traditional ADC approaches. We review these components below.

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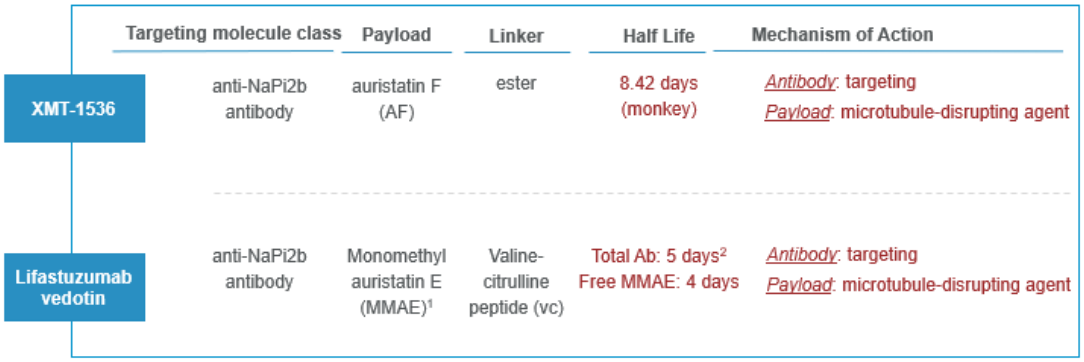
Comparison of XMT-1536 and Lifastuzumab Vedotin



Source: Clin Cancer Res. 2015 Nov 15;21(22):5139-50; 2. Phase I study of DNIB0600A, Presented ASCO 2013. 3. J Nucl Med July 2009 vol. 50 no. 7 1153-1160

XMT-1536 delivers a higher dose on a payload basis than lifastuzumab vedotin

MRSN's Fleximer polymer allows for higher DAR on XMT-1536, which could result in more potent payload delivery than lifastuzumab vedotin. The higher drug-antibody ratio (DAR) of XMT-1536 results in more chemotherapeutic payload being delivered into the tumor cell at each binding and internalization event. XMT-1536 carries approximately 10-12 payloads per ADC molecule, while lifastuzumab vedotin (LIFA) had a DAR of ~3.5. In preclinical models, MRSN saw greater efficacy of XMT-1536 compared to LIFA in both ovarian and lung cancer models, which management attributed to the higher DAR of XMT-1536 (LINK). MRSN also believes that it can dose higher on a payload basis without the dose-limiting toxicities that Roche observed, which were consistent with the MMAE payload (neutropenia, peripheral neuropathy). In both rats and non-human primates (NHP), MRSN was able to dose at 2x the payload level without any neutropenia or peripheral neuropathy, and the drug was well-tolerated overall.

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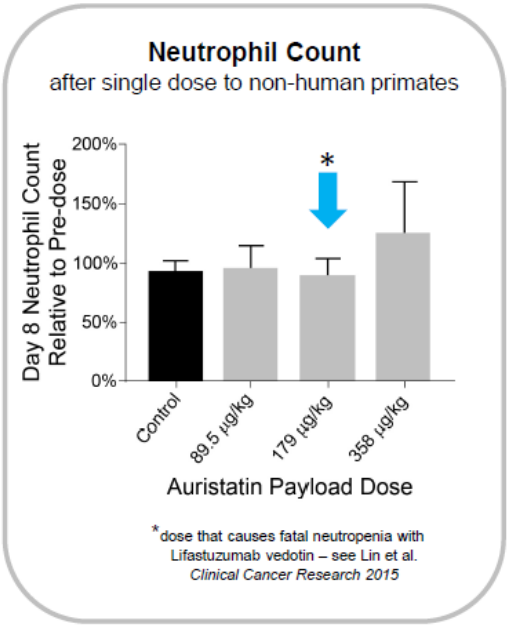
Day 8 Neutrophil Count After Single-Dose of Auristatin Payload



Source: MRSN World ADC 2019 Presentation

MRSN's higher DAR suggests that XMT-1536 can achieve a higher dose than lifastuzumab vedotin on a payload basis (assuming all other unknowns are equal). At dose level 6 (40 mg/m^2; Q3W), the estimated exposure of XMT-1536 was ~41% higher than the Phase II dose of lifastuzumab vedotin. The ASCO data included one patient who received this dose, who had a dose-limiting toxicity (Grade 3 AST elevation, resolved to Grade 1 within 21 days). At dose levels 5 (30 mg/m^2; Q3W) and 6A (36 mg/m^2; Q4W), the estimated exposure is the closest to the LIFA RP2D (~6% higher at dose level 5 and ~5% lower at dose level 6A). Notably, dose level 6A was the initial dose administered in the XMT-1536 expansion cohort, where the line of therapy is more similar to the Phase II LIFA study (1-3 priors). As mentioned, the expansion study dose has been increased to dose level 7A (43 mg/m^2), though patients treated at 36 mg/m^2 will continue to receive that dose (no intra-patient dose escalation). Most importantly for the MRSN bull argument, the amended expansion cohort dose (dose level 7A) provides approximately 18% higher payload exposure than the LIFA RP2D, and MRSN has dosed 7 patients at this dose without observing any dose limiting toxicities. Furthermore, the highest dose in the dose escalation (52 mg/m^2) provides approximately 40% higher payload exposure than the LIFA RP2D. The table below shows a calculation of payload exposure assuming all other factors are equal (blue box denotes dose levels presented at ASCO 2019) and assuming a DAR of 11 (10-12) for XMT-1536.

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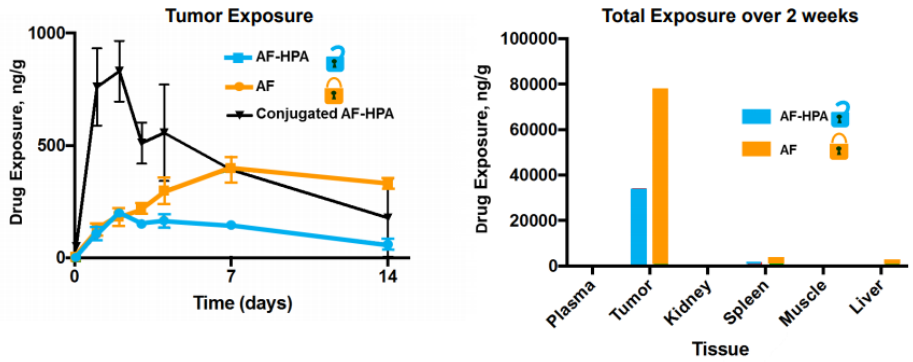
Estimated Payload Exposure of XMT-1536 vs. LIFA (Accounting for Dosing Frequency)

	Dose	Dosing Frequency		Systemic Exposure		Exposure	Difference
	Cohort	Schedule	Conv. Factor	Ab dose (mg/kg)	DAR	Calculated Exposure	%Change vs. LIFA
LIFA	RP2D	Q3W	1.3	2.4	3.5	**11.2**	-
XMT-1536	4 (20 mg/m^2)	Q3W	1.3	0.54	11	**7.9**	**(29%)**
	5 (30 mg/m^2)	Q3W	1.3	0.81	11	**11.9**	**6%**
	6 (40 mg/m^2)	Q3W	1.3	1.08	11	**15.8**	**41%**
	6A (36 mg/m^2)	Q4W	1	0.97	11	**10.7**	**(5%)**
	7A (43 mg/m^2)	Q4W	1	1.2	11	**13.2**	**18%**
	8A (52 mg/m^2)	Q4W	1	1.4	11	**15.4**	**38%**

Source: SVB Leerink Calculations

However, based on the PK comparison between XMT-1536 and LIFA, the frequency of dosing may not be relevant in this calculation. In comparing the effective payload exposure of XMT-1536 and lifastuzumab vedotin, we originally accounted for the dosing frequency (Q3W for LIFA vs. Q4W for XMT-1536 [at higher doses]). However, MRSN argues that the frequency of Q3W vs. Q4W is not important for calculating the effective exposure, as the concentration of their auristatin-F (AF) payload will last much longer than other ADCs based on the DolaLock mechanism. MRSN has not presented data directly comparing the MMAE and AF payloads. Conceptually however, MMAE is bystander capable, meaning it will diffuse out of the tumor on a continuous basis. Alternatively, the DolaLock AF payload is designed to become "locked" into the tumor and accumulate within the tumor (discussed above, shown again below). At 14 days, the AF (locked) payload is still present at high concentrations relative to the AF-HPA and conjugated antibody. It is also worth noting that XMT-1536 has a half-life of ~1 week (*ASCO 2019 poster*), while the half-life of LIFA is 5 days (*ASCO 2013 presentation*).

Tumor Exposure to AF Payload *in vivo* (XMT-1522)



Source: AACR 2018 Poster #754

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If we remove frequency of dosing from the calculation, MRSN has already dosed a number of patients at payload doses of ~30-60% higher than LIFA. Further, the next cohort of dose escalation (52 mg/m^2) is approaching a dose of ~2x higher (83%) than the LIFA Phase II dose. Under these assumptions, the closest dose of XMT-1536 to the LIFA Phase II dose would be dose level 5 (30 mg/m^2). Overall, while we discuss a number of caveats with this calculation below, the directional takeaway is that XMT-1536 is likely providing substantially more payload exposure than LIFA was able to provide in the Phase II study (blue box denotes dose levels presented at ASCO 2019).

Estimated Payload Exposure of XMT-1536 vs. LIFA (Removing Dosing Frequency)

	Dose	Systemic Exposure		Exposure	Difference
	Cohort	Ab dose (mg/kg)	DAR	Calculated Exposure	%Change vs. LIFA
LIFA	RP2D	2.4	3.5	8.4	-
XMT-1536	4 (20 mg/m^2)	0.54	11	5.9	**(29%)**
	5 (30 mg/m^2)	0.81	11	8.9	**6%**
	6 (40 mg/m^2)	1.08	11	11.9	**41%**
	6A (36 mg/m^2)	0.97	11	10.7	**27%**
	7A (43 mg/m^2)	1.2	11	13.2	**57%**
	8A (52 mg/m^2)	1.4	11	15.4	**83%**

Source: SVB Leerink Calculations

A number of additional factors affect payload delivery aside from DAR. We caveat the above calculations by pointing out that the rate and extent of payload delivery depends on both tumor properties (antigen type, antigen expression and turnover rate, tumor type) and ADC characteristics including uptake, internalization, and biochemical transformation (i.e., degradation of antibody, linker cleavage, and immolation to release payload). The level of payload in the tumor is further determined by a variety of additional factors, including the amount of conjugate entering the tissue, the local ADC catabolism rate, and the payload tissue-retention properties (*Zhang, 2019*). Overall, the different dose, linker, antibody PK profile, and (potentially) dosing schedule all serve to complicate any direct comparison of payload exposure beyond a directional level. However, looking at the known variables, MRSN appears to be able to provide meaningfully higher payload exposure with XMT-1536 than lifastuzumab vedotin.

A KOL we spoke with was highly intrigued by XMT-1536 demonstrating a DAR of 10-12 and believes that this will translate to better efficacy. Overall, the KOL believes that if MRSN is able to continue dosing above the LIFA dose (on a payload basis) with controlled toxicity, then they will be able to administer more drug and also get more of a bystander effect as well. The KOL noted that early generation ADC development was less precise and created a distribution of antibodies, some with higher amounts of drug conjugated and others with less drug, which resulted in unpredictable toxicity profiles. Subsequent ADC constructs then went the opposite direction, attaching 2-3 chemotherapy moieties to each antibody, which controlled the toxicity but didn't deliver enough of the drug (the KOL notes that this is where he believes Roche/Genentech

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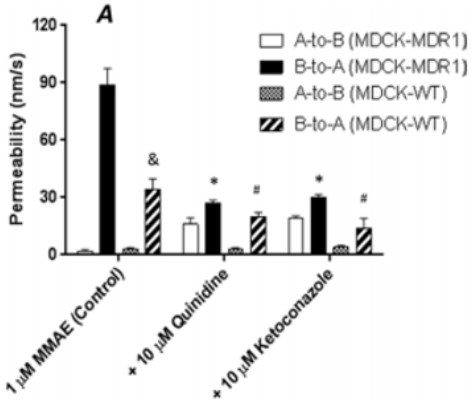
was). The KOL also noted the importance of the linker, and believes that the LIFA linker was not conjugated tightly enough to the drug, which resulted in early drug release and toxicity. Overall, the KOL believes that MRSN is in the 3rd generation of ADC and believes the DAR of 10-12 will translate into better efficacy than past approaches.

The XMT-1536 payload is not a substrate of P-glycoprotein

P-glycoprotein is a known resistance mechanism to chemotherapeutic agents, including MMAE. The P-glycoprotein (P-gp) pump binds several drugs (including chemotherapeutic microtubule-disrupting agents) and transports them outside of the cell, preventing them from reaching the intended targets and leading to drug resistance. Common ADC toxins, such as DM4 and DALVBH, are substrates of P-glycoprotein (P-gp). MMAE, the payload attached to lifastuzumab vedotin, has been shown to be a substrate for P-gp (*Liu-Kreyche, 2019*) in a study of the role of P-gp on the cytotoxic effects of brentuximab vedotin and its payload MMAE. In this study, Liu-Kreyche and colleagues demonstrated that P-gp mediated efflux (transport) of MMAE occurred in both MDCK-wild-type and MDCK-MDR1 cells, and that the P-gp mediated efflux was completely eliminated by introducing known P-gp-MDR1 inhibitors (quinidine or ketaconazole). Furthermore, the cytotoxic effect of MMAE was potentiated by combination treatment of elacridar (a potent P-gp inhibitor) in cell lines with higher P-gp expression, while the impact of the P-gp inhibitor was less pronounced in the cell lines with low levels of P-gp.

P-gp mediated efflux of MMAE is decreased with introduction of P-gp inhibitors

Transepithelial transport of MMAE from basolateral to apical compartment (B-to-A) is decreased with the introduction of P-gp inhibitors



Source: Liu-Kreyche, et al. Front Pharmacol. 2019 Jul 17;10:749

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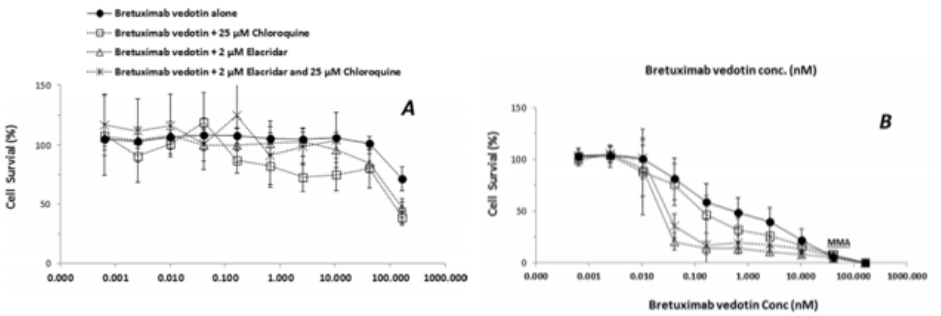
MMAE cytotoxicity is potentiated with introduction of P-gp inhibitors

Potentiation of MMAE cytotoxicity with the addition of P-gp inhibitors in tumor cells with high levels of P-gp expression

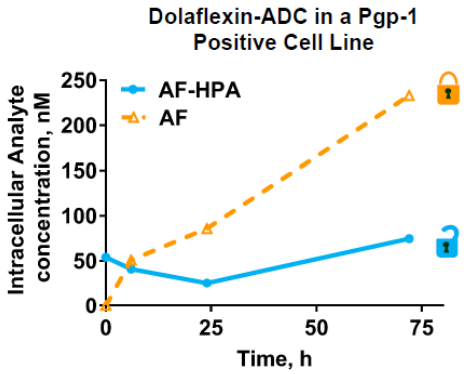


Source: Liu-Kreyche, et al. Front Pharmacol. 2019 Jul 17;10:749

MRSN's auristatin-F (AF) toxin is not a substrate of P-glycoprotein. Multi-drug resistance (MDR) transporter studies have demonstrated that AF, in contrast to AF-HPA, is not a P-gp substrate. AF was shown to accumulate in a P-gp positive cell line, while AF-HPA was effluxed. In the presence of valspodar (P-gp inhibitor), the efflux of AF-HPA was substantially reduced, indicating that AF-HPA is a P-gp substrate. Thus, AF-HPA (P-gp substrate) remains freely cell-permeable, which supports the mechanism of bystander killing of nearby tumor cells. Alternatively, when AF-HPA accumulates in the cells and then converts to AF, the AF (not a P-gp substrate) remains locked inside the tumor cell.

Auristatin-F Is Not A Substrate of P-gp-1 Drug Efflux Pump

Test Article	Direction	Efflux Ratio	Pgp substrate classification
AF	A-to-B	1.8	Negative
	B-to-A		
AF + 1 uM Valspodar	A-to-B	1.4	
	B-to-A		
AF-HPA	A-to-B	32	Positive
	B-to-A		
AF-HPA + 1 uM Valspodar	A-to-B	4.7	
	B-to-A		



- Even in the presence of a major drug efflux pump, AF is still locked in the cell.

Source: MRSN AACR 2018 Poster (#754)

According to a KOL, the fact that AF is not a P-gp substrate is helpful, but the impact of the clinical significance in ovarian cancer is still unclear. A KOL we spoke with pointed out that P-gp has been around for "centuries" but has never been clinically relevant, and the closest relevance it has is in leukemia. The KOL noted that elevated levels of P-gp are correlated with lack of response in leukemia, but that this correlation has not been observed in solid tumors. However, the KOL also noted that this could be because in general, current treatments in solid

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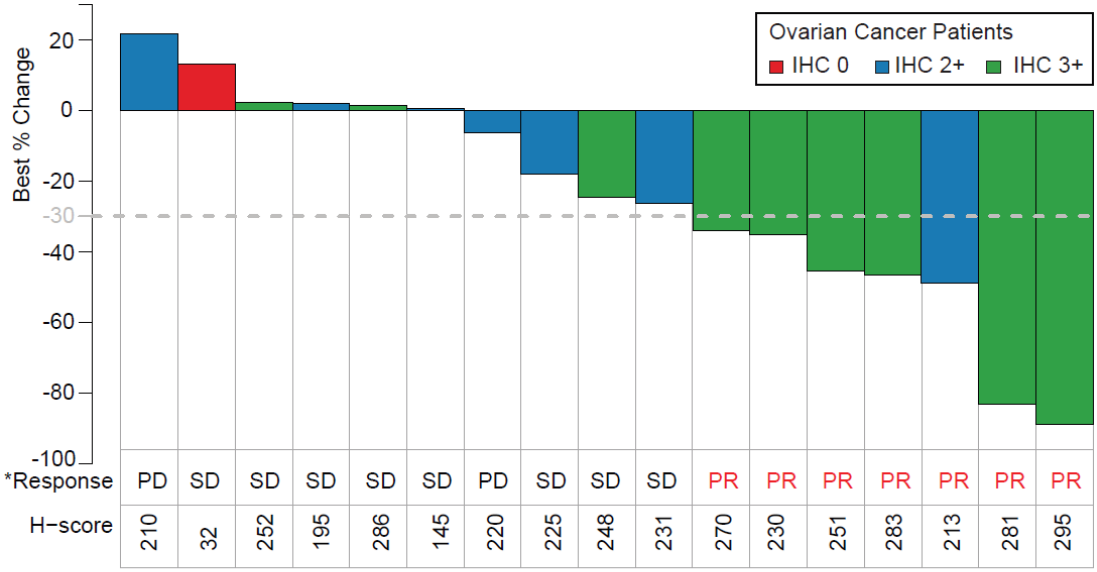
tumors are so [ineffective] that it is hard to tease out the role of P-gp as a pure mechanism of resistance. So overall, the KOL views that the fact that AF cannot be pumped out of the tumor as helpful, but it is still unclear as to how important that will be.

XMT-1536 has shown efficacy at lower NaPi2b expression levels than LIFA

XMT-1536 has demonstrated clinical activity at lower NaPi2b expression levels (by H-score) than lifastuzumab vedotin (although small patient numbers). As mentioned above, a possible early trend in XMT-1536 activity correlated with NaPi2b expression levels, as the two prOC patients who saw a partial response (PR) had H-scores of 295 and 120 respectively (a third PR had undetermined H-score). While it is unclear which doses these specific patients received (2 PRs were at dose level 5/5A, 1 PR was at dose level 4A), it is worth noting that RHHBY/Genentech did not observe any PRs with a NaPi2b-expression H-score of lower than 213 at the RP2D (2.4 mg/kg).

Responses to Lifastuzumab Vedotin of prOC patients at the RP2D (Phase I)



*Response for ovarian cancer patients was same by RECIST and Best Response. RECIST required confirmation (i.e., 2 responses at least 4 weeks apart) and for Best Response the patients responded at least once;
PD: progressive disease; PR: partial response; SD: stable disease

Source: Gerber, et al, Clin Cancer Res. 2019 Sep 20

NaPi2b expression is highly heterogenous, which MRSN believes is an advantage for the bystander killing of its DolaLock platform. While NaPi2b is highly expressed in non-mucinous ovarian cancer and NSCLC, its expression is highly heterogenous and the number of NaPi2b-positive cells varies significantly (*Kiyamova, 2011*). MRSN believes that its AF-HPA payload, and the controlled bystander-effect killing that it induces, will be beneficial in tumors with heterogeneous antigen expression. AF-HPA is a tubulin-disrupting agent, so as the cleaved AF-HPA diffuses from the antigen-positive cells to nearby antigen-negative cells, it is more likely to kill tumor cells (where there is more tubulin to disrupt) than normal cells.

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The high DAR of XMT-1536 could be well-suited for NaPi2b because the target is selectively expressed and not highly expressed. Management has previously provided that there are ~80k-100k copies of NaPi2b per cell, based on ovarian cancer cells from an OVCAR model, which is relatively low in comparison to a target like HER2 (800k-1M copies/cell). We discuss this in greater detail in a subsequent section, but it is worth noting here because MRSN believes they can get much more efficient delivery to tumor bearing cells by delivering ~10-12 drugs per internalization rather than ~3.5 (LINK).

One KOL described 2 key variables of NaPi2b expression in ovarian cancer: topographic distribution and longitudinal expression. According to this KOL, we don't really know at this point how heterogeneously NaPi2b is expressed in ovarian cancer. Because of the genomic instability of high grade serous ovarian cancer, the KOL views the topographic distribution of NaPi2b at time of diagnosis as primarily important. For instance, at the time of debulking surgery, there will be several pounds of tumor removed. The KOL believes we need to understand the expression of NaPi2b across that entire mass of tumor—does all of the tumor overexpress NaPi2b? Or just in certain sections of the tumor? This is seen as very relevant for selection and development of resistance. The other issue, in the KOL's mind, is that we don't yet know what the longitudinal expression of NaPi2b is yet (a key risk, described later). Specifically, we don't know if patients who express high levels of NaPi2b at time of diagnosis will continue to have high levels of expression over the natural history of their disease. For example, regarding the potential of downregulation of NaPi2b, the KOL believes it is unknown whether there is actual downregulation of the target vs. heterogeneity with the selection of lower-expression clones in a given tumor sample. Finally, in regard to NaPi2b as a marker rather than a driver of oncogenic growth, the KOL does not see this as a big issue at all, noting that the purpose of an ADC target is simply to get the drug to the tumor cell in a protected way and avoid off-target toxicity.

Overall, the KOL remains "quite bullish" on NaPi2b as a target and noted that the Roche/Genentech experience does not make it any less reasonable of a target. The KOL noted that target downregulation is the million dollar question for ADCs in solid tumors, but determining actual downregulation of the target vs. heterogeneity with the selection of lower expression clones is something that we don't really know. The KOL noted that he is unaware of any data showing NaPi2b downregulation in patients. Notably, when Roche/Genentech published the Phase II study of lifastuzumab vedotin, the investigators suggested that "one possibility is downregulation of the ADC target; while NaPi2b is highly expressed in 90% of ovarian cancer, it is not clear how vital it is to continued survival of the OC cell" (*Banerjee, 2018*). However, there remains very little data from this study (or others) on the longitudinal expression and potential downregulation of NaPi2b. Overall, the KOL remains optimistic on NaPi2b, and views the bystander effect as a way to mitigate the heterogeneity issue and potential target downregulation. Notably, the KOL believes that if you can deliver enough drug to the target while the target is still there, and enough drug leaks out into the tumor, you can kill a lot of tumor cells there that either (a) don't express the same level of target or (b) have downregulated the target.

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ASCO 2019 likely represents the floor for efficacy

Given the lower doses presented at ASCO, we think there is a reasonable chance that the XMT-1536 response rate improves over ASCO in the higher dose escalation cohorts. As mentioned above, the response rate at ASCO was 19% for prOC patients treated at ≥ 20 mg/m^2 and 28% for patients treated at ≥ 30 mg/m^2 (including both Q3W and Q4W dosing). In dose escalation, MRSN has now dosed 7 patients at 43 mg/m^2 (Q4W) without any dose limiting toxicities, and has progressed dose escalation to dose level 8A (52 mg/m^2; Q4W). Based on the early trend toward a dose response that was observed in the ASCO dataset, we would expect the clinical activity of XMT-1536 to continue to improve, even in dose-escalation, when compared to the patients at ASCO who received 20 and 30 mg/m^2 and had partial responses.

ASCO response rate of 28% in n=7 patients treated at ≥30 mg/m^2 in the 6L (median 5 prior lines) setting bodes favorably for the dose expansion population in the 2L-4L setting. In the dose expansion study, prOC patients are now being treated at the 43 mg/m^2 (Q4W) dose. These patients likely represent a much earlier line population (1-3 priors) than the dose-escalation population (median 5 priors), though the expansion cohort also allows patients with 4 priors, regardless of platinum status. Given what we know about diminishing response rates by line of therapy in prOC, we would expect the response rates to improve in the 2L-4L patient population with a higher dose (43 vs. 30 mg/m^2) and a better-tolerated dosing schedule (Q4W vs. Q3W).

BEAR CASE FOR THE PHASE I DATA READOUT

What happened with XMT-1522 (HER2-targeting ADC)?

XMT-1522 was officially discontinued "due to the competitive environment for HER2-targeted therapies". But a safety issue may cloud this rationale in some investors' minds. In January 2019, with limited cash resources and stock levels near historic lows following a Phase I clinical hold, MRSN made the difficult decision to discontinue XMT-1522 and focus their resources on advancing XMT-1536. Although MRSN cites the competitive HER2 environment as the reason for discontinuation, investors still question the efficacy and tolerability profile of XMT-1522 and wonder what role this played in the decision to discontinue the program. Given that XMT-1536 and XMT-1522 were designed with the same Dolaflexin ADC technology, investors are justified to wonder and analyze what happened with XMT-1522 and how that may impact the potential of XMT-1536. Specifically, investors often ask (1) will the safety issues that XMT-1522 experienced extend to XMT-1536? and (2) was there an emerging dose-response relationship with XMT-1522? While valid questions, we believe MRSN has adequately addressed the safety concerns and provided additional evidence of a dose-response relationship with XMT-1536 to assuage any concerns stemming from XMT-1522.

A dose limiting toxicity and Grade 5 patient death tarnished the XMT-1522 program's image as a safer platform. At the time of discontinuation, XMT-1522 was in a Phase I dose

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escalation study treating HER2-expressing patients with breast, gastric or lung cancers. MRSN had cleared dose level 7 (28.3 mg/m^2, Q3 weeks) and proceeded into dosing patients at dose level 8 (37 mg/m^2, Q3 weeks) when a partial clinical hold was placed on the trial (July 2019). The hold was placed due to a Grade 5 adverse event (patient death) in dose level 7, which was deemed to be "possibly drug-related". The Grade 5 patient was described to have had metastatic disease since 2014 with advanced hepatic cirrhosis. The patient tolerated the first dose well, but on receiving the second dose came into the hospital complaining of illness, fever, and was found to have elevated liver enzymes and high ammonia levels. The patient was treated and seemed to get better, but when released from the hospital the patient decided to leave the trial and enter hospice. Because of this, it is not known exactly what the cause of death was and thus designated "possibly drug-related".

This series of events led MRSN to submit protocol amendments which included (1) increased patient monitoring, (2) exclusion of patients with advanced hepatic impairment and (3) evaluation of alternative dosing regimens. Under these amendments, the partial clinical hold was lifted approximately 2 months later. The amendments were also concurrently implemented to the Phase I trial of XMT-1536. Additionally, at ASCO 2018 (before the partial clinical hold was announced), there was one dose-limiting toxicity (DLT) reported in the 7th dose level. The patient developed transient high fever, grade 3 AST (aspartate transaminase) elevation and grade 2 ALT (alanine transaminase) elevation, but symptoms resolved to grade 1 and patient continued treatment at reduced dose. Overall, these issues tarnished the image of XMT-1522 as a potentially safer next-generation ADC, even though there was limited evidence of other toxicities typically seen with other ADCs such as neutropenia, ocular toxicities, peripheral neuropathy, or pneumonitis.

MRSN is now at a higher dose with XMT-1536 than ever reached with XMT-1522 – without any new safety signals. Despite the disappointing safety signals seen with XMT-1522, MRSN learned from the situation and pressed forward with the development of XMT-1536. MRSN maintained confidence in the safety profile of their platform, believing that the Grade 5 event was not an indication of a larger platform tolerability issue. Supporting this belief is the fact that MRSN is now dosing XMT-1536 patients well above the highest dose achieved on the XMT-1522 program. As mentioned previously, XMT-1536 has treated 7 patients at 43 mg/m^2 (Q4W) and has not observed any DLTs. By comparison, the highest dose cleared with XMT-1522 was 28.3 mg/m^2, Q3 weeks. This is reassuring to know, because if the safety issues were a larger platform issue MRSN probably would have seen the same issues with XMT-1536 by now. However, there is one wrinkle to this argument: XMT-1536 is going after a different target than XMT-1522. For HER2, there are 800k-1M copies per cell in HER2+ breast cancer. For NaPi2b, there are closer to 80k-100k copies per cell based on measurements in ovarian cancer cells from an OVCAR model. Management previously acknowledged to us that with regards to their high DAR platform, it is not one size fits all and one must consider the context of the specific target. In this context, management

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views the DAR of XMT-1536 (10-12) as well suited for NaPi2b, because NaPi2b is selectively expressed and not highly expressed.

KOL is impressed with XMT-1536 toxicity profile. Based on the data available for XMT-1536, our KOL was impressed with the toxicity profile. He pointed out that the biggest concern was the increase in transaminases (the presence of elevated transaminases can be an indicator of liver and cardiac damage). But in his words "I don't get too excited about those, they are basically just numbers." He points out that there are several drugs (e.g. rucaparib) that cause elevated transaminases. Unless there is elevated bilirubin or decreased albumin, he generally doesn't worry about it.

NaPi2b may not be critical for cell survival. Could this lead to NaPi2b downregulation post treatment?

NaPi2b could be a risky target as it's unclear how vital the target is to survival of the cancer cell. However, KOLs don't see this as a problem because the ADC just uses NaPi2b as a target. NaPi2b is a multi-transmembrane, sodium-dependent phosphate transporter, which is expressed in human lung, ovarian, and thyroid cancers. Although NaPi2b is upregulated in high growth rate environments (such as tumors), there are several families of phosphate transporters, providing a high degree of control and redundancy to phosphate transport. Because of the phosphate transport protein redundancy, NaPi2b may not be a critical to cell function or growth. One KOL we spoke to believes that for an antigen to go from a good to a great target, it needs to have a function. If an antigen just happens to be on the cell surface without a function, these antigens can just be downregulated after targeting (or the low expressing cells are selected for), leaving you with a cell population that doesn't express the antigen any longer. However, another KOL we spoke with (who has extensive experience with ADCs) doesn't think the target being critical for cancer cell growth is a big issue at all. His view is that since this is an ADC system, they are only using NaPi2b as a target to get the drug/toxin to the tumor cell. With this view, function may not necessarily be as important for an ADC system.

In their publication, Genentech hypothesized that downregulation of NaPi2b could be a resistance mechanism for lifastuzumab vedotin, but no evidence was given in support. Related to the bear thesis that NaPi2b may not be a critical protein for cell survival is the belief that NaPi2b target downregulation could be a resistance mechanism for an ADC targeting approach. Generally, if a protein is not critical for cell survival, selective pressures are more likely to cause the target to be (1) downregulated or (2) low expressing clones will be selected for (high expressers killed). The reality, however, is that these selective pressures could apply to any target. Experimental data of target expression over time (longitudinal expression) is the best way to determine how a target reacts to treatment. Unfortunately, at this moment in time we have very limited data to describe the longitudinal expression of NaPi2b. To both our and our KOL's knowledge, Genentech did not present any

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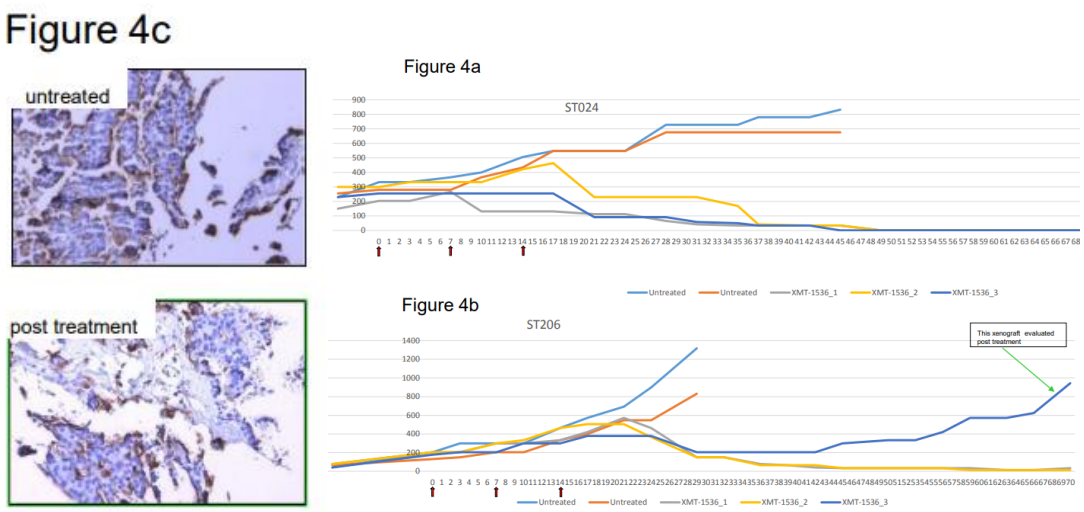
data on the topic. In 2017, MRSN presented one experimental model, ST206, where tissue obtained at the end of an extended time course study was evaluated for target expression in a xenograft that showed an incomplete response. IHC performed on an untreated control xenograft and a xenograft with incomplete response (treated tumor) at the end of study both show NaPi2b expression (4c, below). Of course, this is only one data point and further studies are greatly desired to help understand how expression changes with time and treatment. Unfortunately, with such limited data, the impact of NaPi2b downregulation (or if it even happens at all) on the efficacy and durability of XMT-1536 treatment is unknown.

NaPi2b Target Expression Can Remain Post Treatment



Source: [MRSN 2017 Triple Meeting Poster]

KOL thinks longitudinal expression is the "million dollar question" for ADCs in solid tumors. Based on his knowledge of the literature, our KOL also confirmed that the longitudinal expression of NaPi2b is not known. Yet he sees it as a very important question to answer. And we don't know if this happens *in vivo*, or in patients, or whether it is actual downregulation of target vs. target heterogeneity leading to selection of the lower expression clones. This KOL believes MRSN should do a full IHC analysis of newly diagnosed patients to understand the anatomic and topographic expression of NaPi2b. Then compare these samples to recurrent cases to solve the question of longitudinal expression of NaPi2b. This can and should first be done with patients who are not exposed to any NaPi2b therapy. Secondarily, our KOL was also interested in the expansion cohort biopsy samples because patients will be under the selective pressure of a NaPi2b targeting therapy. Overall, his opinion is that characterizing target expression is very important.

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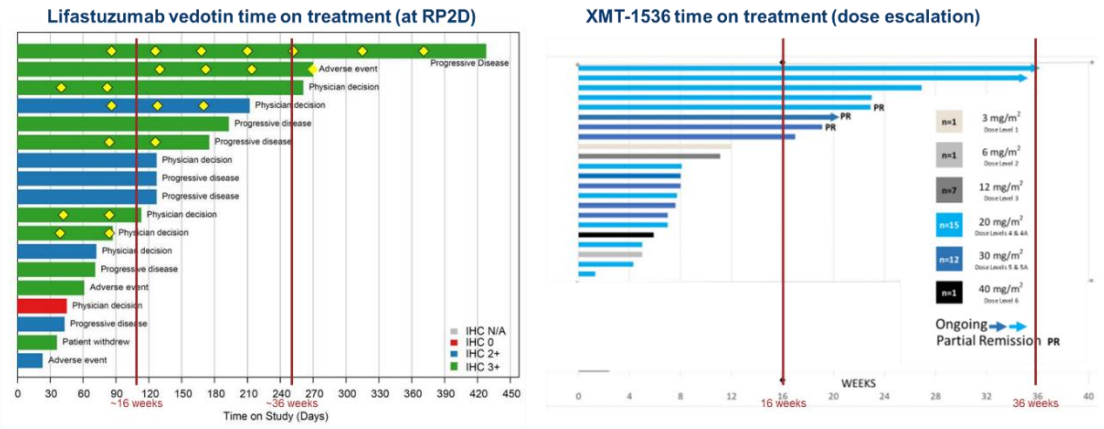
How much of a concern is durability?

Durability will be an important consideration moving forward based on the past failure of Roche's lifastuzumab vedotin. In their end of Phase II publication, Genentech specifically stated that "while the response rate for [lifastuzumab] was promising, response durations were relatively short". Given the durability disappointment with lifastuzumab, durability of response will remain a key question for MRSN to answer. Unfortunately, there are many unanswered questions when it comes to predicting how MRSN could perform with their eventual durability results. What causes the durability concerns? Is it a target problem: does the target need to be vital to ovarian cancer cell survival to result in higher durability, as discussed above? Or was the lifastuzumab durability a platform issue. If so, will MRSN's platform see similar durability concerns, or is the platform differentiated enough to believe they can overcome the issue?

Evaluating the MRSN durability data in comparison to the lifastuzumab data is a useful first step to understand if MRSN is on the right track. Overall, the MRSN duration of treatment data presented at ASCO 2019 trended in the right direction, given the stage of dose escalation. The early duration data compare favorably to the lifastuzumab vedotin data from the Phase I study (in a similar patient population). Also working in MRSN's favor for this comparison is the fact that lifastuzumab patients were already being treated at the RP2D, while MRSN has not yet chosen a RP2D. MRSN has however dose-escalated beyond the dose levels in the ASCO dataset – indicating there could be room for improvement for XMT-1536. While the 1H20 presentation won't have definitive durability data (which we view as important to the story given Roche's past NaPi2b disappointments), we do expect that MRSN will provide a swimmer plot, similar to past publications.

Duration of Treatment of Lifastuzumab vedotin Phase I vs. XMT-1536 Phase I



Source: Genentech Phase I Lifastuzumab Data, ASCO 2014; MRSN ASCO 2019 Poster

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The DAR Plateau effect. Does more payload provide better efficacy?

The "Plateau Effect" is likely real. But that doesn't mean higher DAR is useless. DAR provides another useful parameter to design a successful ADC therapeutic approach. There is a longstanding debate in the ADC field regarding the impact and importance of drug to antibody ratio (DAR) in ADC design. DAR is said to have an impact on many parameters of an ADC, including antitumor efficacy, antibody structure, antibody stability, antigen binding, and therapeutic index (Tang Y, 2017). More specifically, one debate about DAR is whether more payload (higher DAR) leads to better efficacy or whether there is a "plateau effect". The plateau effect suggests that a minimal threshold concentration of intratumoral payload is required to support sustained efficacy and that payload levels delivered in excess of this level will not enhance efficacy. While some debate remains, the reality is that there are numerous publications suggesting the plateau effect is a real phenomenon (Zhang, 2018; Zhang, 2019). Essentially what this means is (all variables being equal: dose, target expression, payload release, etc.), an ADC with higher DAR is unlikely to have enhanced efficacy vs. an ADC with lower DAR if both drugs can reach the threshold concentration for efficacy. In colloquial discussion, however, the plateau effect is commonly misinterpreted. People often speak of the plateau effect as a threshold in the useful number of payloads per antibody. But this is not true. Rather, the plateau refers to a threshold concentration of delivered payload within the tumor that leads to efficacy. Consider a theoretical situation of delivering two ADCs: (1) DAR 4 at 2 mg/kg and (2) DAR 8 at 1 mg/kg. Presuming all other parameters are equal, both systems could deliver the same amount of payload to the tumor, creating the same tumor concentration and same efficacy. Now imagine it turns out that (1) cannot reach a dose of 2 mg/kg. In this case, DAR becomes a useful, engineerable parameter to reach the desired efficacious concentration. While this is a simple example, an ADC is a highly complex system further underlining the usefulness of DAR as an engineerable parameter to help in the design of a successful ADC therapeutic approach.

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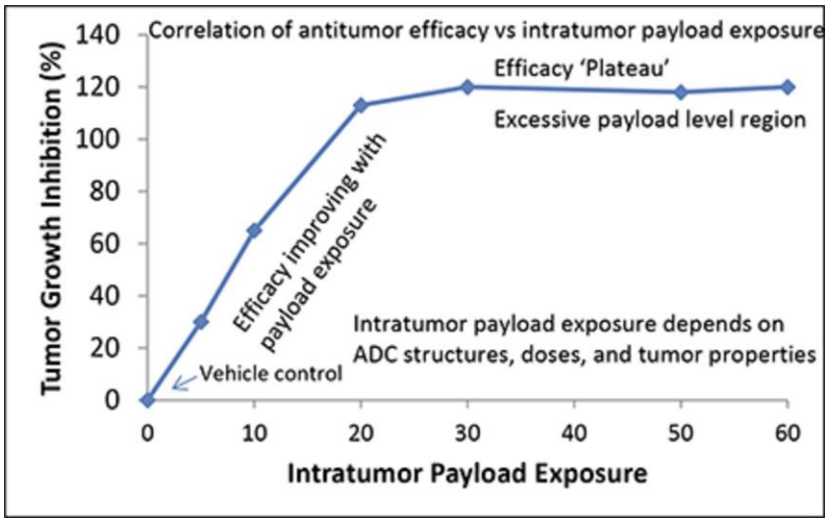
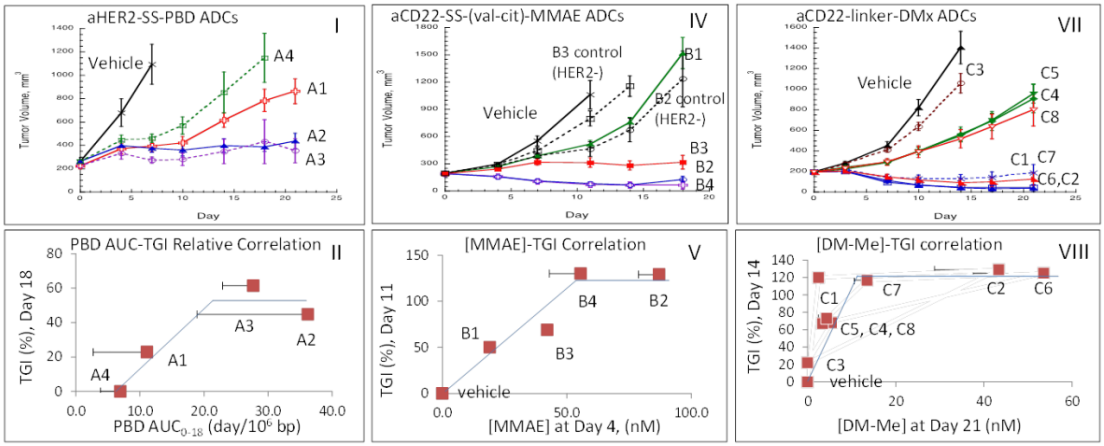
"Plateau effect": Visual Overview



Source: [Zhang D et al., Drug Metab Dispos 2019]

"Plateau effect": threshold concentration of intratumoral payload that leads to sustained efficacy



Source: [Zhang D et al., Drug Metab Dispos 2019]

SCENARIO ANALYSIS AND STOCK SET-UP

Our current price target of $11 reflects a probability-adjusted ~$500M peak US revenue opportunity for XMT-1536. We estimate a peak US probability-unadjusted revenue opportunity of ~$1.4B in 2L-4L platinum-resistant ovarian cancer and ~$600M in 2L-3L NSCLC, to which we apply a probability of success of 30% and 10% respectively for

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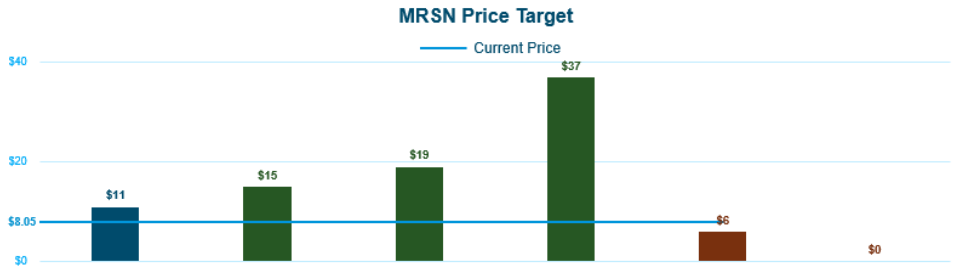
valuation purposes. Additional opportunities in other ovarian cancer indications (e.g., platinum-sensitive disease) and potential combinations with XMT-1536 represent upside to our current estimates. Earlier stage programs such as XMT-1592 (NaPi2b-targeting Dolasynthen ADC), the B7-H4 targeting ADC, and a STING agonist ADC also represent upside to our current estimates.

Potential scenarios following the Phase I data readout are outlined below:

- **Base Case:** 30% POS in prOC, 10% POS in NSCLC
- **Scenario 1:** Bull case prOC data (+10% POS in prOC to 40% overall)
- **Scenario 2:** Best case prOC data (+20% POS in prOC to 50% overall)
- **Scenario 3:** Future best case prOC scenario (100% POS in prOC)
- **Scenario 4:** Bear case prOC data (-15% POS in prOC to 15% overall)
- **Scenario 5:** Worst case data scenario (0% POS for prOC and NSCLC)

Impact of Various Data Scenarios on MRSN Price Target



Assumption	Base (1)	2	3	4	5	6
prOC (POS)	30%	40%	50%	100%	15%	0%
NSCLC (POS)	10%	10%	10%	10%	10%	0%

Source: SVB Leerink MRSN Company Model; MRSN closing price at 1/17/19

APPENDIX A: OVARIAN CANCER TREATMENT LANDSCAPE

Ovarian cancer is the second most common gynecologic malignancy (~22,000 new cases per year, SEER) and the most common cause of gynecologic cancer death in the United States. Only approximately 25 percent of women will be diagnosed with early stage ovarian cancer, either confined to the ovary (stage I) or confined to the pelvis (stage II). For women with cancer confined to the ovary (IA or IB) and/or well-differentiated (grade 1) tumors, prognosis is excellent with survival of at least 90 percent following surgery alone. For all others, adjuvant chemotherapy is recommended. Approximately 75 percent of women have stage III (disease that has spread throughout the peritoneal cavity or that involves lymph nodes) or stage IV (disease spread to more distant sites) disease at diagnosis. Primary surgical cytoreduction followed by systemic platinum-based chemotherapy is the

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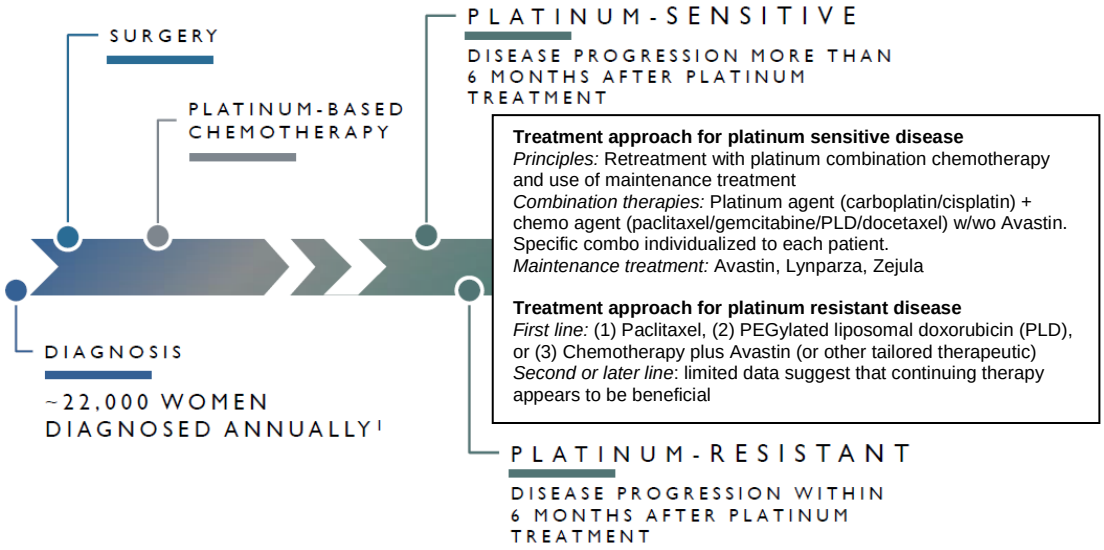
preferred initial management for women with stage III or IV ovarian cancer. How a patient responds to platinum-based chemotherapy will define the next stages of treatment.

US Ovarian Cancer Treatment Landscape in 2018



Source: IMGN Corporate Slides, UpToDate, [1]SEER (Surveillance, Epidemiology, and End Results)

Overview of the treatment approach for platinum-resistant ovarian cancer. Platinum-resistant disease is typically defined as disease progression within six months after platinum treatment. Outcomes for these patients remain poor, with low response rates to further chemotherapy (~15-20%), progression-free survival (PFS) of 3-4 months, and a median overall survival rate of less than a year. In addition, this treatment approach is associated with additional, cumulative toxicities and limited tolerability for patients. Hence, treatments should aim to maximize quality of life while attempting to control disease. Though there are a number of active treatment options available for women with platinum-resistant ovarian cancer, there is no consensus on the ideal treatment. For patients who have not previously been treated specifically for platinum-resistant disease, the preferred first-line treatment option is single-agent paclitaxel, especially in those patients who have not previously been treated with paclitaxel for recurrent disease. For patients who progressed on or are not candidates for single-agent paclitaxel, PEGylated liposomal doxorubicin (PLD) is used because of its schedule and lack of typical side effects associated with chemotherapy. For appropriately selected women with platinum-resistant recurrent disease, single-agent chemotherapy plus Avastin (bevacizumab) can be administered.

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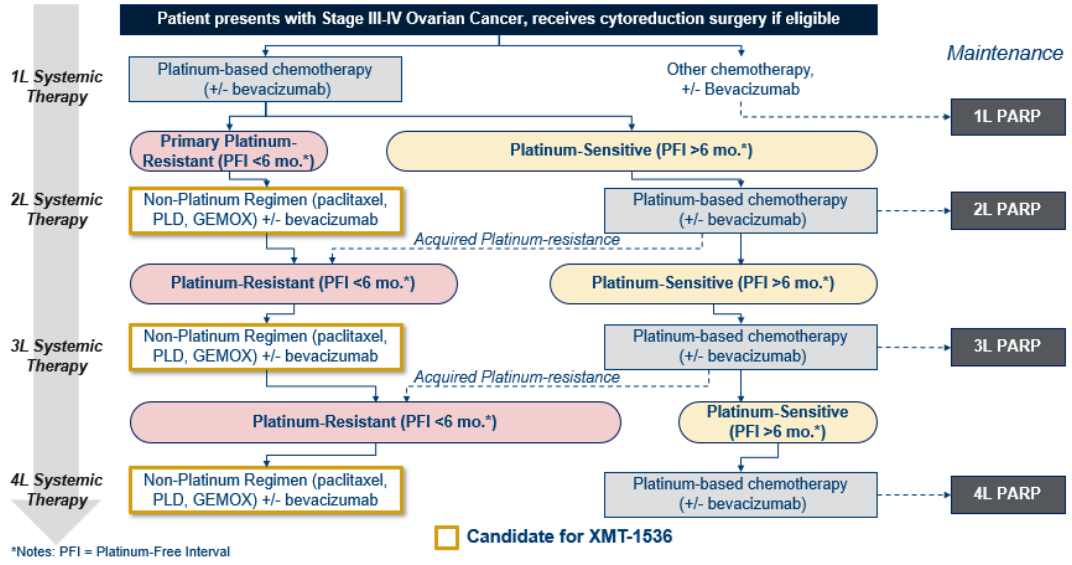
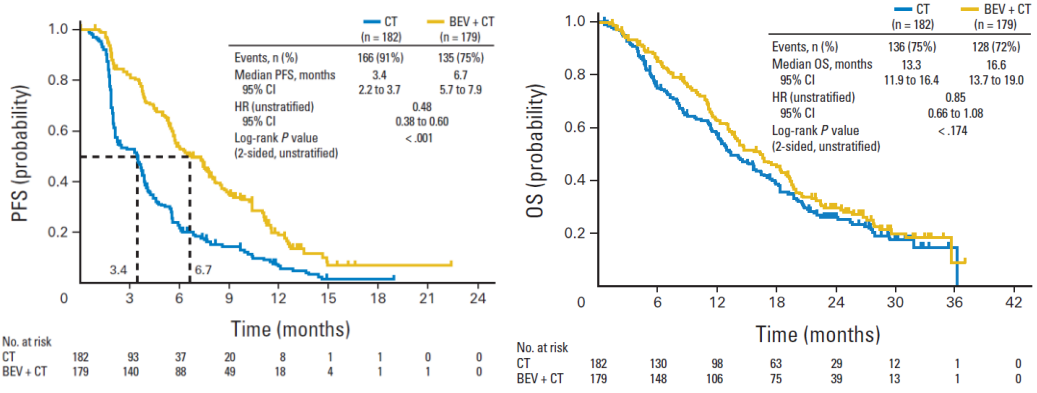
Treatment Options for prOC Patients



Source: Adapted from NCCN Clinical Guidelines 2018. Leerink Interviews with MEDACorp KOLs

The AURELIA trial showed an advantage of combining chemotherapy with Avastin, raising median PFS from ~4 months to ~7 months, but did not show improved overall survival. However, not all patients are suited for this treatment since you must have received two or fewer prior treatment regimens, have not received Avastin previously, and have no history of a bowel obstruction within six months. Avastin combined with chemotherapy also has high levels of toxicity which limits its use. For patients who relapse after first-line platinum resistant treatment or subsequent treatment and desire further therapy, limited data suggest that continuing therapy appears to be beneficial (Hanker, LC, et al., Ann Oncol., 2012).

AURELIA trial PFS and OS of Avastin plus chemotherapy (Phase III)



Source: J Clin Oncol. 2014; 32:1302-1308

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Results of IMGN's (MP) Phase III FORWARD-I study provide additional single-agent chemo benchmarks in prOC (1-3 priors). FORWARD-I trial was a Phase III trial in 366 platinum-resistant ovarian cancer patients (with medium-high FRα expression), randomized 2:1 to receive either mirvetuximab soravtansine or investigator's choice chemotherapy. IMGN presented detailed results and exploratory analyses from the FORWARD-I study at the European Society for Medical Oncology (ESMO) 2019 conference (LINK). The investigator's choice (IC) control arm included paclitaxel, PLD, or topotecan. In the ITT population (medium-high FRα expression) the ORR was 22% for mirvetuximab soravtansine and 12% for IC chemotherapy. In the FRα-high population, the ORR was 22% mirvetuximab and 10% for IC chemotherapy. Overall, the response rates for the IC chemo arm are consistent with the control arm of the AURELIA study (12% ORR) and continue to support the benchmark of 12% ORR for single-agent chemo in prOC patients with 1-3 prior treatments.

Efficacy Results from Phase III FORWARD-I Study

ITT Population

Endpoint	Treatment effect size [Mirv (n=248) vs IC Chemo (n=118)]	P value*
PFS by BIRC (mo.)	HR: 0.981 (0.734, 1.310) mPFS: 4.1 vs 4.4	0.897^
ORR by BIRC 95% CIs	22% vs 12% (17%, 28%) vs (7%, 19%)	0.015
OS (August 2019)	HR: 0.846 (0.625, 1.145) mOS: 15.6 vs 13.9	0.278
PRO†	32% vs 14%	0.011

FRα High Population

Endpoint	Treatment effect size [Mirv (n=147) vs IC Chemo (n=71)]	P value*
PFS by BIRC (mo.)	HR: 0.693 (0.480, 1.000) mPFS: 4.8 vs 3.3	0.049^
ORR by BIRC 95% CIs	24% vs 10% (17%, 32%) vs (4%, 19%)	0.014
OS (August 2019)	HR: 0.678 (0.460, 0.999) mOS: 16.4 vs 12.0	0.048
PRO†	28% vs 13%	0.096

*Nominal p-value
^Not significant based on Hochberg Procedure
†≥15-point improvement in the EORTC QLQ-OV28 Abdominal/GI Symptom Subscale

Source: IMGN Presentation at ESMO 2019

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Platinum Resistant Ovarian Cancer Benchmarks by Line of Therapy (Phase II)

Platinum-Resistant Benchmarks by Line of Therapy (# Prior Treatments)							
Study	McGonigle (2011)[1]	Tillmanns (2013)[2]	Liu (2015)[3]	Verschraegen (2012)[4]	McGonigle (2011)[1]	Cannistra (2007)[5]	Barber (2013)
% Platinum-Resistant	100%	100%	100%	100%	100%	100%	100%
Prior Systemic Treatments*	**1**	**1.8**	**1.8**	**2**	**2**	**2.5**	**6.5**
Regimen	Bev + Topotecan	Bev + paclitaxel	Bev + irinotecan	Bev + PLD	Bev + Topotecan	Bev monotherapy	Bev + cyclophosphamide
Phase	II	II	II	II	II	II	Review
N	21	48	52	43	19	44	66
ORR (RECIST, unless otherwise noted)	23.8%	50.0%	42.3%	30.0%	26.3%	15.9%	42.4%
mPFS (mo.)	2.8	8.1	8.0	6.6	10.9	4.4	5.0

1-2 Prior Treatments	
ORR (RECIST, unless otherwise noted)	42.1%
mPFS (mo.)	7.13

2+ Prior Treatments	
ORR (RECIST, unless otherwise noted)	23.5%
mPFS (mo.)	6.46

Source: Cancer. 2011 Aug 15; 117(16):3731-40; 2. Gynecol Oncol. 2013 Feb;128(2):221-8; 3. Cancer Chemother Pharmacol. 2015; 75(3): 645–651; 4. Ann Oncol. 2012 Dec;23(12):3104-10; 5. J Clin Oncol. 2007 Nov 20;25(33):5180-6; 6. J Gynecol Oncol. 2013 Jul;24(3):258-64.

Impact of 2nd to 6th Line of Therapy on Survival

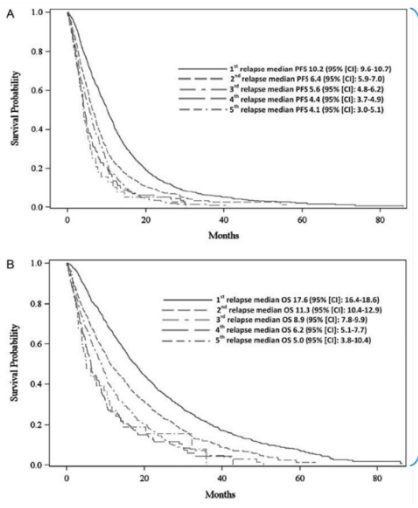

Figure 2. Kaplan-Meier analyses of progression-free survival (PFS) and overall survival (OS) were carried out in the whole patients' cohort depending on subsequent relapse. Panel A shows PFS after relapse 1-5. Panel B shows OS after relapse 1-5.

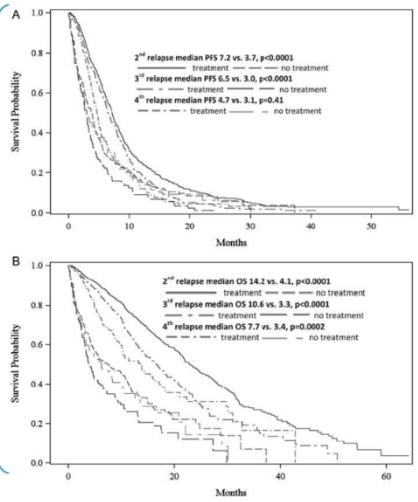

Figure 3. Kaplan-Meier analyses of progression-free survival (PFS) and overall survival (OS) depending on subsequent relapse according to treatment decision at relapse. Panel A shows PFS after relapse 2-4 stratified by the patients who did receive relapse treatment and the patients who did not receive treatment anymore. Panel B shows corresponding OS.

Source: Hanker LC, et al., Ann Oncol 2012

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Ovarian Cancer Competitive Landscape

Clinical Trial	Drug Name	Company	Ticker	Status	Molecule	Indication	Target / Mechanism
Approved	Avastin	Roche Holding AG	RHHBY	III	mAb	1L	VEGF
Approved	Rubraca	Clovis Oncology, Inc.	CLVS	III	SM	1L, PS	PARP
Approved	Lynparza	AstraZeneca PLC	AZN	III	SM	1L, PS	PARP
Approved	Zejula	GlaxoSmithKline	GSK	III	SM	1L, PS	PARP
NCT02289950	Farletuzumab	Eisai Co., Ltd.	ESALY	III	mAb	PS	FRα
Multiple	Veliparib	AbbVie Inc.	ABBV	III	SM	1L	PARP
Multiple	Mirvetuximab Soravtansine	ImmunoGen, Inc.	IMGN	III	mAb	PS, PR	FRα
NCT03398655	VB-111	VBL Therapeutics	VBLT	III	*	PR	TNFα
Multiple	Keytruda	Merck & Co., Inc.	MRK	III	mAb	1L, PR	PD-1/L1
Multiple	Opdivo	Bristol-Myers Squibb	BMY	III	mAb	1L, PS	PD-1/L1
Multiple	Tecentriq	Roche Holding AG	RHHBY	III	mAb	PR, PS	PD-1/L1
Multiple	Dostarlimab	GlaxoSmithKline plc	GSK	III	mAb	1L, PS	PD-1/L1
Multiple	Bavencio	Pfizer	PFE	III	mAb	1L, PR	PD-1/L1
Multiple	Recentin	AstraZeneca PLC	AZN	II/III	SM	PS	VEGFR
NCT02490488	Masitinib	AB Science S.A.	AB:FP	II/III	SM	PR	FGFR/PDGFR
NCT03100006	Oregovomab	Quest PharmaTech	QPT:CN	IIb	mAb	PR	MUC-16
Multiple	Yervoy	Bristol-Myers Squibb	BMY	II	mAb	PS	CTLA-4
NCT01199263	Pelareorep	Oncolytics Biotech	ONCY	II	Viral	PR	Onc. Virus
Multiple	AZD1775	AstraZeneca PLC	AZN	II	SM	PR	TKI
NCT03029403	DPX-Survivac	IMV Inc.	IMV	II	Peptide	PR	Survivin
NCT03587311	BAY 94-9343	NCI, Bayer AG	BAYRY	II	mAb	PR	Mesothelin ADC
NCT03639246	AVB-500	Aravive Inc.	ARAV	II	Protein	PR	Axl TKI
NCT03395080	DKN-01	Leap Therapeutics	LPTX	II	mAb	-	DKK-1
NCT03878849	2X-121	Oncology Venture	OV.ST	II	SM	PS	PARP
NCT03776812	Relacorilant	Corcept Therapeutics	CORT	II	SM	PR	GR
NCT03657043	Tisotumab Vedotin	Genmab A/S	GMAB	II	mAb	-	Tissue Factor ADC
NCT03933761	Pamiparib	Merck KGaA	MKGAY	II	SM	PS	PARP
NCT03734692	Ampligen	AIM ImmunoTech, Inc.	AIM	I/II	*	PR	TLR3
NCT02901899	Guadecitabine	Otsuka Holdings Co	4768:JP	I/II	SM	-	DNMT
NCT02963831	ONCOS-102	Targovax AS	TRVX:NO	I/II	Viral	PR	Onc. Virus
Multiple	Imfinzi	AstraZeneca PLC	AZN	I/II	mAb	-	PD-1/L1
NCT03992131	Lucitanib	Clovis Oncology, Inc.	CLVS	I/II	SM	-	VEGFR
Multiple	Tremelimumab	AstraZeneca PLC	AZN	I/II	mAb	-	CTLA-4
NCT03761914	Zeltherva	SELLAS Life Sciences	SLS	I/II	Peptide	-	WT1
NCT02759588	GL-ONC1	Genelux Corporation		I/II	Viral	PR	-
NCT01690468	PTX-200	Prescient Therapeutics	ASX:PTX	I/II	SM	PR	PI3K/AKT
NCT03564340	REGN4018	Regeneron	REGN	I/II	mAb	PR	MUC-16
NCT03634150	Nerofe	Immune System Key		I/II	Peptide	PR	TCApF
NCT01631552	Sacituzumab Govitecan	Immunomedics	IMMU	I/II	mAb	PR	Trop-2 ADC

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NCT03907852	TC-210	TCR2 Therapeutics	TCRR	I/II	Cellular	PR	TRuC Mesothlin
NCT02269293	Xpovio	Karyopharm	KPTI	I	SM	PR, PS	XPO1
NCT03319628	XMT-1536	Mersana Therapeutics	MRSN	I	mAb	PR	NaPi2b ADC
NCT02892123	ZW25	Zymeworks	ZYME	I	mAb	PR	HER2
NCT03784677	SOR-C13	Soricimed Biopharma		I	Peptide	PR	TRPV6
NCT03213964	FATE-NK100	Fate Therapeutics	FATE	I	Cellular	PR	-
NCT03748186	STRO-002	Sutro Biopharma	STRO	I	mAb	PR	FRα ADC
NCT02978755	GM102	GamaMabs Pharma		I	mAb	PR	Anti-Mullerian HR
NCT03695380	Cotellic	Roche Holding AG	RHHBY	I	SM	PS	MEK
NCT03608618	MCY-M11	MaxCyte, Inc.	MXCT:LN	I	Cellular	PR	Mesothelin
	IGEM-F	IGEM Therapeutics		I	mAb	-	FRα
NCT03907527	PRGN-3005	Precigen, Inc.	PGEN	I	Cellular	PR	IL-15/R
NCT03839524	TG4050	Transgene S.A.	TNG:FP	I	Viral	PS	Onc. Virus
NCT01623349	Piqray	Novartis AG	NVS	I	SM	PR	p110α
	BNT115	BioNTech AG	BNTX	I	mRNA	PR	-
	CT900	Carrick Therapeutics		I	SM	PR	FRα
					* Other Nucleic Acid		

Source: SVB Leerink Research; BioMedTracker Database, accessed 1/16/20

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INVESTMENT THESIS

MRSN has developed a proprietary next-generation antibody-drug conjugate (ADC) platform which holds the promise of generating ADCs with differentiated properties compared to currently available agents. XMT-1536 is currently being evaluated in a Phase I dose escalation study in platinum-resistant ovarian cancer, non-small cell lung cancer, and other tumors likely to express NaPi2b. While there are risks associated with this early stage program, we view the risk/reward as attractive with NaPi2b representing a potentially significant commercial opportunity with expression levels of ~60%-90% in large oncology indications such as lung and ovarian cancer. We continue to view MRSN as being led by an experienced management team and see the potential for long-term appreciation as XMT-1536 and MRSN's platform become de-risked over 2020 clinical updates.

VALUATION

Our price target for MRSN is $11/share based on a 50%/50% blend of a revenue multiple analysis and DCF analysis. We assign value to probability-weighted sales and royalties of XMT-1536 (30% probability of success in ovarian and 10% probability of success in lung cancer). Additional opportunities in other ovarian cancer indications (e.g., platinum-sensitive disease) and potential combinations with XMT-1536 represent upside to our current estimates. Earlier stage programs such as XMT-1592 (NaPi2b-targeting Dolasynthen ADC), the B7-H4 targeting ADC, and a STING agonist ADC also represent upside to our current estimates. We use a 15% discount rate and a 0% terminal growth rate.

RISKS TO VALUATION

MRSN's pipeline programs face clinical and regulatory development risks, as well as commercial and intellectual property risks. MRSN also faces execution risk and financial risk. MRSN may have additional financing needs before turning cash flow positive.

FINANCIAL MODEL

In our financial model we have updated our estimates for shares outstanding in 4Q19 and our 2019 and 2020 annual EPS estimates to bring them in line with GAAP standards. Our estimates for net income in 2019 and 2020 remain unchanged.

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MRSN P&L (in MM, except per share data)	2017A	2018A	1Q19A	2Q19A	3Q19A	4Q19E	2019E	1Q20E	2Q20E	3Q20E	4Q20E	2020E	2021E
Product sales (p/w)	-	-	-	-	-	-	-	-	-	-	-	-	-
Royalties (p/w)	-	-	-	-	-	-	-	-	-	-	-	-	-
License, collaboration and other revenue	17.5	10.6	41.0	0.2	0.8	1.0	43.1	0.6	0.6	0.6	0.6	2.5	2.1
Total Revenue	**17.5**	**10.6**	**41.0**	**0.2**	**0.8**	**1.0**	**43.1**	**0.6**	**0.6**	**0.6**	**0.6**	**2.5**	**2.1**
COGS	-	-	-	-	-	-	-	-	-	-	-	-	-
R&D	46.7	59.9	15.1	13.8	13.7	14.0	56.6	14.0	15.0	16.0	17.0	62.0	68.2
SG&A	10.5	16.3	4.4	4.2	4.4	4.5	17.6	4.5	4.5	4.5	4.5	18.0	19.8
Total Operating Expense	57.2	76.2	19.6	18.0	18.1	18.5	74.2	18.5	19.5	20.5	21.5	80.0	88.0
Operating Income (Loss)	**(39.6)**	**(65.7)**	**21.4**	**(17.8)**	**(17.3)**	**(17.5)**	**(31.1)**	**(17.9)**	**(18.9)**	**(19.9)**	**(20.9)**	**(77.5)**	**(85.9)**
Total other, net	0.9	1.4	0.5	0.7	0.5	(0.1)	1.6	(0.1)	(0.1)	(0.0)	(0.0)	(0.2)	(0.1)
Net Income before Taxes	**(38.7)**	**(64.3)**	**21.9**	**(17.1)**	**(16.8)**	**(17.6)**	**(29.5)**	**(17.9)**	**(18.9)**	**(19.9)**	**(20.9)**	**(77.7)**	**(85.9)**
Tax expense (benefit)	-	-	-	-	-	-	-	-	-	-	-	-	-
Net Income (Loss)	**(38.7)**	**(64.3)**	**21.9**	**(17.1)**	**(16.8)**	**(17.6)**	**(29.5)**	**(17.9)**	**(18.9)**	**(19.9)**	**(20.9)**	**(77.7)**	**(85.9)**
Diluted EPS	**(3.22)**	**(2.79)**	**0.70**	**(0.36)**	**(0.35)**	**(0.39)**	**(0.69)**	**(0.40)**	**(0.42)**	**(0.44)**	**(0.39)**	**(1.63)**	**(1.31)**
Basic shares outstanding (MM)	12.0	23.0	30.3	47.7	47.8	45.3	42.8	45.3	45.3	45.3	54.3	47.6	65.5
Dilutive securities	4.8	4.8	4.8	4.8	4.8	4.8	4.8	4.8	4.8	4.8	4.8	4.8	4.8
Diluted Shares Outstanding (MM)	12.0	23.0	31.5	47.7	47.8	45.3	42.8	45.3	45.3	45.3	54.3	47.6	65.5

MRSN BS/CFS (in MM $)	2017A	2018A	1Q19A	2Q19A	3Q19A	4Q19E	2019E	1Q20E	2Q20E	3Q19E	4Q20E	2020E	2021E
Cash & equivalents	**125.2**	**70.1**	**137.3**	**128.2**	**112.0**	**96.3**	**96.3**	**80.5**	**63.9**	**46.1**	**127.1**	**127.1**	**170.9**
Debt	-	-	-	5.0	5.0	5.0	5.0	5.0	5.0	4.8	4.3	4.3	2.3

Change in Cash	**(73.7)**	**33.0**	**77.6**	**(21.1)**	**(58.1)**	**(15.7)**	**(17.2)**	**(15.8)**	**(16.7)**	**(17.7)**	**81.0**	**30.8**	**43.7**
Operating Cash Flows	**(42.7)**	**(55.2)**	**(24.7)**	**(14.2)**	**(16.3)**	**(15.7)**	**(70.9)**	**(15.8)**	**(16.7)**	**(17.6)**	**(18.5)**	**(68.5)**	**(79.3)**
Net Income	(38.7)	(64.3)	21.9	(17.1)	(16.8)	(17.6)	(29.5)	(17.9)	(18.9)	(19.9)	(20.9)	(77.7)	(85.9)
Stock-Based Compensation	1.4	3.9	1.2	1.2	1.3	1.9	5.5	1.9	2.0	2.1	2.2	8.0	8.8
D&A	0.6	1.0	0.3	0.3	0.2	-	0.9	0.3	0.3	0.3	0.3	1.2	-
adjustments	(9.9)	(6.7)	-	-	-	-	-	-	-	-	-	-	(2.1)
Other	3.9	10.9	(48.1)	1.4	(1.0)	-	(47.7)	-	-	-	-	-	-
Investing Cash Flows	**(99.6)**	**87.2**	**10.1**	**(12.2)**	**(41.8)**	**-**	**(43.8)**	**-**	**-**	**-**	**-**	**-**	**-**
CapEx	(1.1)	(1.4)	(0.4)	(0.2)	(0.0)	-	(0.6)	-	-	-	-	-	-
Other	(98.5)	88.6	10.5	(11.9)	(41.7)	-	(43.2)	-	-	-	-	-	-
Financing Cash Flows	**68.6**	**1.1**	**92.2**	**5.3**	**(0.0)**	**-**	**97.5**	**-**	**-**	**(0.2)**	**99.5**	**99.3**	**123.0**
Equity Raise (Buyback)	68.1	-	92.2	-	-	-	92.2	-	-	-	100.0	**100.0**	125.0
Debt Issuance (Retirement)	-	-	-	5.0	-	-	5.0	-	-	(0.2)	(0.5)	**(0.7)**	(2.0)
Other	0.5	1.1	0.0	0.3	(0.0)	-	0.3	-	-	-	-	-	-

Source: SEC Filings and SVB Leerink Estimates

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MRSN, Pipeline and Upcoming Events							
Program	**Partner**	**Mechanism**	**Indication**	**Status**	**Events**	**Timing**	**Clinicaltrials.gov**
XMT1536	Proprietary	NaPi2b	Ovarian, NSCLC, Papillary RCC, Papillary Thyroid, Salivary Gland, Endometrial	Phase I dose escalation (ongoing)	Update on dose-escalation data	1H20	NCT03319628
			Platinum resistant ovarian cancer & NSCLC adenocarcinoma	Phase I expansion cohorts at 36 and 43 mg/m2	Interim expansion cohort data	1H20	NCT03319628
					More mature dataset	2H20	NCT03319628
XMT-1592	Proprietary	NaPi2b	Oncology	Preclinical	Initiate Phase I dose escalation	1H20	-
B7-H4 ADC Candidate	Proprietary	B7-H4	Oncology	Preclinical	Disclose candidate and supporting data	2H20	-
STING agonist ADC Candidate	Proprietary	STING	Oncology	Preclinical	Disclose candidate and supporting data	2H20	-
				Additional preclinical data	Disclose additional preclinical data at scientific meetings throughout 2020	2020	-
Immunosynthen Platform	Proprietary	-	-	Preclinical	-	-	-
6 undisclosed programs	Merck KGaA	NA	-	Preclinical	-	-	-
ASN004	Asana/ENDP	5T4	-	Preclinical	-	-	-

Source: SVB Leerink Research; Company Updates

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MRSN DCF Analysis	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E	2034E	2035E	2036E	2037E	2038E	2039E	2040E	TV
CFO	(55.2)	(70.9)	(68.5)	(79.3)	(86.2)	20.6	46.6	74.2	129.3	155.5	181.4	203.4	216.2	226.9	238.1	249.8	262.1	275.0	288.6	302.8	200.5	117.6	65.2	
FCF	(56.6)	(71.5)	(68.5)	(79.3)	(86.2)	20.6	46.6	74.2	129.3	155.5	181.4	203.4	216.2	226.9	238.1	249.8	262.1	275.0	288.6	302.8	200.5	117.6	65.2	434.6
Discount Periods	0	0.0	0.0	1.0	2.0	3.0	4.0	5.0	6.0	7.0	8.0	9.0	10.0	11.0	12.0	13.0	14.0	15.0	16.0	17.0	18.0	19.0	20.0	20.0
NPV FCFF	-	(71.5)	(68.5)	(69.0)	(65.2)	13.6	26.6	36.9	55.9	58.5	59.3	57.8	53.4	48.8	44.5	40.6	37.0	33.8	30.8	28.1	16.2	8.3	4.0	26.6

SUM NPV ($MM)	$ 478
Net Cash YE20 ($MM)	$ 23
Valuation ($MM)	$ 501
MRSN DCF Valuation ($/share)	**10**

Discount Rate	15%
TG	0%
Diluted Shares Outstanding (MM)	50

Source: SVB Leerink Estimates; Figures in $MM, except per share data

MRSN Multiples Valuation ($/share)	**12**

MRSN Blended PT ($/share)	**11**

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Discounted Sales Multiple	2030E Sales	2030E Royalty	Sales Multiple	Royalty Multiple	Periods	DR	NPV	POS	P/W NPV	NPV per share
XMT1536 NSCLC	455	91	5	10	10	15%	787	10%	79	2
XMT1536 2L-4L Platinum-Resistant Ovarian	990	198	5	10	10	15%	1,713	30%	514	10
EV									592	12
Cash									23	0
Total									**615**	**12**

Source: SVB Leerink Research; Numbers in $MM, except per share data

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Disclosures Appendix

Analyst Certification

I, Jonathan Chang, Ph.D., CFA, certify that the views expressed in this report accurately reflect my views and that no part of my compensation was, is, or will be directly related to the specific recommendation or views contained in this report.

Distribution of Ratings/Investment Banking Services (IB) as of 12/31/19

Rating	Count	Percent	IB Serv./Past 12 Mos. Count	IB Serv./Past 12 Mos. Percent
BUY [OP]	153	73.9	59	38.6
HOLD [MP]	53	25.6	4	7.5
SELL [UP]	1	0.5	0	0.0

Explanation of Ratings

Outperform (Buy): We expect this stock to outperform its benchmark over the next 12 months.

Market Perform (Hold/Neutral): We expect this stock to perform in line with its benchmark over the next 12 months.

Underperform (Sell): We expect this stock to underperform its benchmark over the next 12 months.

The degree of outperformance or underperformance required to warrant an Outperform or an Underperform rating should be commensurate with the risk profile of the company.

For the purposes of these definitions the relevant benchmark will be the S&P 600® Health Care Index for issuers with a market capitalization of less than $2 billion and the S&P 500® Health Care Index for issuers with a market capitalization over $2 billion.

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