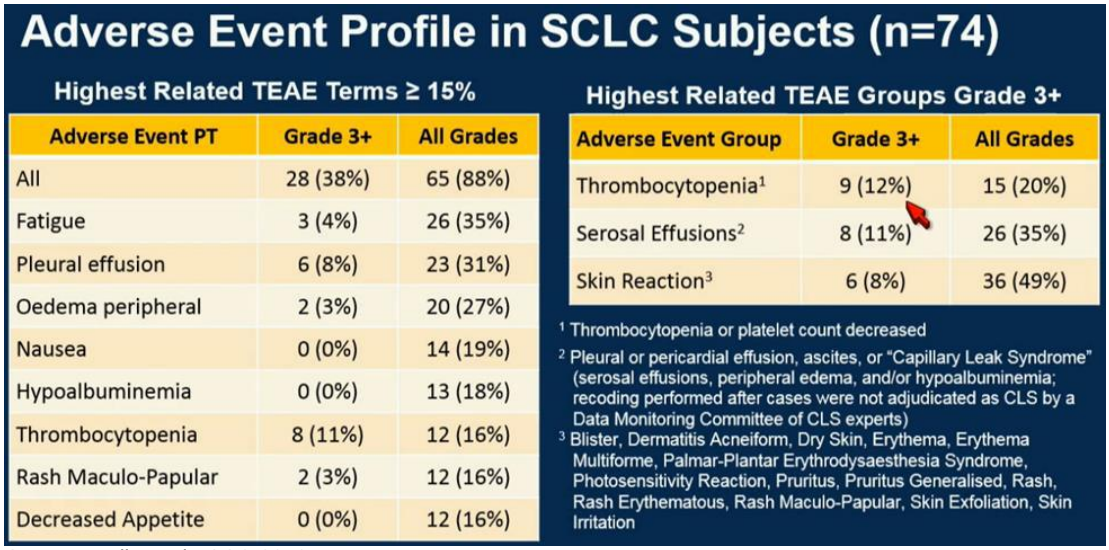
Exhibit 15: Rova-T phase I adverse events



Adverse Event Profile in SCLC Subjects (n=74)

Highest Related TEAE Terms ≥ 15%

Adverse Event PT	Grade 3+	All Grades
All	28 (38%)	65 (88%)
Fatigue	3 (4%)	26 (35%)
Pleural effusion	6 (8%)	23 (31%)
Oedema peripheral	2 (3%)	20 (27%)
Nausea	0 (0%)	14 (19%)
Hypoalbuminemia	0 (0%)	13 (18%)
Thrombocytopenia	8 (11%)	12 (16%)
Rash Maculo-Papular	2 (3%)	12 (16%)
Decreased Appetite	0 (0%)	12 (16%)

Highest Related TEAE Groups Grade 3+

Adverse Event Group	Grade 3+	All Grades
Thrombocytopenia[1]	9 (12%)	15 (20%)
Serosal Effusions[2]	8 (11%)	26 (35%)
Skin Reaction[3]	6 (8%)	36 (49%)

[1] Thrombocytopenia or platelet count decreased

[2] Pleural or pericardial effusion, ascites, or "Capillary Leak Syndrome" (serosal effusions, peripheral edema, and/or hypoalbuminemia; recoding performed after cases were not adjudicated as CLS by a Data Monitoring Committee of CLS experts)

[3] Blister, Dermatitis Acneiform, Dry Skin, Erythema, Erythema Multiforme, Palmar-Plantar Erythrodysaesthesia Syndrome, Photosensitivity Reaction, Pruritus, Pruritus Generalised, Rash, Rash Erythematous, Rash Maculo-Papular, Skin Exfoliation, Skin Irritation

Source: Rudin et al ASCO 2016

AbbVie's JAKi Upadacitinib Still Has Risk

AbbVie's most important pipeline asset is upadacitinib, a JAK1-specific inhibitor for inflammatory diseases. We forecast 2025E global revenues of $2.4bn ($3.7bn risk-unadjusted), consensus forecasts 2025E global revenues of $2.6bn, and AbbVie guided to sales >$6.5bn in 2025. AbbVie has press released and presented successful phase III results from 3 rheumatoid arthritis trials, and the efficacy of the drug has been consistent and impressive. However, the reported 3 venous thromboembolic events (VTEs), including pulmonary emboli (PEs) and deep vein thromboses (DVTs), in the placebo-controlled portion of these phase III trials remains a concern, particularly in the context of Lilly's delayed approval for baricitinib and certain disclosures about upadacitinib' s relative JAK1 vs JAK2 specificity. There were 2 further VTEs reported in the published upadacitinib phase II results, and 4 more VTEs in the crossover (i.e. non placebo-controlled) portion of the SELECT-BEYOND phase III trial from week 12 to week 24. This has been a concern for investors since Lilly received a complete response letter for baricitinib from the FDA on the basis of 5 VTEs, which translated into an event rate of 4.6 per 1,000 patient years of exposure. The clinical literature suggests that an event rate of 6.1 per 1,000 patient years is typical for rheumatoid arthritis patients, which would appear to be consistent with the observed rate in the baricitinib studies. Nevertheless, the FDA appears to have had concerns that led to the complete response letter and delayed potential approval. Our recent research into the safety of the entire JAK class, including the post approval reporting for Pfizer's (PFE, MP) Xeljanz, did not suggest that JAK inhibition led to more VTEs than would be expected in the RA population (see our note here: "Ain't Worried About JAK...Safety! AbbVie's '494 Profile So Far Ok").

However, AbbVie still has to present and publish the full results for 3 phase III rheumatoid arthritis trials in 3,000 patients, and any heightened safety signal will put at risk the multi-billion peak consensus forecasts and management's guidance. As we have pointed out previously, even 1 or 2 VTE events could result in a change in the event rate, and could cross the threshold of regulatory concern. We still believe upadacitinib is a very promising drug, but we also believe it has more risk for labelling restrictions than its direct rival, Gilead/Galapagos' filgotinib. At last

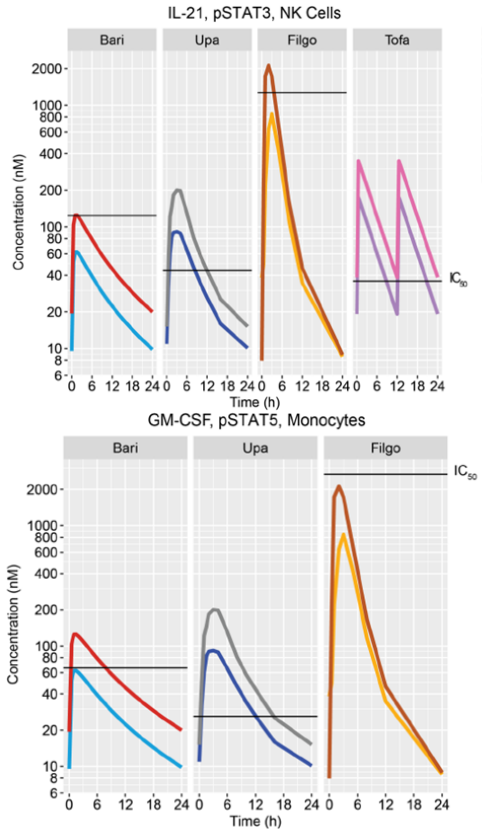
public disclosure, filgotinib had only had 3 cases of VTE events in the entire development experience with the drug in 1,900 patients (event rate of 1.6 per 1,000 patient years), although this has largely been an inflammatory bowel disease population with lower baseline VTE risk, and no phase III rheumatoid arthritis data has been released to date. Filgotinib has also consistently lowered platelet levels, rather than increased them, whereas the effect of upadacitinib on platelets has been mixed, with lower levels at lower doses and then flat to slightly increased levels at higher doses. There are no publications that thoroughly explore the effects of upadacitinib on platelets in human studies, but AbbVie's management has publicly stated that the drug led to "modest decreases" in platelet levels. However, negative hematopoietic effects, namely decreased hemoglobin levels, have been seen with the higher doses being advanced for upadacitinib, and there appears to be a dose-dependent loss of JAK1 selectivity within the therapeutic window. If VTEs are associated with JAK2 inhibition, which baricitinib inhibits to a greater extent than Xeljanz (primarily JAK3), then upadacitinib's suggested in vivo, dose-dependent inhibition of JAK2 may be a liability for the drug either in larger clinical trials or when it is subject to increased regulatory scrutiny.

This lack of JAK1 specificity for upadacitinib has been corroborated by a pre-clinical study funded by the developers of baricitinib, Lilly and Incyte (INCY, OP). AbbVie's internal data suggest that upadacitinib has specificity to JAK1 that is 74-fold higher to JAK2 in cellular assays and 10-fold higher than JAK2 in whole blood assays (IL-6 signaling then GMCSF signaling). In this analysis, peripheral blood mononuclear cells (PBMC's) were collected from healthy donors and used in an assay with the estimated plasma concentrations of each product's phase III dose, or doses, for all the JAK inhibitors in development. This analysis provided drug by drug dose-response curves for stat inhibition as measured by the inhibition of downstream phosphorylation of STAT proteins by JAK1- and JAK2-dependent cytokines. From these dose response curves, the investigators established the half maximum inhibitory concentration (IC50) required for each drug.

STAT5 is phosphorylated by GM-CSF cytokine signaling through JAK2 only, and is a good marker to JAK2 specificity. While the results suggested that upadacitinib is a more potent inhibitor of JAK1 signaling compared to baricitinib and filgotinib, upadacitinib was also a more potent inhibitor of JAK2 compared to filgotinib and of equal potency to baricitinib. Specifically, upadacitinib had a JAK2 IC50 of approximately 30nM compared to 65nM for baricitinib, but filgotinib had an IC50 >2000nM, or over 60-fold higher than upadacitinib (Exhibit 16). Given the once daily doses being studied by all these developers, the investigators estimated that at the highest dose JAK2 would be inhibited (above the IC50) by upadacitinib for 11 hours out of 24, or 45% of the day, whereas for baricitinib it would be inhibited 50% of the day, and for filgotinib for only 5% of the day.

Even more importantly, the relative JAK2/JAK1 (GM-CSF to pSTAT5 / IL-21 to pSTAT3) IC50 for upadacitinib is 0.5, whereas for baricitinib it is 0.6 and for filgotinib it is 2.2. This study was conducted and sponsored by Lilly, and therefore is subject to all the liabilities and biases of company-sponsored research. However, this study suggests that on this dimension at least, filgotinib might have the best profile, and baricitinib's is somewhere in between.

Exhibit 16: Relative JAK1 and JAK2 IC50 for JAK inhibitors by STAT phosphorylation



JAK1/3-dependent cytokine

	Bari 2-mg 4-mg	Upa 15-mg 30-mg	Filgo 100-mg 200-mg	Tofa 5-mg 10-mg
Time above IC_{50} (h)	0	7.7***†††	0	17.5***†††
	1.2	11.6***†††	2.9*	23.9***†††
STAT inhibition (%)	15	40***††	7*†††	61***†††
	27	52***†††	14††	76***†††

JAK2/2-dependent cytokine

	Bari 2-mg 4-mg	Upa 15-mg 30-mg	Filgo 100-mg 200-mg
Time above IC_{50} (h)	3.0	12.3**	0††
	8.5	17.9***††	1.1††
STAT inhibition (%)	28	51***	5***†††
	41	61***††	9**†††

Source: McInnes et al ACR 2017

Long Term Forecasts from Management Seem to be Best Case Scenarios

On the Q3 2017 earnings presentation AbbVie's management surprised us and investors by providing new long-term revenue guidance to 2025 for key products ($11.5bn in US alone), Venclexta ($6bn), upadacitinib (>$6.5bn), risankizumab (>$5bn) and Elagolix (>$2bn). They also highlighted the peak sales opportunities for all Rova-T indications and treatment lines, which exceed $5bn in total. Finally, they disclosed that non-Humira revenues would reach $35bn by 2025 on a risk-adjusted basis, and $47bn on a nominal basis. The market reacted positively to this release, and with the extra help of US tax reform the stock has performed +30% since that date. However, neither our estimates nor consensus reach management's forecasts. In fact, even when we remove our probability of success (PoS) adjustments from our forecasts, most of AbbVie's forecasts are considerably above our models (Exhibit 17). It is hard to reconcile these large differences in revenue outlook, particularly since 2025 is still 8 years away, and a multitude of new and/or transformative medicines and treatments could emerge in that time, and the industry pricing and payment environment could also be substantially altered by that date. We find it hard to understand the logic of offering such forecasts in the first place, given all the uncertainty in the industry and these categories, and we fundamentally find it very difficult to get to most of these individual product forecasts under any but the most optimistic scenario (i.e. all competitors fail in development or commercialization).