SUPPLEMENTAL MATERIAL

Contents

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Supplemental table S1. Definition of safety outcomes from ORAL Surveillance and tofacitinib RA, PsA, and UC development programs.

		ORAL Surveillance		Tofacitinib RA, PsA, and UC development programs	
Outcome	**Definition**	**Adjudication**	**Censoring time***	**Adjudication**	**Censoring time****
Malignancies (excl NMSC)	All malignant cancers excluding NMSC	Yes	Total time	Yes	28 days
MACE	3-point MACE, i.e., composite of CV death (i.e., death due to MI, stroke, sudden cardiac death, heart failure, CV procedures, CV haemorrhage and other CV causes, but not death due to PE), non-fatal MI and non-fatal stroke (including reversible focal neurological defects with imaging evidence of a new cerebral lesion consistent with ischaemia or haemorrhage)	Yes	60 days	Yes[†]	28 days
MI	Non-fatal or fatal MI	Yes	60 days	Yes[†]	28 days
VTE	DVT or PE (non-fatal or fatal)	Yes	28 days	No	28 days
All-cause death	Any fatal event regardless of cause	Yes	28 days	No	28 days

*In ORAL Surveillance, censoring times were applied as defined in the statistical analysis plan. If censoring time was total time, this was defined as time from first dose of trial drug until last contact date. Other censoring times were on-treatment times, which was defined as time from first dose of a trial drug until end of the censoring time/risk period (i.e., last contact date or last trial dose plus 28/60 days (depending on outcome), whichever was earliest). Last contact date was the latest of the following: start date of an adverse event, end date of an adverse event, date of last trial visit, withdrawal date, telephone contact date, or date of death. ** In the tofacitinib development programs other than ORAL Surveillance, the censoring time was the 28-day on-treatment time, defined as time from first dose of tofacitinib until last dose of tofacitinib + 28 days or date of death, whichever was earliest. [†]In the tofacitinib RA development program, MACE and MI data included patients from studies (NCT01262118, NCT01484561, NCT00147498, NCT00413660, NCT00550446, NCT00603512, NCT00687193, NCT01164579, NCT00976599, NCT01059864, NCT01359150, NCT02147587, NCT00960440, NCT00847613, NCT00814307, NCT00856544, NCT00853385, NCT01039688, NCT02281552, NCT02187055, NCT02831855, NCT00413699, NCT00661661) with the CV adjudication process applied after 25 February 2009.

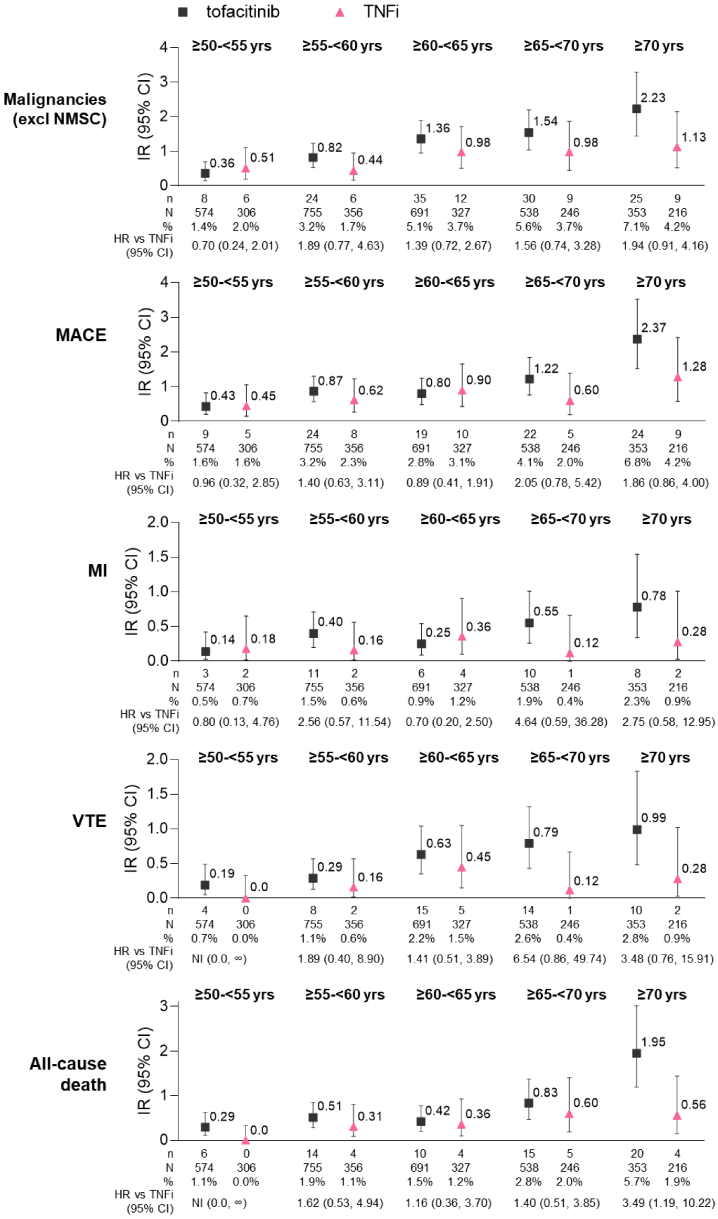
CV, cardiovascular; DVT, deep vein thrombosis; MACE, major adverse CV event; MI, myocardial infarction; NMSC, non-melanoma skin cancer; PE, pulmonary embolism; PsA, psoriatic arthritis; RA, rheumatoid arthritis; UC, ulcerative colitis; VTE, venous thromboembolism.

Supplemental table S2. Demographic and baseline disease characteristics in high-risk and low-risk patients in ORAL Surveillance and in the tofacitinib RA, PsA, and UC development programs.

	ORAL Surveillance		Tofacitinib Development program					
			RA*		PsA		UC	
	High-risk (N=2821)	Low-risk (N=1541)	High-risk (N=3577)	Low-risk (N=4198)	High-risk (N=341)	Low-risk (N=442)	High-risk (N=444)	Low-risk (N=713)
Female, % (n)	72.7% (2052)	88.1% (1358)	72.2% (2583)	89.9% (3773)	52.2% (178)	56.6% (250)	36.3% (161)	44.5% (317)
Duration (yrs) of disease, mean/median	10.2 / 7.4	10.9 / 8.9	8.5 / 5.7	7.7 / 5.3	8.3 / 6.0	7.2 / 6.0	9.3 / 7.5	7.5 / 5.8
Age, mean (SD)	63.3 (7.5)	57.2 (4.1)	57.9 (11.6)	48.4 (10.7)	51.8 (12.2)	46.3 (11.2)	48.4 (14.3)	36.9 (11.7)
≥65 years of age, % (n)	48.0% (1353)	0.0% (0)	35.5% (1270)	0.0% (0)	21.1% (72)	0.0% (0)	17.3% (77)	0.0% (0)
Smoking status**, % (n)								
Current	41.3% (1166)	0.0% (0)	38.2% (1366)	0.0% (0)	41.1% (140)	0.0% (0)	13.3% (59)	0.0% (0)
Past	33.2% (937)	0.0% (0)	38.8% (1388)	0.0% (0)	46.3% (158)	0.0% (0)	80.4% (357)	0.0% (0)
Never	25.5% (718)	100% (1541)	22.3% (798)	100% (4198)	12.6% (43)	100% (442)	6.1% (27)	100% (713)
History of other CV risk factors, % (n)								
Diabetes mellitus	17.1% (482)	18.0% (277)	10.3% (370)	6.4% (269)	14.7% (50)	12.9% (57)	7.0% (31) †	2.4% (17) †
Hyperlipidemia	38.3% (1080)	29.5% (454)	27.1% (968)	12.9% (541)	28.4% (97)	15.8% (70)	NA	NA
Hypertension	63.9% (1803)	69.8% (1075)	41.9% (1497)	28.1% (1179)	47.8% (163)	32.4% (143)	22.1% (98) †	8.8% (63) †
Coronary artery disease	14.5% (410)	5.6% (87)	3.2% (113)	0.3% (12)	7.0% (24)	4.5% (20)	3.6% (16)	0.3% (2)
ASCVD	18.5% (521)	7.7% (119)	6.2% (220)	1.2% (51)	8.5% (29)	5.0% (22)	7.2% (32)	1.8% (13)
Treatment history, % (n)								
Prior TNFi	8.1% (229)	6.6% (101)	18.7% (670)	12.7% (534)	51.0% (174)	45.9% (203)	59.8% (260)	51.1% (352)
Statin at baseline†	26.8% (756)	17.1% (264)	11.8% (421)	4.7% (199)	19.1% (65)	7.9% (35)	12.8% (57)#	2.4% (17)#
Aspirin at baseline†	18.0% (507)	10.4% (160)	11.1% (397)	3.6% (150)	9.7% (33)	3.8% (17)	NA	NA

High-risk patients were ≥65 years of age or ever smoker. Low-risk patients were <65 years of age and never smoker. *Excluding ORAL Surveillance. **In the tofacitinib RA development program, 2.7% (N=214) of patients had unknown smoking status. Patients <65 years old with unknown smoking status were not included in the low-risk group. 25 patients in the high-risk group had unknown smoking status. †Based on presence at day 1 of treatment. #Lipid-lowering agents.
ASCVD, atherosclerotic cardiovascular disease; CV, cardiovascular; n, number of patients with characteristic; NA, not available; PsA, psoriatic arthritis; SD, standard deviation; UC, ulcerative colitis.

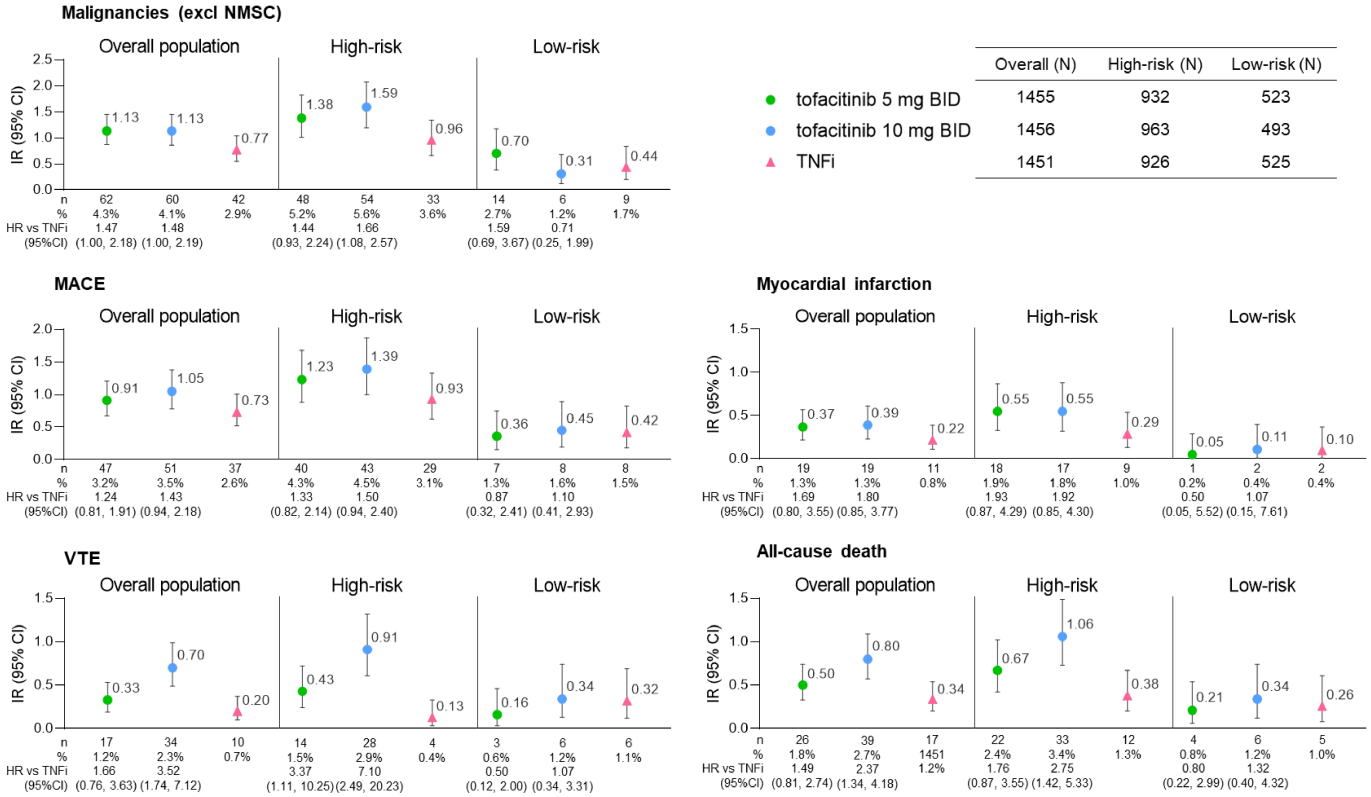
Supplemental figure S1. Risk of malignancies (excl NMSC), MACE, MI, VTE, and all-cause death with tofacitinib (combined doses) vs TNFi in ORAL Surveillance by age (5-year intervals).



IRs express the number of patients with first events per 100 PY. HRs (95% CIs) are based on a simple Cox proportional hazard model comparing tofacitinib (5 mg BID and 10 mg BID combined) vs TNFi.

CI, confidence interval; HR, hazard ratio; IR, incidence rate; MACE, major adverse cardiovascular events; MI, myocardial infarction; n, number of patients with events; N, number of evaluable patients; NI, non-informative; NMSC, non-melanoma skin cancer; PY, patient-years; TNFi, tumour necrosis factor inhibitor; VTE, venous thromboembolism.

Supplemental figure S2. Risk of malignancies (excl NMSC), MACE, MI, VTE, and all-cause death with tofacitinib 5 mg and 10 mg BID vs TNFi in ORAL Surveillance by subgroups of high-risk and low-risk patients.



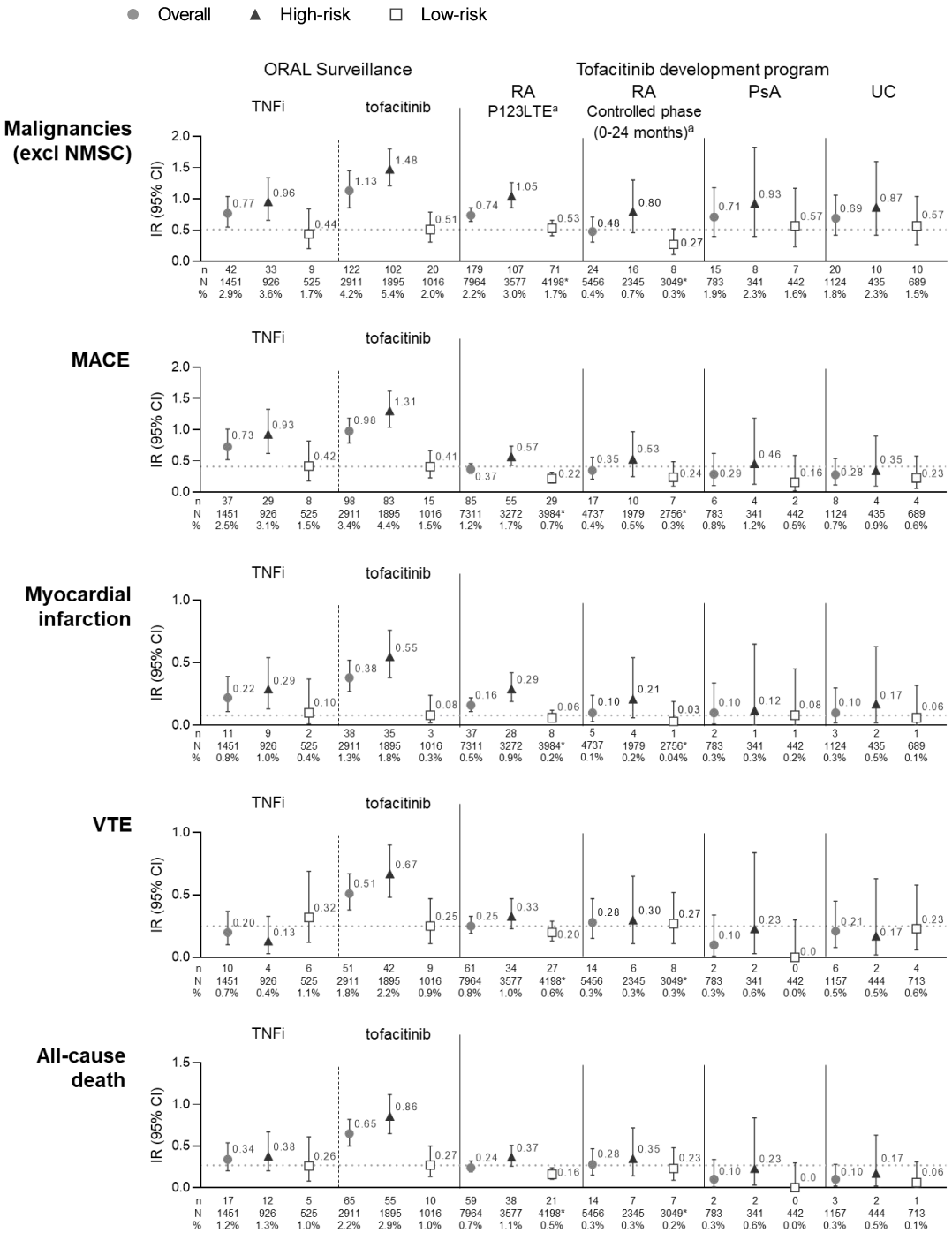
	Overall (N)	High-risk (N)	Low-risk (N)
tofacitinib 5 mg BID	1455	932	523
tofacitinib 10 mg BID	1456	963	493
TNFi	1451	926	525

High-risk patients were ≥65 years of age or ever smoker. Low-risk patients were <65 years of age and never smoker. IRs express the number of patients with first events per 100 PY. HRs (95% CIs) are based on simple Cox proportional hazard models comparing tofacitinib 5 mg BID and tofacitinib 10 mg BID vs TNFi.

Data from overall population have previously been published for and are included for reference; malignancies (excl NMSC) and MACE (Ytterberg et al. [1]), MI (Charles-Schoeman et al. [5])

CI, confidence interval; IR, incidence rate; MACE, major adverse cardiovascular events; MI, myocardial infarction; n, number of patients with events; N, number of evaluable patients; NMSC, non-melanoma skin cancer; PsA, psoriatic arthritis; PY, patient-years; RA, rheumatoid arthritis; TNFi, tumour necrosis factor inhibitor; UC, ulcerative colitis; VTE, venous thromboembolism.

Supplemental figure S3. Risk of malignancies (excl NMSC), MACE, MI, VTE, and all-cause death in ORAL Surveillance and tofacitinib RA, PsA and UC clinical development programs by subgroups of high-risk and low-risk patients.



High-risk patients were ≥65 years of age or ever smoker. Low-risk patients were <65 years of age and never smoker. Horizontal dotted line represents IR in low-risk patients treated with tofacitinib in ORAL Surveillance. IRs express the number of patients with first events per 100 PY. All data are for combined tofacitinib doses.

[a]Excluding ORAL Surveillance.

*In the tofacitinib RA development program, 2.7% (N=214) of patients had unknown smoking status. Patients <65 years old with unknown smoking status were not included in the low-risk group.

Data from ORAL Surveillance overall populations have previously been published and are included for reference; malignancies (excl NMSC) and MACE (Ytterberg et al. [1]), MI (Charles-Schoeman et al. [5]). Also previously published are data from the tofacitinib RA and PsA development programs on MACE (Burmester et al. [25]) and VTE (Mease et al. [26]).

CI, confidence interval; IR, incidence rate; MACE, major adverse cardiovascular events; MI, myocardial infarction; n, number of patients with events; N, number of evaluable patients; NMSC, non-melanoma skin cancer; PsA, psoriatic arthritis; PY, patient-years; RA, rheumatoid arthritis; TNFi, tumour necrosis factor inhibitor; UC, ulcerative colitis; VTE, venous thromboembolism.

Supplemental table S3. Summary of phase 1, phase 2, phase 3, phase 3/4b and LTE studies in the tofacitinib RA, PsA, and UC development programs

ClinicalTrials.gov identifier	Protocol number	Patients initially receiving tofacitinib, n	Patient population	Tofacitinib doses	Control arm	Study duration
RA clinical trials						
Phase 1						
NCT01262118[1]	A3921130	36 (RA), 33 (healthy volunteers)	Active RA and healthy volunteers	10 mg BID (background methotrexate permitted)	None	6 weeks
NCT01484561[2]	A3921152	97	Active RA with inadequate response to ≥1 DMARD	10 mg BID (background csDMARDs permitted)	Placebo BID	6 weeks (for tofacitinib treatment)
Phase 2						
NCT00147498[3]	A3921019	199	Active RA with inadequate response or unacceptable toxicity to methotrexate or to any of the following: etanercept, infliximab or adalimumab	5 mg BID, 15 mg BID, 30 mg BID monotherapy	Placebo BID	6 weeks
NCT00413660[4]	A3921025	438	Active RA with inadequate response to methotrexate	1, 3, 5, 10 or 15 mg BID or 20 mg QD with background methotrexate	Placebo	24 weeks
NCT00550446[5]	A3921035	272	Active RA with inadequate response or toxicity to ≥1 DMARD	1, 3, 5, 10 or 15 mg BID monotherapy	Adalimumab SC 40 mg Q2W; placebo	24 weeks

ClinicalTrials.gov identifier	Protocol number	Patients initially receiving tofacitinib, n	Patient population	Tofacitinib doses	Control arm	Study duration
NCT00603512[6]	A3921039	108	Active RA with inadequate response to methotrexate	1, 3, 5, 10 mg BID plus background methotrexate	Placebo	12 weeks
NCT00687193[7]	A3921040	265	Active RA with inadequate response to ≥1 DMARD	1, 3, 5, 10 or 15 mg BID monotherapy	Placebo	12 weeks
NCT01164579[8]	A3921068	72	Early active RA, methotrexate-naïve	10 mg BID plus methotrexate, 10 mg BID monotherapy	Methotrexate	12 months
NCT00976599[9]	A3921073	15	Active RA with inadequate response to methotrexate	10 mg BID plus background methotrexate	Placebo	4 weeks
NCT01059864[10]	A3291109	111	Active RA	10 mg BID, half of patients received concomitant atorvastatin 10 mg QD for Weeks 6-12	None	12 weeks
NCT01359150[11]	A3921129	102	Active RA	10 mg BID monotherapy (half of patients) or with background methotrexate	Placebo only (half of patients), or placebo plus methotrexate	9 weeks
NCT02147587[12]	A3921237	55	Moderate to severe RA inadequately controlled by methotrexate	5 mg BID plus background methotrexate; 2-3 weeks post-HZ vaccination	Placebo	14 weeks

ClinicalTrials.gov identifier	Protocol number	Patients initially receiving tofacitinib, n	Patient population	Tofacitinib doses	Control arm	Study duration
Phase 3						
NCT00960440[13]	ORAL Step, A3921032	267	Moderate to severe RA with inadequate response to TNFi	5 or 10 mg BID with background methotrexate	Placebo (advanced to tofacitinib at Month 3)	6 months
NCT00847613[14]	ORAL Scan, A3921044	637	Active RA with inadequate response to methotrexate	5 or 10 mg BID with background methotrexate	Placebo (advanced to tofacitinib at Month 3 (non-responders) or Month 6 (remaining patients))	24 months
NCT00814307[15]	ORAL Solo, A3921045	488	Active RA with inadequate response to ≥1 DMARD	5 or 10 mg BID monotherapy	Placebo (advanced to tofacitinib at Month 3)	6 months
NCT00856544[16]	ORAL Sync, A3921046	636	Active RA with inadequate response to ≥1 DMARD	5 or 10 mg BID with background csDMARD	Placebo (advanced to tofacitinib at Month 3 (non-responders) or Month 6 (remaining patients))	12 months
NCT00853385[17]	ORAL Standard, A3921064	405	Active RA with incomplete response to methotrexate	5 or 10 mg BID with background methotrexate	Adalimumab 40 mg SC Q2W; placebo (patients receiving placebo were advanced to tofacitinib at Month 3 (non-responders) or Month 6 (remaining patients))	12 months

ClinicalTrials.gov identifier	Protocol number	Patients initially receiving tofacitinib, n	Patient population	Tofacitinib doses	Control arm	Study duration
NCT01039688[18]	ORAL Start, A3921069	770	Active RA, methotrexate-naïve	5 or 10 mg BID monotherapy	Methotrexate	24 months
NCT02281552[19]	A3921215	209	Japanese patients with active RA with inadequate response to methotrexate	11 mg MR QD or 5 mg IR BID with background methotrexate	None	12 weeks
Phase 3b/4						
NCT02187055[20]	ORAL Strategy, A3921187	760	Active RA with inadequate response to methotrexate	5 mg BID monotherapy or with background methotrexate	Adalimumab 40 mg SC Q2W with background methotrexate	12 months
NCT02831855[21]	ORAL Shift, A3921192	623	Active RA with inadequate response to methotrexate	11 mg MR QD monotherapy or with background methotrexate	None	48 weeks
LTE						
NCT00413699[22]	ORAL Sequel, A3921024	4481 (final data cut March 2017)	Active RA who participated in the above studies	5 or 10 mg BID, concomitant DMARDs permitted	None	114 months

ClinicalTrials.gov identifier	Protocol number	Patients initially receiving tofacitinib, n	Patient population	Tofacitinib doses	Control arm	Study duration
NCT00661661[23]	A3921041	486	Japanese patients with active RA who participated in studies A3921039, A3921040 or A3921044	5 or 10 mg BID, concomitant DMARDs permitted after Week 12	None	72 months
Pooled LTE NCT00413699; NCT00661661[24, 25]	ORAL Sequel, A3921024; A3921041	4967 (ORAL Sequel final data cut March 2017)	Active RA who participated in the above studies	5 or 10 mg BID, concomitant DMARDs permitted	None	114 months; 72 months
PsA clinical trials						
Phase 3						
NCT01877668[26]	OPAL Broaden, A3921091	211	Active PsA, TNFi-naïve with an inadequate response to ≥1 csDMARD	5 or 10 mg BID with a stable dose of a single DMARD	Placebo, adalimumab 40 mg SC Q2W	12 months

ClinicalTrials.gov identifier	Protocol number	Patients initially receiving tofacitinib, n	Patient population	Tofacitinib doses	Control arm	Study duration
NCT01882439[27]	OPAL Beyond, A3921125	263	Active PsA with an inadequate response to ≥1 TNFi	5 or 10 mg BID with a stable dose of a single DMARD	Placebo	6 months
LTE						
NCT01976364[28]	OPAL Balance, A3921092	686	Patients from OPAL Broaden, A3921091 and OPAL Beyond A3921125	5 mg BID or 10 mg BID, concomitant DMARDs permitted	None	36 months
UC clinical trials						
Phase 2						
NCT00787202[29]	A3921063	146	Moderate to severe UC	0.5, 3, 10 or 15 mg BID	Placebo	8 weeks
Phase 3						
NCT01465763[30]	OCTAVE Induction 1, A3921094	492	Moderate to severe UC with prior failure/intolerance to corticosteroids, immunomodulators and/or TNFi	10 or 15 mg BID	Placebo	8 weeks

ClinicalTrials.gov identifier	Protocol number	Patients initially receiving tofacitinib, n	Patient population	Tofacitinib doses	Control arm	Study duration
NCT01458951[30]	OCTAVE Induction 2, A3921095	435	Moderate to severe UC with prior failure/intolerance to corticosteroids, immunomodulators and/or TNFi	10 or 15 mg BID	Placebo	8 weeks
NCT01458574[30]	OCTAVE Sustain, A3921096	395	Moderate to severe UC, completing OCTAVE Induction 1 or 2 with clinical response	5 or 10 mg BID	Placebo	52 weeks
LTE						
NCT01470612[31]	OCTAVE Open, A3921139	944	Patients from OCTAVE Induction 1, A3921094; OCTAVE Induction 2, A3921095; and OCTAVE Sustain, A3921096	5 or 10 mg BID	None	≥12 months

BID, twice daily; csDMARD, conventional synthetic disease-modifying antirheumatic drug; DMARD, disease-modifying antirheumatic drug; HZ, herpes zoster; IR, immediate release; LTE, long-term extension; MR, modified release; n, number of patients; QD, once daily; Q2W, every 2 weeks; PsA, psoriatic arthritis; RA, rheumatoid arthritis; SC, subcutaneous; TNFi, tumour necrosis factor inhibitor; UC, ulcerative colitis.

References – Tofacitinib development programs

1 Charles-Schoeman C, Fleischmann R, Davignon J, et al. Potential mechanisms leading to the abnormal lipid profile in patients with rheumatoid arthritis versus healthy volunteers and reversal by tofacitinib. *Arthritis Rheumatol* 2015;67:616–25.

2 Kremer JM, Kivitz AJ, Simon-Campos JA, et al. Evaluation of the effect of tofacitinib on measured glomerular filtration rate in patients with active rheumatoid arthritis: results from a randomised controlled trial. *Arthritis Res Ther* 2015;17:95.

3 Kremer JM, Bloom BJ, Breedveld FC, et al. The safety and efficacy of a JAK inhibitor in patients with active rheumatoid arthritis: results of a double-blind, placebo-controlled phase IIa trial of three dosage levels of CP-690,550 versus placebo. *Arthritis Rheum* 2009;60:1895–905.

4 Kremer JM, Cohen S, Wilkinson BE, et al. A phase IIb dose-ranging study of the oral JAK inhibitor tofacitinib (CP-690,550) versus placebo in combination with background methotrexate in patients with active rheumatoid arthritis and an inadequate response to methotrexate alone. *Arthritis Rheum* 2012;64:970–81.

5 Fleischmann R, Cutolo M, Genovese MC, et al. Phase IIb dose-ranging study of the oral JAK inhibitor tofacitinib (CP-690,550) or adalimumab monotherapy versus placebo in patients with active rheumatoid arthritis with an inadequate response to disease-modifying antirheumatic drugs. *Arthritis Rheum* 2012;64:617–29.

6 Tanaka Y, Suzuki M, Nakamura H, et al. Phase II study of tofacitinib (CP-690,550) combined with methotrexate in patients with rheumatoid arthritis and an inadequate response to methotrexate. *Arthritis Care Res (Hoboken)* 2011;63:1150–8.

7 Tanaka Y, Takeuchi T, Yamanaka H, et al. Efficacy and safety of tofacitinib as monotherapy in Japanese patients with active rheumatoid arthritis: a 12-week, randomized, phase 2 study. *Mod Rheumatol* 2015;25:514–21.

8 Conaghan PG, Østergaard M, Bowes MA, et al. Comparing the effects of tofacitinib, methotrexate and the combination, on bone marrow oedema, synovitis and bone erosion in methotrexate-naive, early active rheumatoid arthritis: results of an exploratory randomised MRI study incorporating semiquantitative and quantitative techniques. *Ann Rheum Dis* 2016;75:1024–33.

9 Boyle DL, Soma K, Hodge J, et al. The JAK inhibitor tofacitinib suppresses synovial JAK1-STAT signalling in rheumatoid arthritis. *Ann Rheum Dis* 2015;74:1311–6.

10 McInnes IB, Kim HY, Lee SH, et al. Open-label tofacitinib and double-blind atorvastatin in rheumatoid arthritis patients: a randomised study. *Ann Rheum Dis* 2014;73:124–31.

11 Winthrop KL, Silverfield J, Racewicz A, et al. The effect of tofacitinib on pneumococcal and influenza vaccine responses in rheumatoid arthritis. *Ann Rheum Dis* 2016;75:687–95.

12 Winthrop KL, Wouters AG, Choy EH, et al. The safety and immunogenicity of live zoster vaccination in patients with rheumatoid arthritis before starting tofacitinib: a randomized phase II trial. *Arthritis Rheumatol* 2017;69:1969–77.

13 Burmester GR, Blanco R, Charles-Schoeman C, et al. Tofacitinib (CP-690,550) in combination with methotrexate in patients with active rheumatoid arthritis with an inadequate response to tumour necrosis factor inhibitors: a randomised phase 3 trial. *Lancet* 2013;381:451–60.

14 van der Heijde D, Tanaka Y, Fleischmann R, et al. Tofacitinib (CP-690,550) in patients with rheumatoid arthritis receiving methotrexate: twelve-month data from a twenty-four-month phase III randomized radiographic study. *Arthritis Rheum* 2013;65:559–70.

15 Fleischmann R, Kremer J, Cush J, et al. Placebo-controlled trial of tofacitinib monotherapy in rheumatoid arthritis. *N Engl J Med* 2012;367:495–507.

16 Kremer J, Li Z-G, Hall S, et al. Tofacitinib in combination with nonbiologic disease-modifying antirheumatic drugs in patients with active rheumatoid arthritis: a randomized trial. *Ann Intern Med* 2013;159:253–61.

17 van Vollenhoven RF, Fleischmann R, Cohen S, et al. Tofacitinib or adalimumab versus placebo in rheumatoid arthritis. *N Engl J Med* 2012;367:508–19.

18 Lee EB, Fleischmann R, Hall S, et al. Tofacitinib versus methotrexate in rheumatoid arthritis. *N Engl J Med* 2014;370:2377–86.

19 Tanaka Y, Sugiyama N, Toyoizumi S, et al. Modified- versus immediate-release tofacitinib in Japanese rheumatoid arthritis patients: a randomized, phase III, non-inferiority study. *Rheumatology (Oxford)* 2019;58:70–9.

20 Fleischmann R, Mysler E, Hall S, et al. Efficacy and safety of tofacitinib monotherapy, tofacitinib with methotrexate, and adalimumab with methotrexate in patients with rheumatoid arthritis (ORAL Strategy): a phase 3b/4, double-blind, head-to-head, randomised controlled trial. *Lancet* 2017;390:457–68.

21 Cohen SB, Pope J, Haraoui B, et al. Methotrexate withdrawal in patients with rheumatoid arthritis who achieve low disease activity with tofacitinib modified-release 11 mg once daily plus methotrexate (ORAL Shift): a randomised, phase 3b/4, non-inferiority trial. *Lancet Rheumatol* 2019;1:E23–E34.

22 Wollenhaupt J, Lee EB, Curtis JR, et al. Safety and efficacy of tofacitinib for up to 9.5 years in the treatment of rheumatoid arthritis: final results of a global, open-label, long-term extension study. *Arthritis Res Ther* 2019;21:89.
23 Yamanaka H, Tanaka Y, Takeuchi T, et al. Tofacitinib, an oral Janus kinase inhibitor, as monotherapy or with background methotrexate, in Japanese patients with rheumatoid arthritis: an open-label, long-term extension study. *Arthritis Res Ther* 2016;18:34.
24 Wollenhaupt J, Silverfield J, Lee EB, et al. Tofacitinib, an oral Janus kinase inhibitor, in the treatment of rheumatoid arthritis: safety and efficacy in open-label, long-term extension studies over 8 years [abstract]. *Ann Rheum Dis* 2017;76(Suppl 2):THU0197.
25 Wollenhaupt J, Silverfield J, Lee EB, et al. Safety and efficacy of tofacitinib, an oral Janus kinase inhibitor, for the treatment of rheumatoid arthritis in open-label, longterm extension studies. *J Rheumatol* 2014;41:837–52.
26 Mease P, Hall S, FitzGerald O, et al. Tofacitinib or adalimumab versus placebo for psoriatic arthritis. *N Engl J Med* 2017;377:1537–50.
27 Gladman D, Rigby W, Azevedo VF, et al. Tofacitinib for psoriatic arthritis in patients with an inadequate response to TNF inhibitors. *N Engl J Med* 2017;377:1525–36.
28 Nash P, Coates LC, Kivitz AJ, et al. Safety and efficacy of tofacitinib in patients with active psoriatic arthritis: interim analysis of OPAL Balance, an open-label, long-term extension study. *Rheumatol Ther* 2020;7:553–80.
29 Sandborn WJ, Ghosh S, Panés J, et al. Tofacitinib, an oral Janus kinase inhibitor, in active ulcerative colitis. *N Engl J Med* 2012;367:616–24.
30 Sandborn WJ, Su C, Sands BE, et al. Tofacitinib as induction and maintenance therapy for ulcerative colitis. *N Engl J Med* 2017;376:1723–36.
31 Lichtenstein GR, Loftus Jr EV, Wei SC, et al. DOP61 tofacitinib, an oral, small-molecule Janus kinase inhibitor, in the treatment of ulcerative colitis: analysis of an open-label, long-term extension study with up to 5.9 years of treatment [abstract]. *J Crohns Colitis* 2020;14(Suppl 1):DOP61.