Table 2. Treatment-emergent AEs through week 52

Event, n (%)	FIL 200 (n = 475)	FIL 100 mg (n = 480)	ADA (n = 325)
All AEs	352 (74.1)	350 (72.9)	239 (73.5)
Serious AEs	35 (7.4)	40 (8.3)	22 (6.8)
Infection	206 (43.4)	194 (40.4)	129 (39.7)
Serious infection	13 (2.7)	13 (2.7)	10 (3.1)
Herpes zoster	6 (1.3)	4 (0.8)	2 (0.6)
VTE	1 (0.2)	0	1 (0.3)
MACE (adjudicated)	0	2 (0.4)	1 (0.3)
Malignancy (excluding NMSC)	2 (0.4)	2 (0.4)	2 (0.6)
NMSC	1 (0.2)	1 (0.2)	0
Death	3 (0.6)	1 (0.2)	1 (0.3)

Data omitted for patients rerandomised from placebo to FIL.

ADA, adalimumab; AE, adverse event; FIL, filgotinib; MACE, major adverse cardiovascular event; NMSC, nonmelanoma skin cancer; VTE, venous thromboembolism.

Figure 1. Proportion of patients with DAS28(CRP) <2.6 through week 52 with nonresponder imputation

Conclusion: Through W52, both FIL 200 and 100 mg showed sustained efficacy based on clinical and pt-reported outcomes and radiographic progression and were well tolerated in MTX-IR pts with RA, with faster onset and numerically greater efficacy for FIL 200 vs 100 mg.

Figure 2. Patients in remission at weeks 12, 24, and 52

References:

[1] Combe et al., *Ann Rheum Dis*. 2019; 78 (Suppl 2):77–8.

Disclosure of Interests: Bernard Combe Grant/research support from: Novartis, Pfizer, Roche-Chugai, Consultant of: AbbVie; Gilead Sciences, Inc.; Janssen; Eli Lilly and Company; Pfizer; Roche-Chugai; Sanofi, Speakers bureau: Bristol-Myers Squibb; Gilead Sciences, Inc.; Eli Lilly and Company; Merck Sharp & Dohme; Pfizer; Roche-Chugai; UCB, Alan Kivitz Shareholder of: AbbVie, Amgen, Gilead, GSK, Pfizer Inc, Sanofi, Consultant of: AbbVie,

Boehringer Ingelheim, Flexion, Genzyme, Gilead, Janssen, Novartis, Pfizer Inc, Regeneron, Sanofi, SUN Pharma Advanced Research, UCB, Paid instructor for: Celgene, Genzyme, Horizon, Merck, Novartis, Pfizer, Regeneron, Sanofi, Speakers bureau: AbbVie, Celgene, Flexion, Genzyme, Horizon, Merck, Novartis, Pfizer Inc, Regeneron, Sanofi, Yoshiya Tanaka Grant/research support from: Asahi-kasei, Astellas, Mitsubishi-Tanabe, Chugai, Takeda, Sanofi, Bristol-Myers, UCB, Daiichi-Sankyo, Eisai, Pfizer, and Ono, Consultant of: AbbVie, Astellas, Bristol-Myers Squibb, Eli Lilly, Pfizer, Speakers bureau: Daiichi-Sankyo, Astellas, Chugai, Eli Lilly, Pfizer, AbbVie, YL Biologics, Bristol-Myers, Takeda, Mitsubishi-Tanabe, Novartis, Eisai, Janssen, Sanofi, UCB, and Teijin, Désirée van der Heijde Consultant of: AbbVie, Amgen, Astellas, AstraZeneca, BMS, Boehringer Ingelheim, Celgene, Cytex, Daiichi, Eisai, Eli-Lilly, Galapagos, Gilead Sciences, Inc., Glaxo-Smith-Kline, Janssen, Merck, Novartis, Pfizer, Regeneron, Roche, Sanofi, Takeda, UCB Pharma; Director of Imaging Rheumatology BV, J-Abraham Simon-Campos: None declared, Herbert S.B. Baraf Grant/research support from: Horizon; Gilead Sciences, Inc.; Pfizer; Janssen; AbbVie, Consultant of: Horizon; Gilead Sciences, Inc.; Merck; AbbVie, Speakers bureau: Horizon, Uma Kumar: None declared, Franziska Matzkies Shareholder of: Gilead Sciences, Inc., Employee of: Gilead Sciences, Inc., Beatrix Bartok Shareholder of: Gilead Sciences Inc., Employee of: Gilead Sciences Inc., Lei Ye Shareholder of: Gilead Sciences Inc., Employee of: Gilead Sciences Inc., Ying Guo Shareholder of: Gilead Sciences, Inc., Employee of: Gilead Sciences, Inc., Chantal Tasset Shareholder of: Galapagos (share/warrant holder), Employee of: Galapagos, John Sundy Shareholder of: Gilead Sciences, Inc., Employee of: Gilead Sciences, Inc., Angelika Jahreis Shareholder of: Gilead Sciences, Inc., Employee of: Gilead Sciences, Inc., Neelufar Mozaffarian Shareholder of: Gilead, Employee of: Gilead, Robert B.M. Landewé Consultant of: AbbVie; AstraZeneca; Bristol-Myers Squibb; Eli Lilly & Co.; Galapagos NV; Novartis; Pfizer; UCB Pharma, Sang-Cheol Bae: None declared, Edward Keystone Grant/research support from: AbbVie; Amgen; Gilead Sciences, Inc.; Lilly Pharmaceuticals; Merck; Pfizer Pharmaceuticals; PuraPharm; Sanofi, Consultant of: AbbVie; Amgen; AstraZeneca Pharma; Bristol-Myers Squibb Company; Celltrion; F. Hoffman-La Roche Ltd.; Genentech, Inc; Gilead Sciences, Inc.; Janssen, Inc; Lilly Pharmaceuticals; Merck; Myriad Autoimmune; Pfizer Pharmaceuticals, Sandoz, Sanofi-Genzyme, Samsung Bioepis., Speakers bureau: AbbVie; Amgen; Bristol-Myers Squibb; Celltrion; F. Hoffman-La Roche Ltd, Janssen, Inc; Merck; Pfizer Pharmaceuticals; Sanofi-Genzyme; UCB, Peter Nash Grant/research support from: AbbVie, Bristol-Myers Squibb, Celgene, Eli Lilly and Company, Gilead, Janssen, MSD, Novartis, Pfizer Inc, Roche, Sanofi, UCB, Consultant of: AbbVie, Bristol-Myers Squibb, Celgene, Eli Lilly, Gilead, Janssen, MSD, Novartis, Pfizer Inc, Roche, Sanofi, UCB, Speakers bureau: AbbVie, Bristol-Myers Squibb, Celgene, Eli Lilly, Gilead, Janssen, MSD, Novartis, Pfizer Inc, Roche, Sanofi, UCB

DOI: 10.1136/annrheumdis-2020-eular.276

THU0199

ABX464, A NOVEL DRUG IN THE FIELD OF INFLAMMATION, INCREASES MIR-124 AND MODULATES MACROPHAGES AND T-CELL FUNCTIONS.

C. Begon-Pescia¹, J. Mielles¹, N. Campose², K. Chebli¹, L. Manchon¹, J. Santo², C. Apollit², K. Martin², L. Lapasset², A. Vautrin², D. Scherrer², A. Garcel², J. Tazi², C. Daïen^{1,3}. ¹IGMM, UMR5535, Cooperative Lab, Montpellier, France; ²Abivax Therapeutics, Cooperative Lab, Montpellier, France; ³Hospital and University of Montpellier, Montpellier, France

Background: ABX464 is a small oral molecule with a novel mode of action. It binds the Cap Binding Complex, involved in the biogenesis of RNAs and predominantly upregulates the expression of a microRNA miR-124 in PBMCs and T cells (1). miR-124 has been widely described for its anti-inflammatory properties, with many confirmed targets i.e. monocyte chemoattractant protein 1 (MCP-1, CXCL-1, SERPIN-E1, STAT-3, IL-6 receptor. It post-transcriptionally regulates the expression of MCP-1 in rheumatoid arthritis (RA) synovocytes and decreases their proliferation (2). While miR-124 is decreased in synovocytes of RA patients, its injection in joint improved arthritis in rats (3). miR-124 expression in macrophages leads to the induction and maintenance of anti-inflammatory M2 phenotype (4). Its effect in T cells remains controversial.

Objectives: (i) To assess the effect of ABX464 on miR-124 expression *in vitro* in macrophages and *in vivo* in patients; (ii) to assess the effect of ABX464