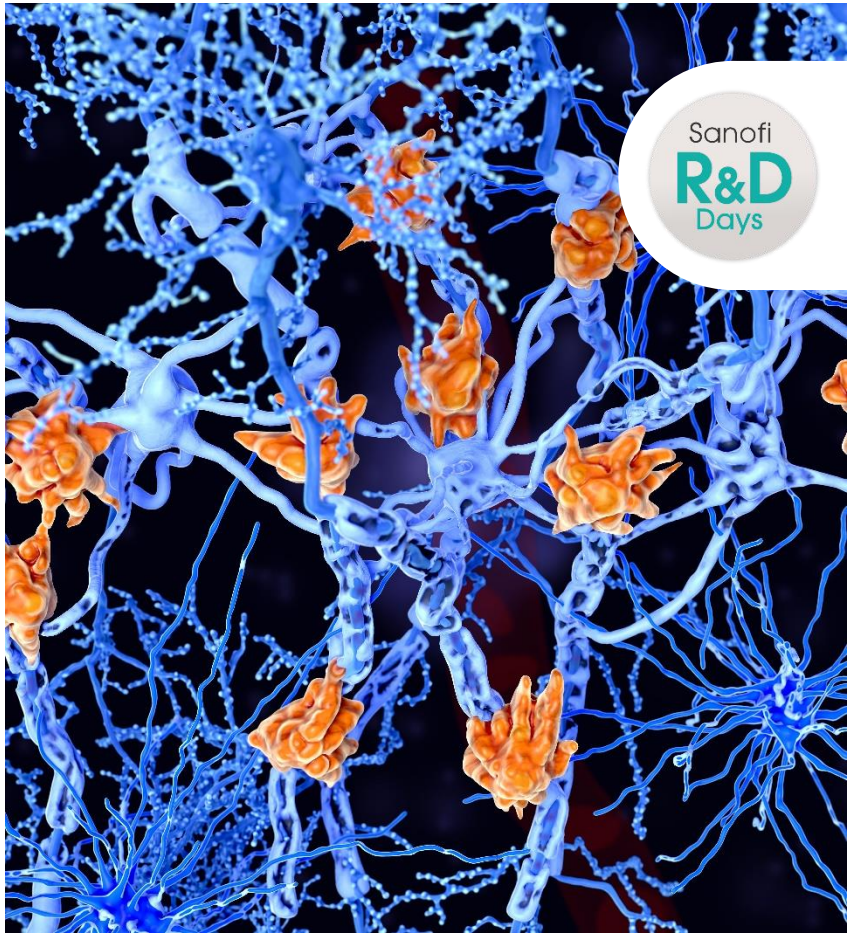

Phase 2b BTKi ('168) Trial Results

Dose-finding Study for SAR442168 in Relapsing Multiple Sclerosis

April 23, 2020

SANOFI

Forward looking statements

This presentation contains forward-looking statements as defined in the Private Securities Litigation Reform Act of 1995, as amended. Forward-looking statements are statements that are not historical facts. These statements include projections and estimates and their underlying assumptions, statements regarding plans, objectives, intentions and expectations with respect to future financial results, events, operations, services, product development and potential, and statements regarding future performance. Forward-looking statements are generally identified by the words “expects”, “anticipates”, “believes”, “intends”, “estimates”, “plans” and similar expressions. Although Sanofi’s management believes that the expectations reflected in such forward-looking statements are reasonable, investors are cautioned that forward-looking information and statements are subject to various risks and uncertainties, many of which are difficult to predict and generally beyond the control of Sanofi, that could cause actual results and developments to differ materially from those expressed in, or implied or projected by, the forward-looking information and statements. These risks and uncertainties include among other things, the uncertainties inherent in research and development, future clinical data and analysis, including post marketing, decisions by regulatory authorities, such as the FDA or the EMA, regarding whether and when to approve any drug, device or biological application that may be filed for any such product candidates as well as their decisions regarding labelling and other matters that could affect the availability or commercial potential of such product candidates, the fact that product candidates if approved may not be commercially successful, the future approval and commercial success of therapeutic alternatives, Sanofi’s ability to benefit from external growth opportunities, to complete related transactions and/or obtain regulatory clearances, risks associated with intellectual property and any related pending or future litigation and the ultimate outcome of such litigation, trends in exchange rates and prevailing interest rates, volatile economic and market conditions, cost containment initiatives and subsequent changes thereto, and the impact that COVID-19 will have on us, our customers, suppliers, vendors, and other business partners, and the financial condition of any one of them, as well as on our employees and on the global economy as a whole. Any material effect of COVID-19 on any of the foregoing could also adversely impact us. This situation is changing rapidly and additional impacts may arise of which we are not currently aware and may exacerbate other previously identified risks. The risks and uncertainties also include the uncertainties discussed or identified in the public filings with the SEC and the AMF made by Sanofi, including those listed under “Risk Factors” and “Cautionary Statement Regarding Forward-Looking Statements” in Sanofi’s annual report on Form 20-F for the year ended December 31, 2019. Other than as required by applicable law, Sanofi does not undertake any obligation to update or revise any forward-looking information or statements.

Agenda

Introduction	**Bill Sibold**	EVP, Specialty Care	
Phase 2 results	**Erik Wallstroem**	Therapeutic Area Head, Neurology Development	
Progression in MS and underlying mechanisms	**Rita Balice-Gordon**	Therapeutic Area Head, Neurologic and Rare Diseases Research	
Phase 3 plan & conclusion	**John Reed**	EVP, Global Head of R&D	
Q&A session *(also joining)*	**Tom Snow**	Global Franchise Head, Neurology and Immunology	

Introduction

Bill Sibold

EVP, Specialty Care

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Sanofi's long-term commitment to Multiple Sclerosis

Once-daily AUBAGIO® (teriflunomide) 14mg tablets

- Only oral DMT proven to reduce the risk of confirmed disability worsening in 2 phase 3 trials[1,2]
- Long-term safety profile confirmed in >15 years of clinical trial and real world experience[3]
- No confirmed cases of PML to date[4]
- High treatment satisfaction reported by both new patients and switchers[5]

LEMTRADA® alemtuzumab 12mg IV

- Long-term disease control in the absence of continuous dosing
- >17,000 patients currently controlled[6]
- Confirmed disability improvement in 9-year follow-up of phase 3's[7]
- Safety profile – infusion reactions and secondary autoimmunity – supported by >10 year data

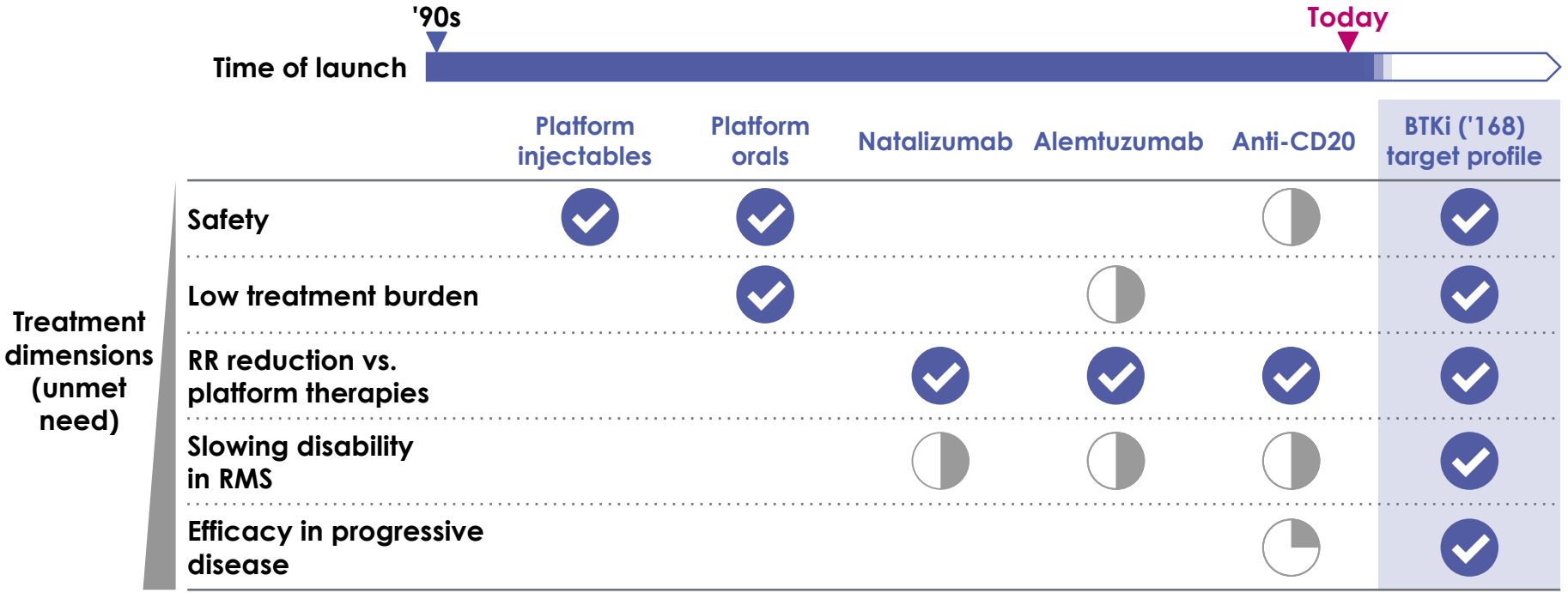
BTKi ('168) target profile

- Aiming at best-in-class across all treatment dimensions
  - Safety
  - Low treatment burden
  - RR reduction
  - Slowing disability in RMS
  - Efficacy in progressive disease
- Supported by dual mechanism
  - Potent B-cell modulation in the periphery
  - Re-establishing microglia homeostasis in CNS

Sanofi is #2 in MS global patient share

CNS: Central Nervous System; DMT: Disease Modifying Therapy; PML: Progressive multifocal leukoencephalopathy; RR: Relapse Rate; RMS: Relapsing Multiple Sclerosis
(1) TEMSO: O'Connor PW et al. N Engl J Med. 2011;365:1293−303 (2) TOWER: Confavreux C et al. Lancet Neurol. 2014;13(3):247−56 (3) Miller AE et al. Mult Scler Relat Disord. 2019;33:131-138 (4) Data on file (5) Teri-PRO: Coyle PK et al. Mult Scler Relat Disord. 2018;26:211-218 (6) Patients previously dosed with Lemtrada and did not require additional Lemtrada doses in 2020 - potential discontinuations not included – Source: Sanofi analysis (7) ECTRIMS Online Library. Comi G. 09/11/19; 279005; P645
BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators

No approved therapy addresses all treatment dimensions despite ongoing improvement in SoC



BTKi ('168) aims to address all 5 treatment dimensions with dual mechanism of action

RR: Relapse Rate; RMS: Relapsing Multiple Sclerosis; SoC: standard of care
No head to head studies comparing the treatments referenced against the investigational treatment BTKi ('168) have been conducted. The BTKi ('168) target profile is aspirational and comparisons with other therapies cannot be made at this time.
BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators.

BTKi ('168) aims at superior target profile through dual MoA

Anti-CD20 MoA & clinical outcomes

Periphery

Acquired immunity

B-cell depletion
(>6m until recovery)

BTKi ('168) MoA & target profile

Periphery

CNS
Exposure confirmed in phase 1 (BBB crossing)

Acquired immunity

Innate immunity
Thought to play a key role in MS progression(3)

B-cell modulation
(5-7d until recovery)

Clinical outcomes:

- Safety concerns due to long-term B-cell depletion
- Infusion burden
- Significant relapse rate reduction(1)
- Impact on disability superior to interferon beta(1)
- Modest effect in primary progressive MS(2)

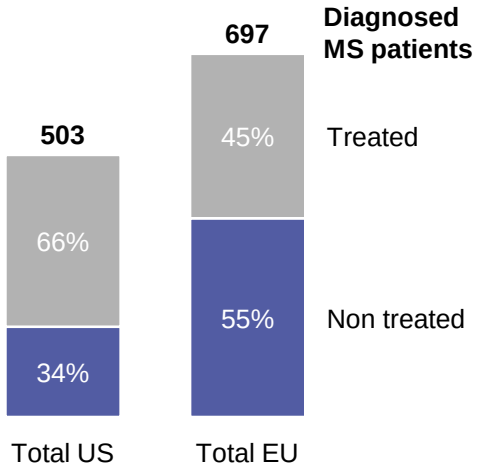
BTKi ('168) target profile:

- Best-in-class safety, with fast-reversible immune-suppression and no off-target toxicity
- Oral once daily
- Best relapse rate reduction among orals
- Significant impact on disability in RMS
- Significant impact on progressive disease

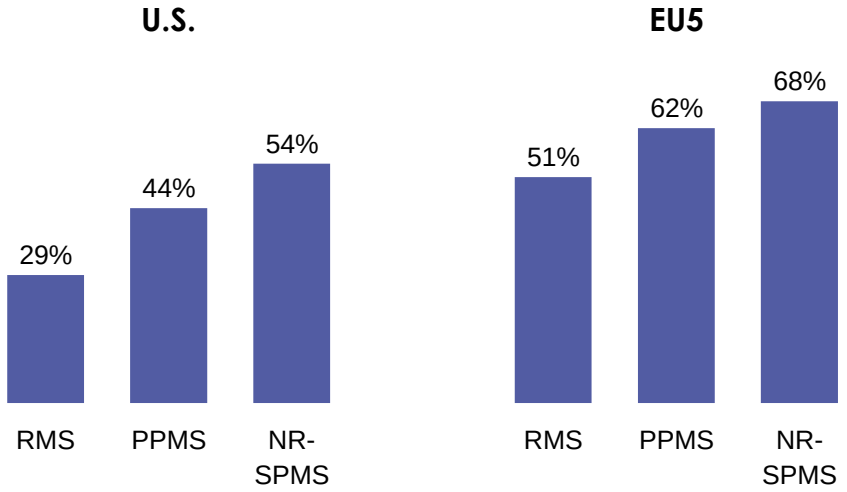
(1) Hauser S et al, N Engl J Med 2017; 376:221-234 (2) Ocrelizumab: 24% relative reduction of 12-week confirmed disability progression; Montalban X et al, N Engl J Med 2017 Jan 19;376(3):209-220 (3) Li R et al. Nat Immunol 2018;19:696-707; Chitnis T, Weiner HL. J Clin Invest 2017;127:3577-87; Hendriks RW. Nat Chem Biol 2011;7:4-5
BTK: bruton tyrosine kinase; CNS: central nervous system; BBB: blood-brain barrier; MoA: mechanism of action
BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators

Large number of diagnosed MS patients receive no DMT

More than 1/3 of U.S. patients and 1/2 of EU5 patients untreated



Higher proportion of untreated patients as treatment options decrease

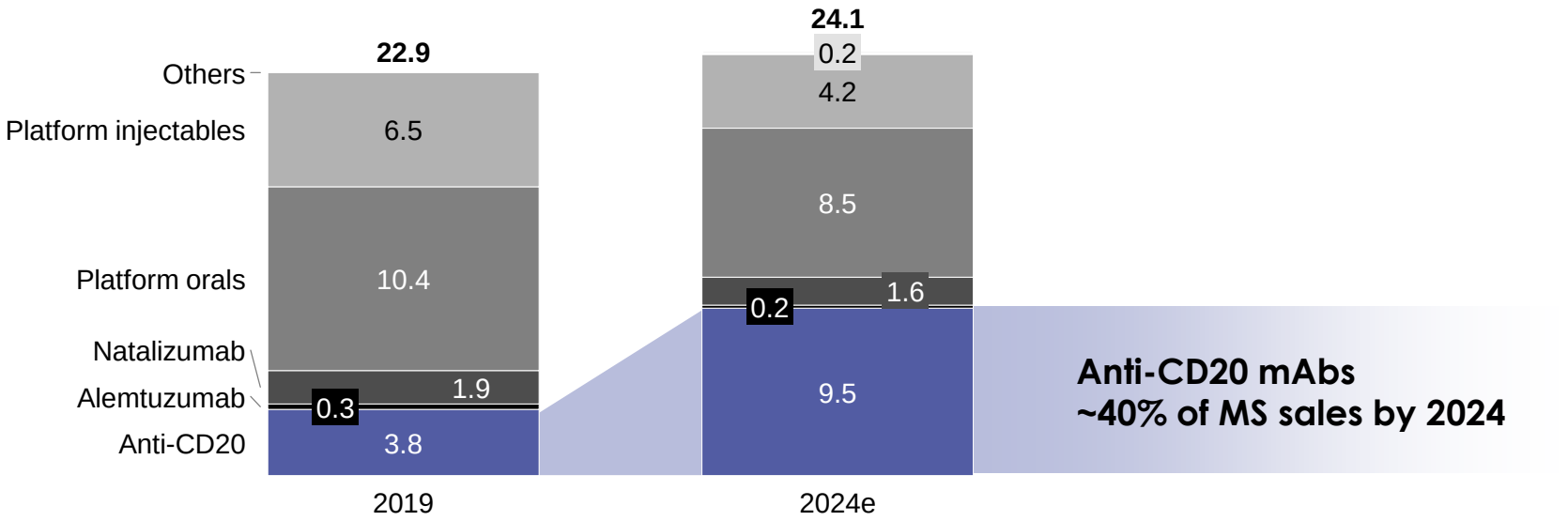


New treatment needed to address disability & disease progression, the key unmet needs in MS

DMT: Disease Modifying Therapy; RMS: Relapsing-remitting Multiple Sclerosis; PPMS: Primary Progressive Multiple Sclerosis; NR-SPMS: Non Relapsing Secondary Progressive Multiple Sclerosis; EU5: Germany, France, Italy, Spain & United Kingdom
Source: Sanofi analysis

Projected strong growth for B-cell targeting therapies

WW MS Sales (Market estimates, US$ bn)



Delivering BTKi ('168) target product profile expected to result in leading market position

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Anti-CD20 mAbs: Ocrevus, ofatumumab, ublituximab; other classes include ABCRs, S1Px, DMFs, Aubagio, Lemtrada, Tysabri. 2024E reflects generics for Aubagio®, Gilenya®, Tecfidera®
Source: Evaluate Pharma (as of April 6, 2020)
BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators.

Phase 2 results

Erik Wallstroem

Therapeutic Area Head,
Neurology Development

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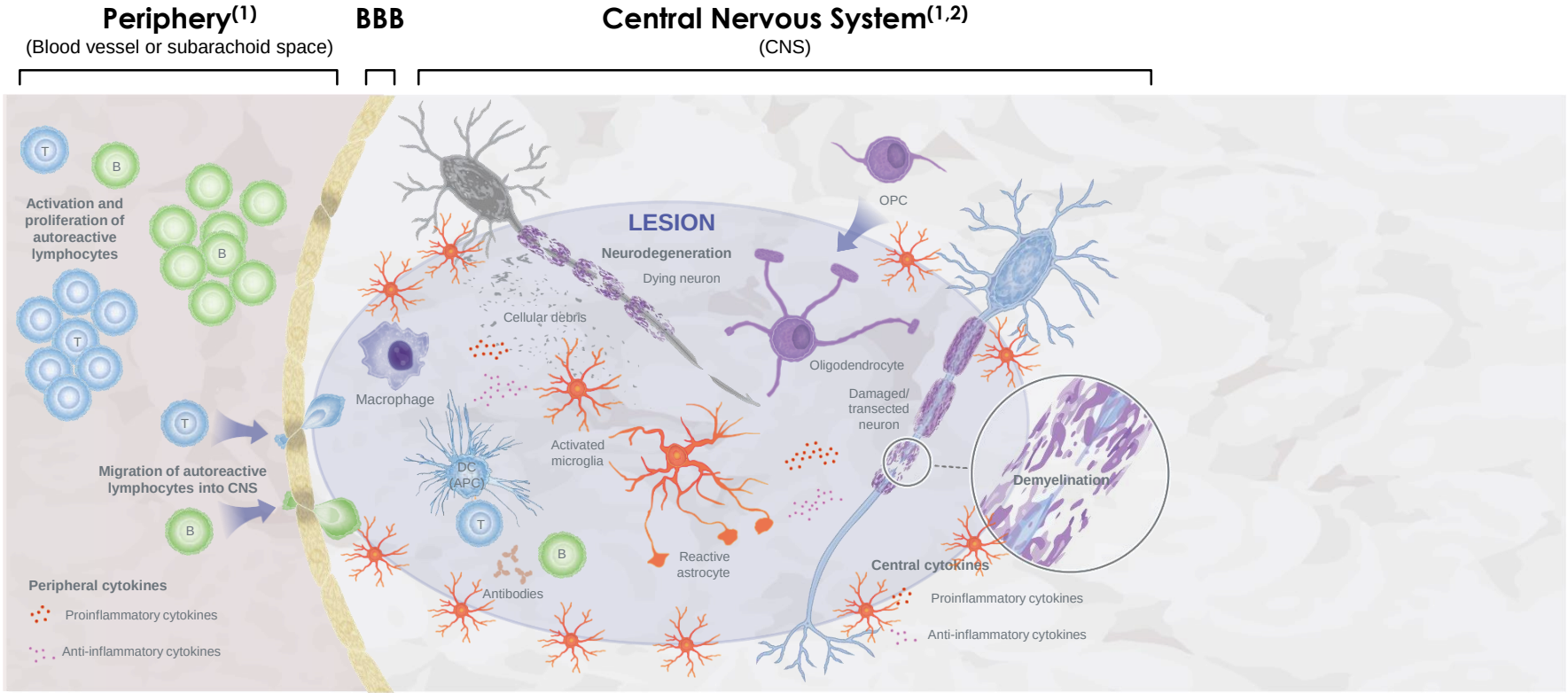
Dual MoA: targeting peripheral immune cell function and CNS-intrinsic inflammation

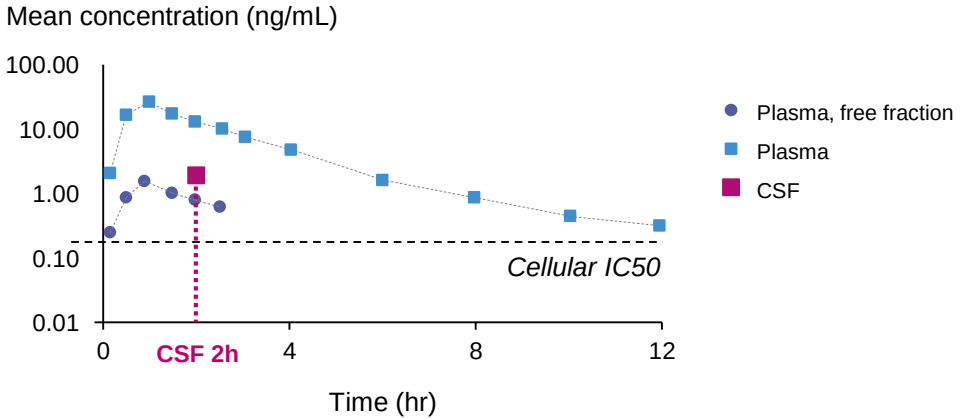


APC: antigen-presenting cell; BBB: blood-brain barrier; DC: dendritic cell; OPC: oligodendrocyte progenitor cell.
(1) Reich DS et al. N Engl J Med 2018;378:169-80. (2) Gandhi R et al. J Neuroimmunol 2010;221:7-14.
BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators.

BTKi ('168) is the only molecule of its class to demonstrate pharmacologically relevant exposure in CNS

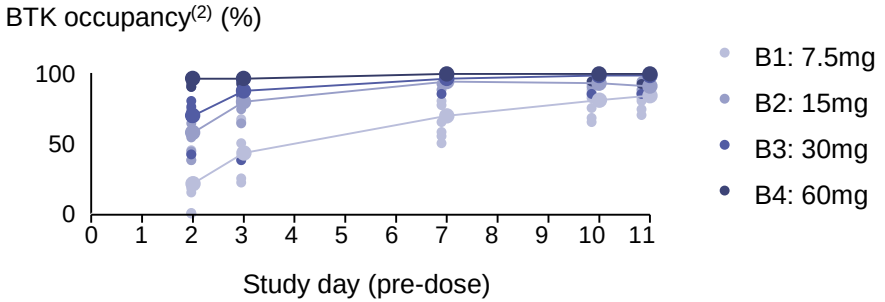
Pharmacologically relevant exposure in CNS demonstrated in Phase 1[(1)]

- Lumbar puncture performed 2hrs post-administration
- Mean cerebrospinal fluid concentration of 1.87 ng/mL (> cell-based in vitro IC50)



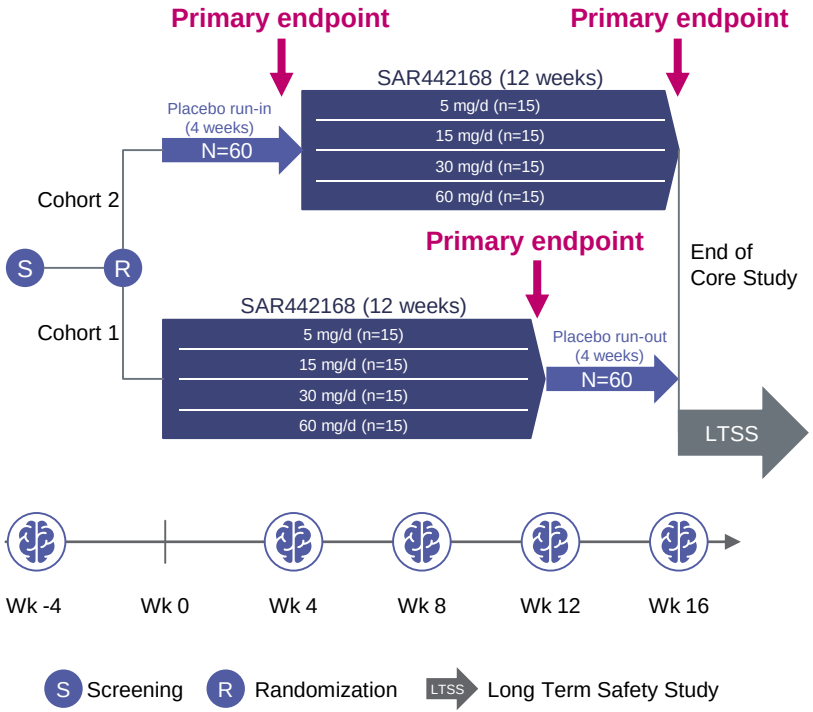
Demonstrated rapid and durable BTK target occupancy in Phase 1

- 100% target occupancy achieved on 15mg dose at steady-state



CSF=cerebrospinal fluid
Human phase 1 results presented at ACTRIMS forum 2019
(1) Data obtained from 4 participants administered a single 120 mg dose p.o. A single lumbar puncture 2 hours post-administration was used to measure drug in CSF (2) Levels of peripheral BTK occupancy were measured at 24 hours after dosing on Day 1, pre-dose on days 3, 7, and 10, and at multiple timepoints out to 168h (7 days) after administration of the final dose.
BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators

Phase 2b study designed to assess dose and minimize exposure to placebo



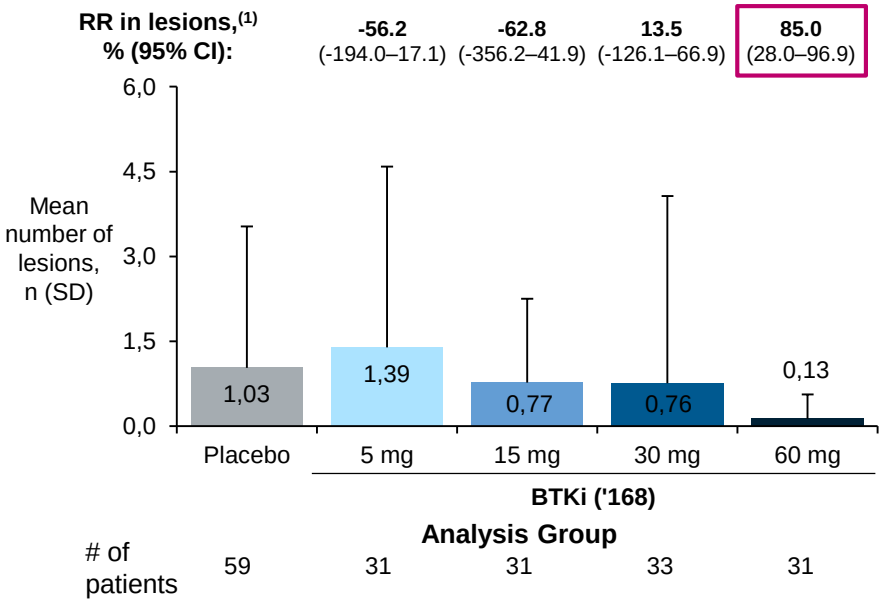
- Randomized, double-blind, placebo-controlled, cross-over, dose-ranging Phase 2 study
- Patient characteristics at baseline consistent with a typical RMS Phase 2 population
- Primary endpoint: Number of new Gd-enhancing T1 hyperintense lesions versus 4 weeks of placebo(1)
- Secondary endpoints: Number of new or enlarging T2 lesions versus 4 weeks of placebo(1) and safety
- Patient discretion whether to take BTKi ('168) fed or fasted

MRI endpoints predictive of clinical outcomes in Phase 3 trials(2)

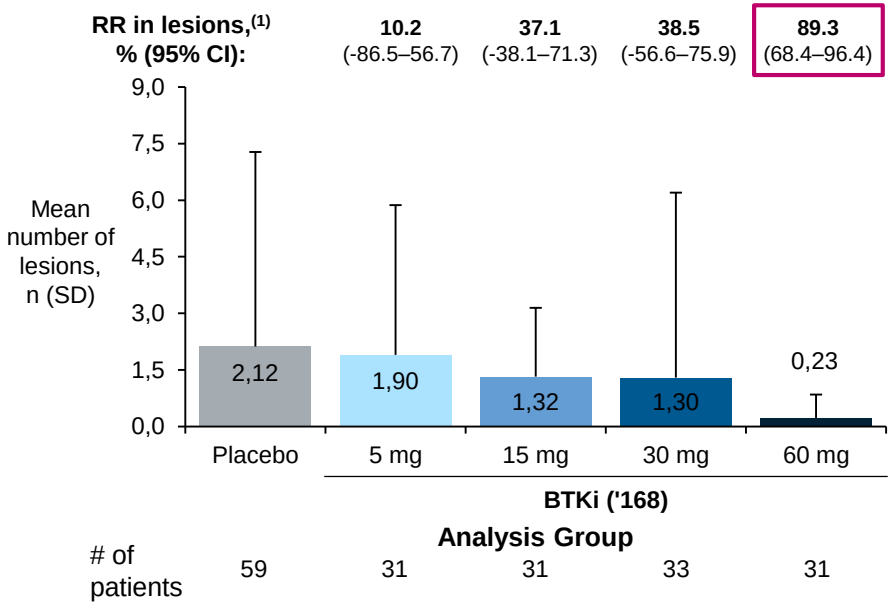
RMS: Relapsing Multiple Sclerosis; Wk=Week
(1) With adjustment for baseline activity within each dosing group
(2) Sormani MP, et al. Ann Neurol 2009;65:268-75; 2. Sormani MP, et al. Neurology 2010;75:302-9.
BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators.

85% relative reduction in Gd+ lesions at 12w on 60mg

Primary endpoint: number of new Gd+ lesions after 12 weeks



Secondary endpoint: number of new or enlarging T2 lesions after 12 weeks



CI: Confidence Interval; RR: relative reduction
Values are based on 12 weeks of SAR442168 exposure (Cohort 1: Week 12; Cohort 2: Week 16), or 4 weeks of placebo for Cohort 2 patients. Relative reductions in lesions were adjusted for baseline Gd-enhancing T1-hyperintense/ T2-hyperintense lesion activity using a negative binomial model
(1) Relative reduction in lesions versus placebo
BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators

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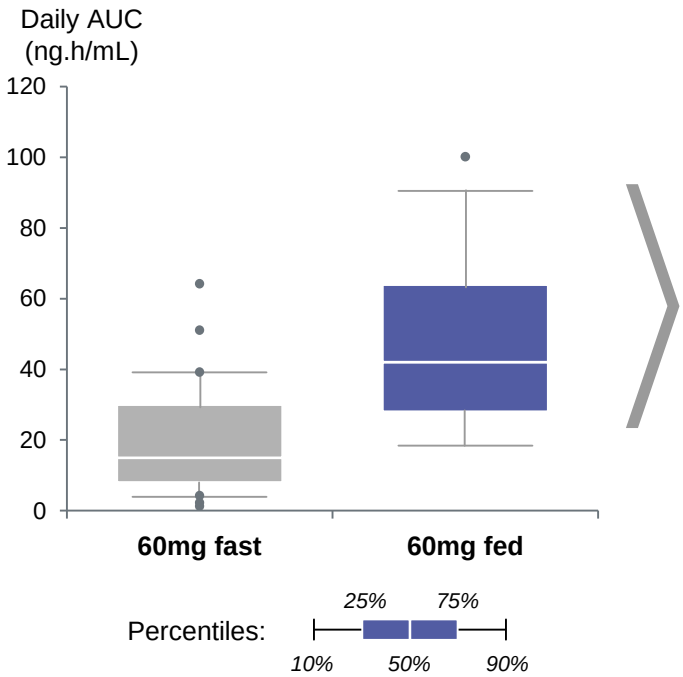
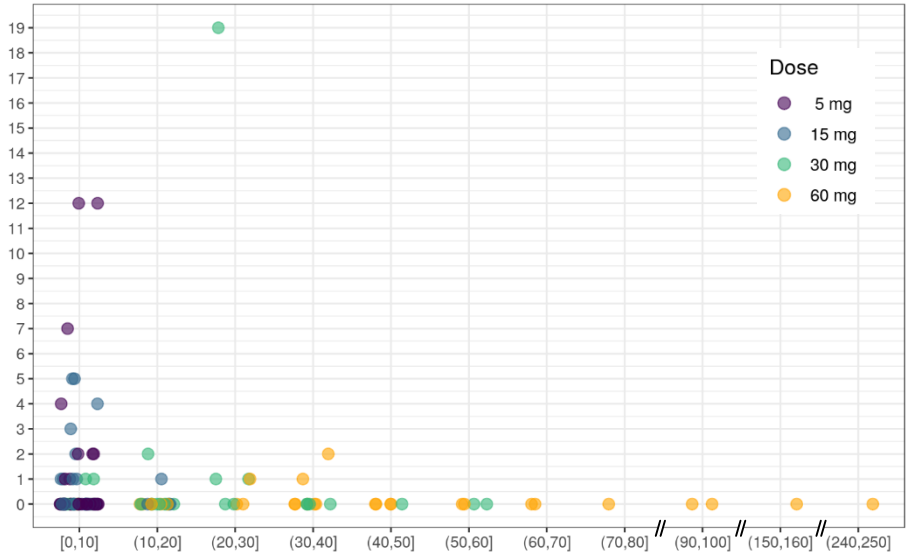
Phase 3 dosing (60mg fed) expected to maintain consistent exposure to achieve AUC

Higher AUC in fed patients
(Phase 1 data)



Higher response at higher exposure
(Phase 2 data)

New Gd-Enhancing T1-hyperintense lesions



AUC bins (ng.h/mL)

AUC: Area Under Curve
AUC values are averaged AUC0-24 from three visits
BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators

BTKi ('168) safety profile in line with placebo

'168 treatment, n (%)	AEs during weeks 1-4						AEs during 12 weeks				
	All Patients (N=130)	Placebo[1] (N=66)	5 mg (N=16)	15 mg (N=16)	30 mg (N=16)	60 mg (n=16)	All Patients (N=130)	5 mg (N=33)	15 mg (N=32)	30 mg (N=33)	60 mg (n=32)
Any AE (4 weeks vs. placebo | 12 weeks)	38 (29.2)	23 (34.8)	5 (31.3)	3 (18.8)	2 (12.5)	5 (31.3)	70 (53.8)	19 (57.6)	17 (53.1)	18 (54.5)	16 (50.0)
Serious AE	-	-	-	-	-	-	1 (0.8)	-	-	-	1 (3.1)
AE leading to death	-	-	-	-	-	-	-	-	-	-	-
AE leading to study discontinuation	-	-	-	-	-	-	-	-	-	-	-
Any AE leading to study treatment discontinuation	-	-	-	-	-	-	-	-	-	-	-
Any treatment-related AE	10 (7.7)	7 (10.6)	2 (12.5)	-	-	1 (6.3)	17 (13.1)	5 (15.2)	1 (3.1)	4 (12.1)	7 (21.9)

After 4 weeks: One patient from the placebo group had elevated ALT
After 12 weeks: One serious AE – hospitalization for MS relapse – was reported in the 60 mg group; the patient recovered and remained on study treatment

AE: Adverse Events; ALT: alanine aminotransferase; ULN: Upper Level Normal
(1) Includes Cohort 2 placebo arm only, which began SAR442168 treatment at Week 4
After 12 weeks: One patient from each of the 30 mg and 60 mg groups had elevated ALT >3 times ULN; the 60-mg patient had elevated ALT at baseline (48-50 vs ULN of 34 units per liter); both patients remained on treatment
BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators

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Sampling of Phase 2 study results in RMS

Molecule	Design, Duration	Primary endpoint	Relative Reduction in T1 lesions vs. PBO[1]		Secondary endpoint	Relative Reduction in T2 lesions vs. PBO[1]	
Modern phase 2 design							
BTKi ('168)[2]	**Placebo-controlled for 4Wk, with 12Wk cross-over (N=130), 16Wk+ext**	**Dose-response for Gd+ lesions at Wk 12**	85%	**60mg qd**	**Dose-response for T2 lesions at Wk 12**	89%	**60mg qd**
siponimod[3]	Placebo-controlled, adaptive, dose-ranging (N = 297), 6m + ext	Dose-response for CUAL at 3 mo	72%[10]	2mg qd	New or newly enlarged T2 lesions at 3mo	73%	2mg qd
ofatumumab[4]	Placebo-controlled (N=231), 24Wk + ext	Cumulative Gd+ lesions at Wk 12	91%[11]	60mg q12w[11] *65% 0-12Wks[12]*	New or newly enlarging T2 lesions at Wk 12	90%[11]	60mg q12w[11] *60% 0-12Wks[12]*
Traditional phase 2 design							
ocrelizumab[5]	Placebo-controlled + Inf-b1a reference arm (N=218), 24Wk + ext	Cumulative Gd+ lesions at Wk 12, 16, 20, and 24	89%	600mg q6mo	New or enlarging T2 lesions at Wk 24	100%	600mg q6mo
evobrutinib[6]	Placebo-controlled + open label DMF (N = 267), 24Wk + ext	Cumulative Gd+ lesions at Wk 12, 16, 20, and 24	70%	75mg qd (56% at 75mg bid)	New or enlarging T2 lesions at Wk 12, 16, 20 and 24	50%	75mg qd (58% at 75mg bid)
dimethyl fumarate[7]	Placebo-controlled (N = 257), 24Wk + ext	Cumulative Gd+ lesions at Wk 12, 16, 20, and 24	69%	240mg tid	New or enlarging T2-hyperintense lesions	48%	240mg tid
fingolimod[8]	Placebo-controlled (N = 281), 6m + ext	Cumulative Gd+ lesions monthly for 6 months	61%	5mg qd (88% at mo. 6)	Cumulative numbers of new T2 lesions	70%	5mg qd
teriflunomide[9]	Placebo-controlled (N = 179), 36Wk + ext	# of CUAL per MRI scan	61%[13]	14mg qd	New or enlarging T2 lesions	43%	14mg qd

(1) Approved dose for fingolimod = 0.5mg qd, dimethyl fumarate = 240mg bid, teriflunomide = 14mg qd, ocrelizumab = 600mg q6mo, siponimod = 2mg qd, Ph3 dose for evobrutinib is 75mg bid (2) ClinicalTrials.gov identifier: NCT03889639 (3) Selmaj K, et al Lancet Neurol 2013;12:756-767 (4) Bar-Or A. et al, Neurology 2018;90:e1805-e1814 (5) Kappos L, et al. Lancet 2011;378:1779-87 (6) Montalban X, et al. N Engl J Med 2019; 380:2406-2417 (7) Kappos L, et al. Lancet 2008;372(9648):1463-72 (8) Kappos L, et al. N Engl J Med 2006; 355:1124-40 (9) O'Connor P, et al. Neurology 2006;66(6) (10) New gadolinium-enhancing lesions (GdEs) on T1-weighted images and new or newly enlarged non-enhancing lesions on T2-weighted monthly MRI scans (without double counting) (11) Post hoc data (4-12 wks) (12) Endpoint with full data (0-12 Wks) (13) New and persisting T1 and T2 lesions

Note: No head to head studies have been conducted comparing the investigational treatment BKTi ('168) with any other therapy. The information on this slide is for the purpose of illustrating the novel design of the BKTi ('168) phase 2b study and Sanofi's decision to continue to study BKTi ('168) in phase 3 trials based on the results of the phase 2b study. The studies listed on this slide involve different study designs, patient populations, and endpoints, and cross trial comparisons of the endpoints should not be made.

BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators

Progression in MS and underlying mechanisms

Rita Balice-Gordon

Therapeutic Area Head,
Neurologic and Rare Diseases Research

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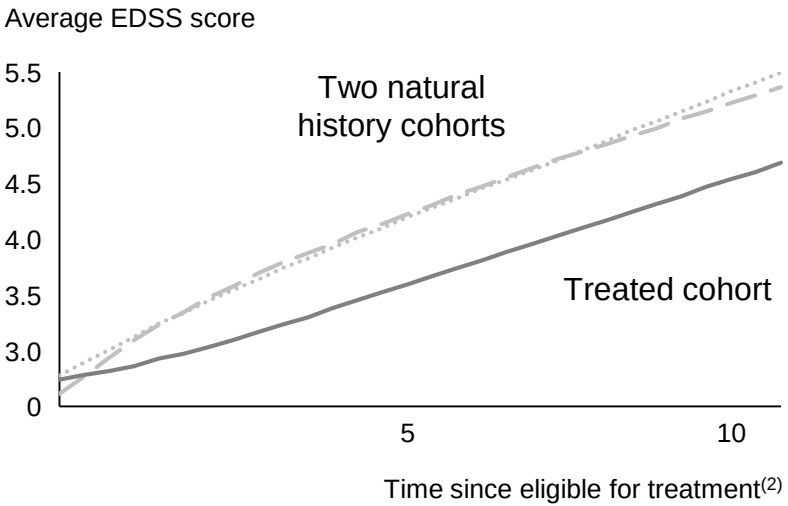
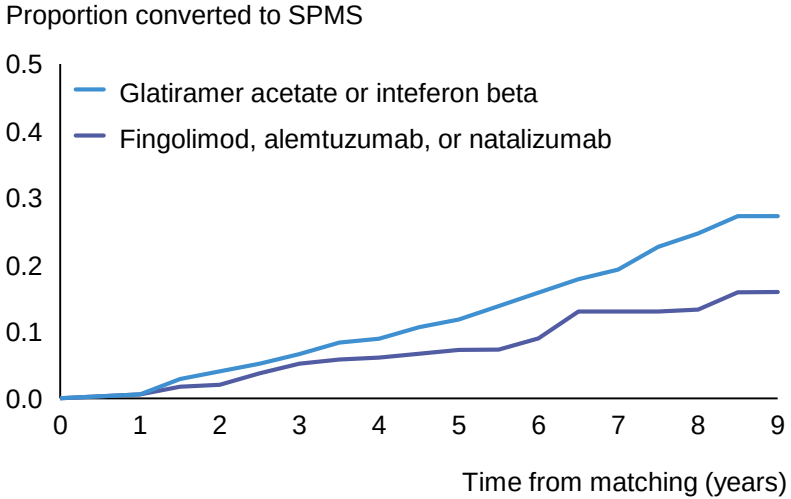
Patients continue to accumulate significant disability with currently approved therapies

Patients continue to accumulate disability[1]



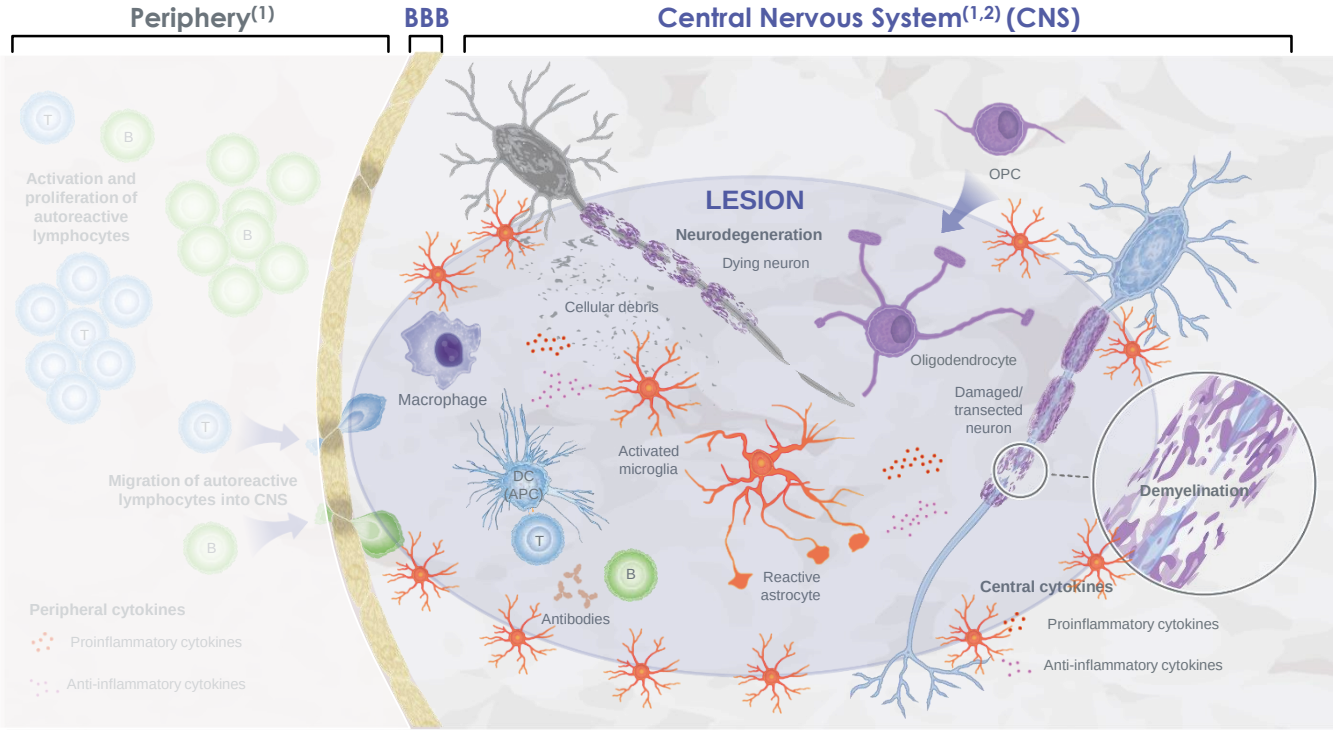
20% of patients still progress in less than 10 years despite current therapies[3]



EDSS: expanded disability status scale; SPMS: secondary progressive multiple sclerosis

(1) Tilling K et al. Health Technol Assess 2016;20:1-48
(2) Criteria for eligibility: age ≥18 years, EDSS score ≤6.5, occurrence of ≥2 relapses in the previous 2 years
(3) Brown J et al. JAMA. 2019;321(2):175-187; based on 380 patients (glatiramer acetate or interferon beta) + 235 patients (fingolimod, alemtuzumab or natalizumab) with follow-up data

Missing link: progression in MS involves mechanisms within the CNS



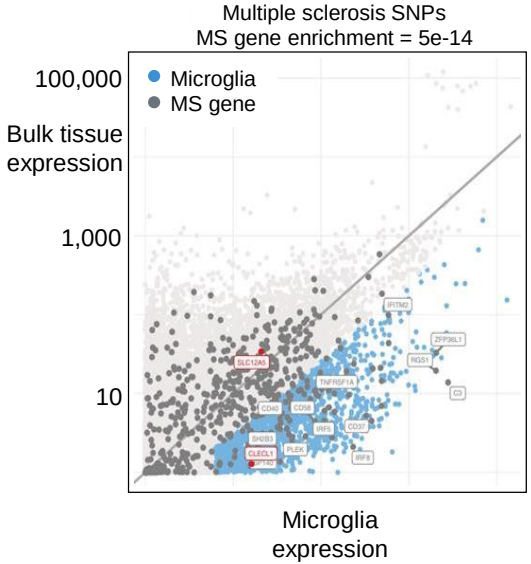
- In the CNS, the presence of proinflammatory factors and microglial activation results in oligodendrocyte and myelin damage
- These lead to axon degeneration, neuron damage and cell death, which exacerbate inflammation in the CNS
- These mechanisms contribute to disease progression and exacerbate disability

BTK inhibition has the potential to play a critical role to reduce microglial activation, re-establish microglial homeostasis and affect MS disease progression and disability accumulation

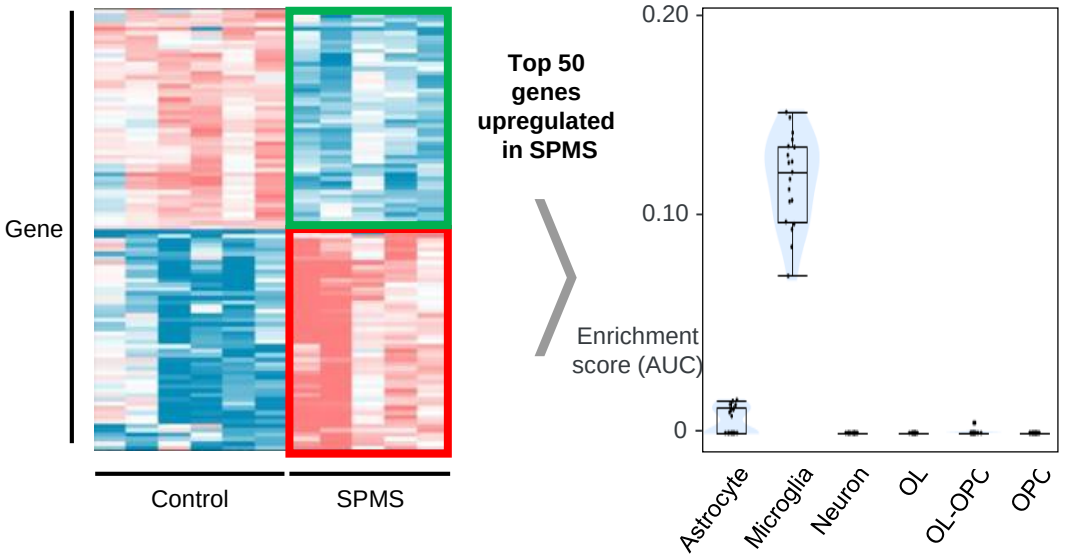
APC: antigen-presenting cell; BBB: blood-brain barrier; DC: dendritic cell; OPC: oligodendrocyte progenitor cell.
(1) Reich DS et al. N Engl J Med 2018;378:169-80. (2) Gandhi R et al. J Neuroimmunol 2010;221:7-14.
BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators.

Microglia play a central role in MS disease progression

GWAS identified enrichment of MS genes in microglia[1]



Transcriptome signatures reveal top upregulated genes in progressive MS are most highly expressed in microglia[2]



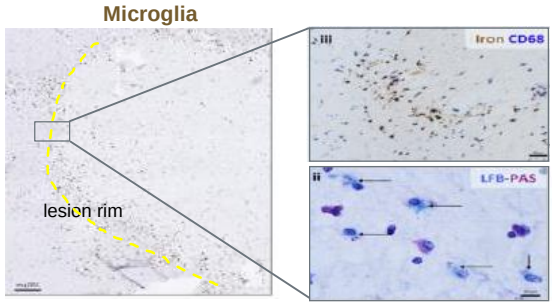
GWAS: Genome-Wide Association Study; SNP: Single Nucleotide Polymorphism; SPMS: Secondary Progressive Multiple Sclerosis; OL: Oligodendrocyte; OL-OPC: OL Progenitor Cells

(1) International Multiple Sclerosis Genetics, Consortium (2019) Science 365: 6450 (2) Proprietary Sanofi analysis

BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators

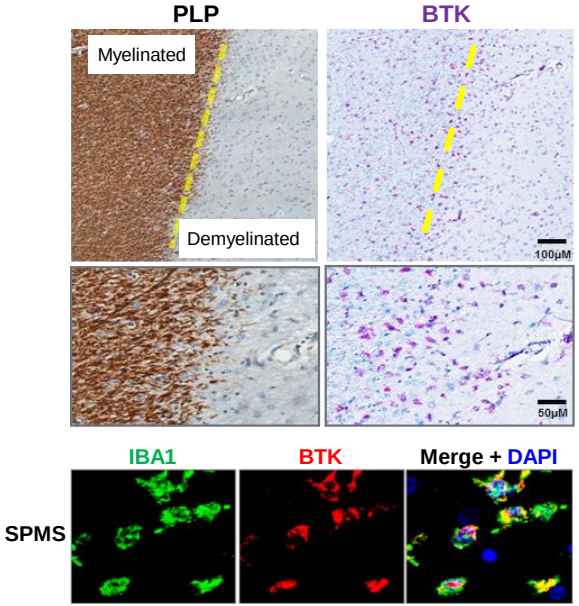
Microglia from lesions of progressive MS patients express high levels of BTK

Microglia are increased in lesion rim

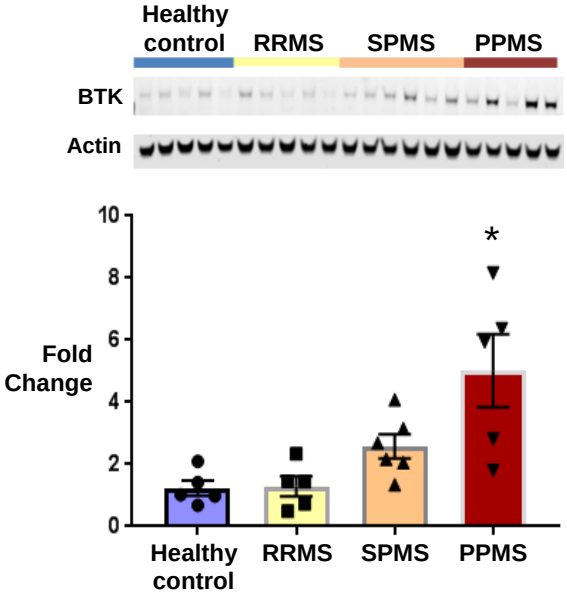


Modified from Absinta et al., (2016) *J Clin Invest*

BTK+ cells are microglia and increased in lesion rim



BTK protein is upregulated in lesions



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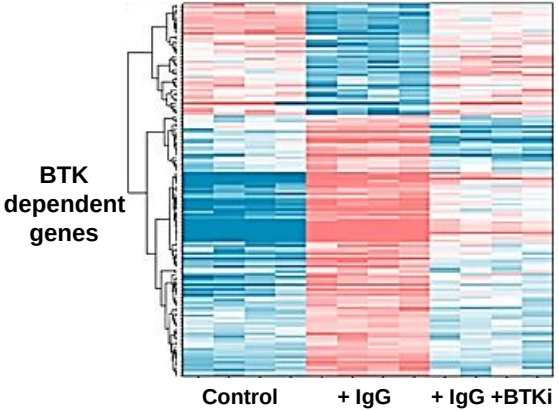
CTL: healthy control; mRNA:messenger ribonucleic acid; NAWM:normal-appearing white matter; PLP:proteolipid protein 1; PPMS:primary progressive MS; RRMS:relapsing-remitting MS; SPMS: secondary progressive MS

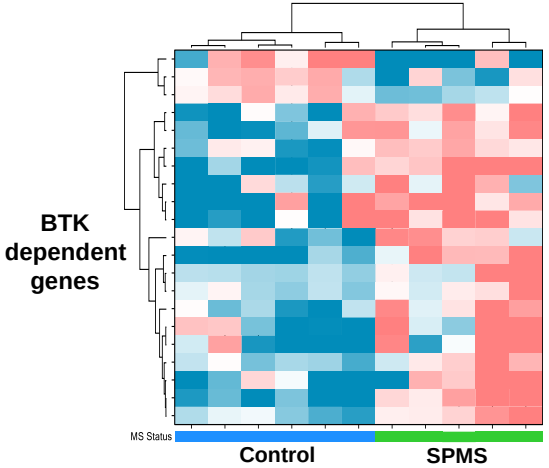
BTK inhibition normalizes proinflammatory signature in microglia

BTK inhibition normalizes the signature of mouse microglia activated by IgG



Upregulated pathways(1)	-log(p-value)
HMGB1 Signaling	7.52
Leukocyte Extravasation Signaling	7.08
PPAR Signaling	5.18
Neuroinflammation Signaling	5.05
Toll-like Receptor Signaling	4.74
Systemic Lupus Erythematosus B Cell Signaling	4.45
IL-6 Signaling	4.42

BTK signature of mouse microglia training set segregates SPMS patient lesions from control



Gene Symbol	Gene Name	Ratio (SPMS vs. Control), log2
SPP1	Secreted phosphoprotein 1	3.14
RGS1	Regulator of G protein signaling 1	2.59
CX3CR1	C-X3-C motif chemokine receptor 1	-2.1
P2RY12	Purinergic receptor P2Y12	-1.8

SPMS: secondary progressive MS

(1) Ingenuity Pathway Analysis® Hendricks RW et al. *Nature Chem Biol* 2011;7:4-5.

BTKi ('168) is an asset under investigation in collaboration with Principia, not approved by regulators

BTK is a key regulator for microglial homeostasis

Scientific evidence

- Microglia play a central role in MS disease progression, as well as in other neurodegenerative disorders
- Microglia from progressive MS patients exhibit an altered, proinflammatory phenotype
- BTK inhibition reduces the proinflammatory microglia signature and restores microglia to a homeostatic phenotype

BTK inhibition has the potential to

- Reduce microglial activation
- Re-establish microglial homeostasis
- Affect disease progression and disability accumulation in MS

BTKi ('168) is an asset under investigation in collaboration with Principia, not approved by regulators

Phase 3 plan

John Reed

EVP, Global Head of R&D

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Launching trials for BTKi ('168) across full MS spectrum

	Phase 3 program			Long Term Study Relapsing (RMS)
	Relapsing (RMS)	Primary Progressive (PPMS)	Non Relapsing Secondary Progressive (NR-SPMS)	
Comparator	vs. Aubagio®	vs. Placebo	vs. Placebo	-
Opportunity	~900K diagnosed(1) Disability accumulates despite treatment	~120K diagnosed(1) Only one approved DMT with modest efficacy(2)	~172K diagnosed(1) No approved DMTs for SPMS without relapses	Confirmation of LT efficacy and safety profile
Target #of patients	**N = 900 + 900**	**N = 1200**	**N = 1290**	**N = 123**

DMT: disease modifying therapy; LT: Long-Term
(1) Source: Sanofi analysis of U.S. and EU5 (UK, France, Germany, Italy, Spain)
(2) Ocrelizumab: 24% relative reduction of 12-week confirmed disability progression; Montalban X et al, N Engl J Med 2017 Jan 19;376(3):209-220
BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators

Exploratory imaging biomarkers to generate evidence for CNS activity

Slowly Enlarging Lesions (SELs)

- Chronic lesions seen on conventional T2 images
- Inactive center, surrounded by a rim of activated microglia / macrophages
- Relatively resistant to current DMTs

Markers of chronic tissue loss in the absence of ongoing acute inflammation[1]

Phase Rim Lesions (PRLs)

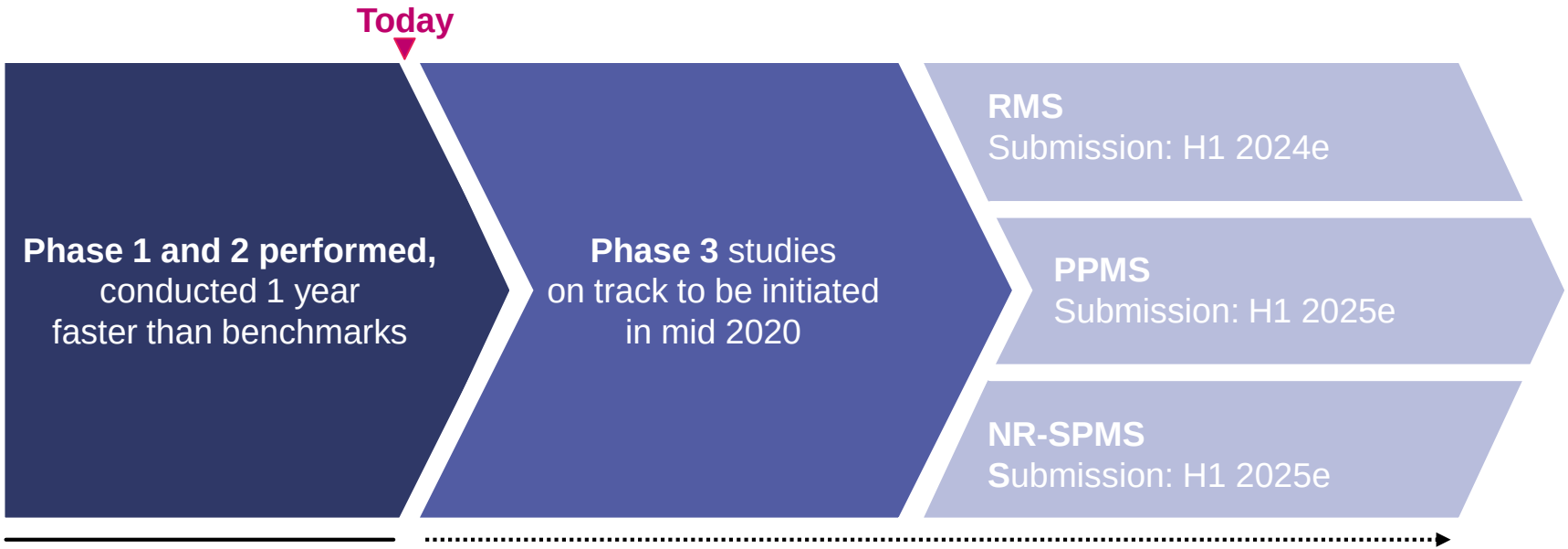
- Chronic lesions seen on Susceptibility Weighted Imaging (SWI)
- Paramagnetic rim seen as a hypointense boundary surrounding a region of tissue loss, corresponding to iron-laden microglia[2]

Markers of disability accumulation[3]
(correlated with number and volume of lesions)

Additional update to be provided at an upcoming conference

(1) Elliot C. et al, MSJ 2019, 25(14), 1915–1925 (2) As shown by histopathology of autopsy tissue (3) Absinta M. et al, JAMA Neurol. 2019;76(12):1474-1483

Beating industry cycle times to target submission in H1'24



(1) BTKi : estimate cycle times
(2) KMR and CMR: 2013-2018 data from Neurology indications
Estimated regulatory submission timing has not been reviewed by any regulatory authority.
BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators.

Conclusion: BTKi ('168) targets BiC profile

Safety	Similar to placebo
Low treatment burden	Oral once-daily, no monitoring
Relapse rate reduction	In line with anti-CD20
Slowing disability in RMS	Only BTKi with demonstrated CNS penetration and engagement of potential markers of disability progression
Efficacy in progressive disease	Accelerated development across full MS spectrum: RMS, PPMS and NR-SPMS, with first target submission in H1 2024

Delivering BTKi ('168) target product profile expected to result in leading market position

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BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators.
BiC: best in class

Q&A session

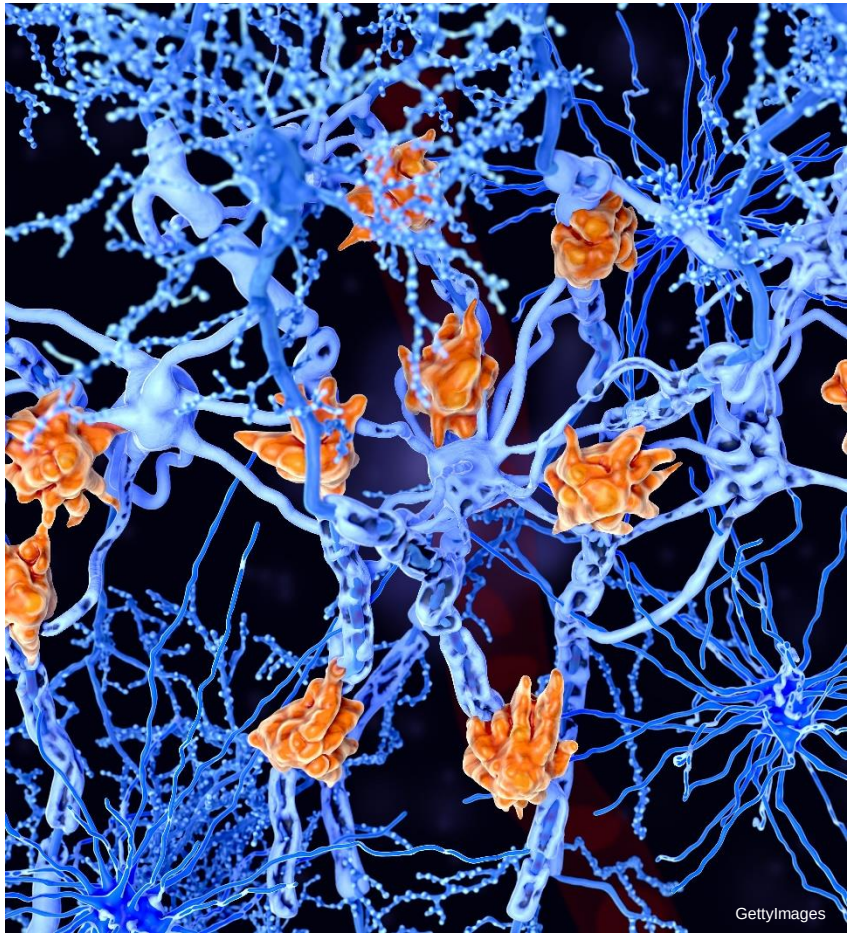
John Reed
EVP, Global Head of R&D

Rita Balice-Gordon
Therapeutic Area Head, Neurologic and Rare Diseases Research

Erik Wallstroem
Therapeutic Area Head, Neurology Development

Bill Sibold
EVP, Specialty Care

Tom Snow
Global Franchise Head, Neurology and Immunology



Phase 2b BTKi (‘168) Trial Results

Appendices

April 23, 2020

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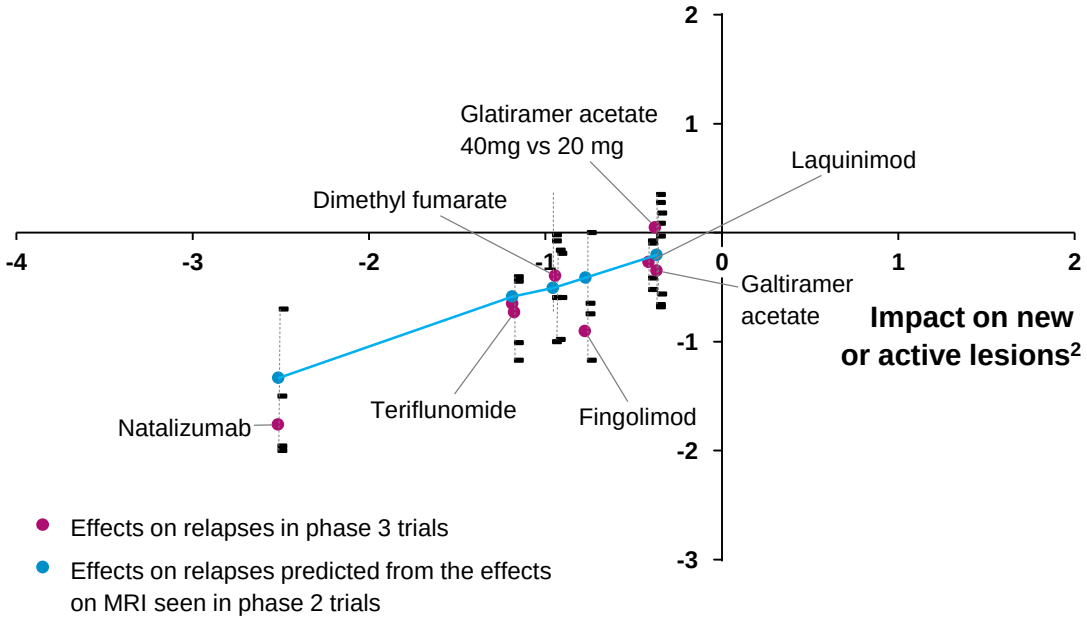
MRI is a predictive biomarker for relapses in MS

Impact on relapses(1)



- Effect of MS treatments on relapses are correlated with effect on MRI lesions (GdE and T2 lesions)
- Effects on MRI lesions over short follow-up periods are predictive of effects on relapses over longer follow-up periods

(1) Ratio between the annualized relapse rate (ARR in experimental group and the control group – log scale
(2) Ratio between the mean cumulative number of new or active T2 lesions, or GdE lesions, per patients in the experimental group and the control group – log scale
Source: Sormani et al. Lancet Neurology 2013

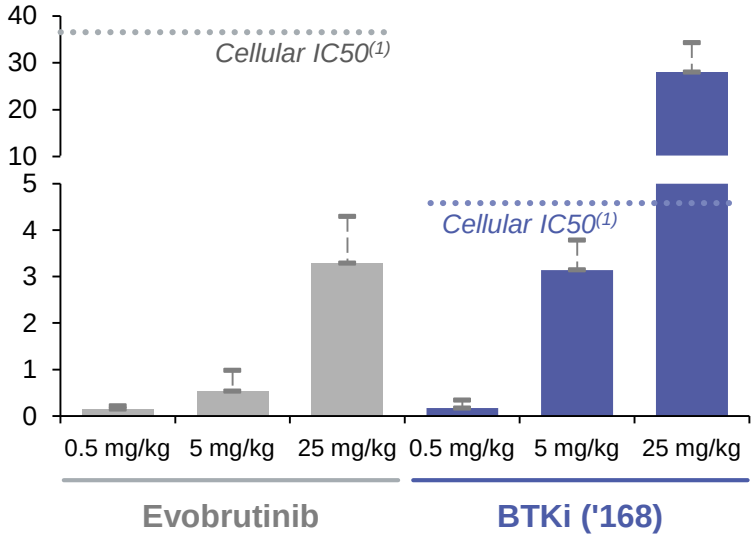
Patient Baseline Characteristics

			SAR442168			
	All Patients (N=130)	Placebo[1] (N=66)	5 mg (N=33)	15 mg (N=32)	30 mg (N=33)	60 mg (n=32)
Age, years	37.1 (9.5)	36.3 (9.8)	36.0 (9.8)	35.9 (9.3)	39.1 (10.2)	37.1 (8.8)
Female, n (%)	91 (70)	46 (70)	25 (76)	21 (66)	21 (64)	24 (75)
RRMS, n (%)	128 (99)	65 (99)	33 (100)	32 (100)	32 (97)	31 (97)
Time since initial relapse, years	7.8 (7.4)	7.7 (7.4)	7.7 (7.8)	8.0 (7.6)	8.1 (7.8)	7.3 (6.7)
Relapses in previous year	1.2 (0.6)	1.2 (0.5)	1.2 (0.5)	1.3 (0.6)	1.3 (0.6)	1.2 (0.4)
Relapses in previous 2 years	1.7 (0.9)	1.7 (0.7)	1.7 (0.8)	1.5 (0.8)	1.8 (1.1)	1.6 (0.9)
EDSS score, median (IQR)	2.5 (1.5–3.5)	2.5 (1.5–3.5)	2.5 (2.0–3.0)	2.0 (1.5–3.0)	2.5 (1.5–3.5)	2.5 (1.5–3.8)
Highly active disease, n (%)	61 (47)	29 (44)	12 (36)	19 (59)	16 (49)	14 (44)
Number of Gd-enhancing lesions	1.8 (4.7)	2.2 (5.9)	2.3 (5.9)	0.7 (1.8)	1.9 (4.9)	2.1 (4.9)
Patients with baseline Gd-enhancing lesions, n (%)	44 (35)[2]	25 (38)	11 (34)[3]	7 (23)[4]	11 (34)[3]	15 (47)

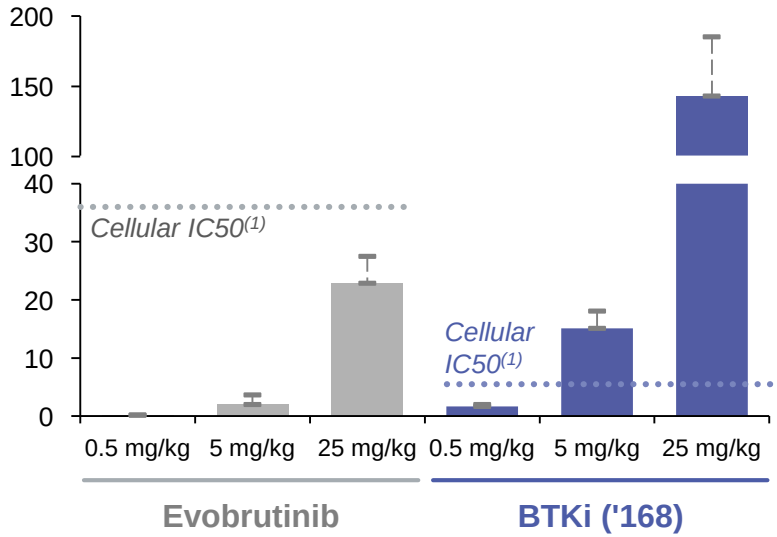
EDSS=Expanded Disability Status Scale; Gd=gadolinium; IQR=interquartile range; RRMS=relapsing-remitting multiple sclerosis; Values are mean (SD) except where noted; (1) Includes Cohort 2 placebo arm only, which began SAR442168 treatment at Week 4; (2) N=127; (3) N=32; (4) N=31. Highly active disease was defined as 1 relapse in the year prior to screening and ≥1 Gd-enhancing lesion on MRI performed within 6 months prior to screening or ≥9 T2 lesions at baseline or ≥2 relapses in the year prior to screening. BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators

CNS exposure and high potency allows ’168 to achieve pharmacologically relevant levels in the brain

Mouse cerebrospinal fluid
1hr post dose (ng/mL)



Mouse perfused brain
1hr post dose (ng/g)



(1) IC50 determined from human B cell activation (whole blood assay): Evobrutinib: 84.1 nM (from Haselmayer et Al, 2019 J Immunol. 2019 May 15;202(10):2888-2906); SAR442168: 10nM.
Source: Sanofi data on file. Mice were dosed orally with the indicated amount of Evobrutinib or SAR442168. CSF and well perfused brains were collected 1hr post-dose and assayed for compound exposure.
The clinical relevance of these pre-clinical findings is currently under investigation. This data is provided to illustrate the preclinical development of BTKi (‘168) and Sanofi’s decision to continue to evaluate BTKi (‘168) in clinical studies. BTKi (SAR442168) is an asset under investigation in collaboration with Principia, not approved by regulators