

Immunic Therapeutics Second Quarter 2022 Financial Results and Corporate Update

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Cautionary Note Regarding Forward-Looking Statements

This presentation contains "forward-looking statements" that involve substantial risks and uncertainties for purposes of the safe harbor within the meaning of Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended. These include statements regarding management's intentions, plans, beliefs, expectations or forecasts for the future, and, therefore, you are cautioned not to place undue reliance on them. No forward-looking statement can be guaranteed, and actual results may differ materially from those projected. Immunic undertakes no obligation to publicly update any forward-looking statement, whether as a result of new information, future events or otherwise, except to the extent required by law. We use words such as "anticipates," "believes," "plans," "expects," "future," "intends," "may," "will," "should," "could," "estimates," "predicts," "potential," "continue," "guidance," and similar expressions to identify these forward-looking statements that are intended to be covered by the safe-harbor provisions of the Private Securities Litigation Reform Act of 1995.

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Agenda Second Quarter 2022 Financial Results and Corporate Update



Second Quarter 2022 and Subsequent Highlights

02 Clinical Updates

03 Financial and Operating Results



Anticipated Clinical Milestones

05 Q&A Session



Summary and Highlights



Second Quarter 2022 and Subsequent Highlights

May: Announced Start of Celiac Disease Cohorts in Ongoing Phase 1 Clinical Trial of IMU-856

Double-blind, randomized, placebo-controlled phase 1 study performed in three parts:

- Safety and pharmacokinetics in healthy human subjects (Part A: single ascending dose, Part B: multiple ascending dose)
- Part C includes a celiac disease patient population, designed to assess safety and tolerability of IMU-856 as well as pharmacokinetics and disease markers

On May 5, 2022, Immunic announced the start of the celiac disease patient cohorts, representing the first time patients will be treated with the orally available small molecule modulator targeting restoration of intestinal barrier function and regeneration of bowel epithelium

Exclusive global rights to commercialization of IMU-856 in all countries obtained through option and licensing agreement with Daiichi Sankyo





June: Reported Top-Line Data from Phase 2 CALDOSE-1 Trial of Vidofludimus Calcium in Patients with Moderate-to-Severe UC

- Revealed a previously unknown interaction with chronic concurrent steroid use, resulting in missing the trial's primary endpoint
- Announced that the company's development programs in the inflammatory bowel disease indications will not be continued without a partner

June: Announced Publication of Data from Phase 2 EMPhASIS Trial of Vidofludimus Calcium in RRMS in Peer Reviewed Journal

of Clinical and Translational Neurology

RESEARCH ARTICLE

A double-blind, randomized, placebo-controlled phase 2 trial evaluating the selective dihydroorotate dehydrogenase inhibitor vidofludimus calcium in relapsing-remitting multiple sclerosis

Robert J. Fox¹, Heinz Wiendl², Christian Wolf³, Nicola De Stefano⁴, Johann Sellner⁵, Viktoriia Gryb⁶, Konrad Rejdak⁷, Plamen Stoyanov Bozhinov⁸, Nataliya Tomakh⁹, Iryna Skrypchenko¹⁰ & Andreas R. Muehler¹¹

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- Published in the peer reviewed journal, Annals of Clinical and Translational Neurology
- Authored by coordinating investigator Robert J. Fox, M.D., Staff Neurologist, Mellen Center for Multiple Sclerosis, Vice-Chair for Research, Neurologic Institute, Cleveland Clinic, Cleveland, Ohio



July: Announced Appointment of Maria Törnsén to Board of Directors



- Industry executive with 20 years of global commercial experience in U.S. and ex-U.S. markets
- Jan Van den Bossche resigned from the Board
- Both effective July 5, 2022



Clinical Updates

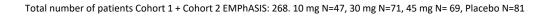
EMPhASIS Trial: Post-Hoc Analysis Shows That Concomitant Corticosteroids Treatment in RRMS Patients is Rare and Short



Concomitant Corticosteroids Treatment Was Rare

- Overall, few patients required concurrent corticosteroid treatment and, when needed, only one or very few courses were required
- A total of only N= 52/268 (19 %) patients had any use of corticosteroid during the main treatment period with an average duration of only 4.4 days

N of Corticosteroid Courses Given	0	1	2	>2
N of Patients	216	43	6	3
	(80 %)	(16 %)	(2 %)	(0.7 %)





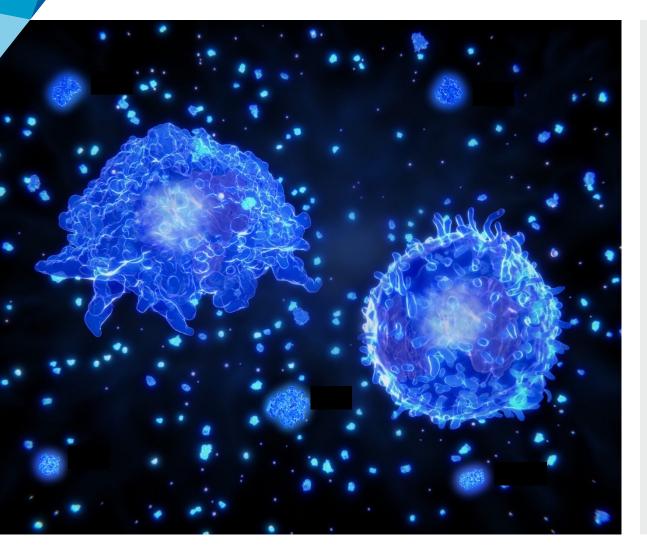
Corticosteroids were used only as short-term concomitant medication for:

- MS relapse (N=48)
- COVID-19 infection (N=1)
- Eczema (N=1)
- Acute bronchitis (N=1)
- Contact urticaria (N=1)

Average Duration of Each Corticosteroid Treatment Course (Pooled Cohorts 1+2)	4.4 days
Minimum	1 day
Maximum	10 days



Exploratory Phase 1 Drug-Drug Interaction (DDI) Study of IMU-935 Raised No Concerns



- Exploratory phase 1 study completed in 15 evaluable healthy human subjects to assess the DDI potential of IMU-935
- No relevant signals for DDI potential observed
- Treatment was safe and welltolerated



Update on Phase 1 Clinical Trial of IMU-935 in mCRPC Initial Safety Data Available

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Initial safety data available so far show a promising safety profile of IMU-935 in metastatic castration-resistant prostate cancer (mCRPC).

- Open-label dose escalation trial to evaluate safety, tolerability, anti-tumor activity, and pharmacokinetics of IMU-935 in patients with progressive, metastatic castration-resistant prostate cancer
 - First two dose cohorts fully recruited, 6 patients enrolled in the 300 mg cohort and 6 patients in the 600 mg cohort
 - Of these patients, all have completed initial 28-day safety study part without reaching dose limiting toxicity (DLT)
 - Third, 900 mg cohort expected to start dosing soon
- Initial safety data available so far show a promising safety profile of IMU-935 in mCRPC, with only benign adverse events and no dose limiting toxicities.
- Immunic plans to provide a more comprehensive update on safety and also on potential signs of anti-tumor activity of IMU-935 in this trial as soon as data from the planned dose expansion part are available.



Principal Investigator

Johann Sebastian de Bono, M.D., Ph.D.

Regius Professor of Cancer Research and Professor in Experimental Cancer Medicine

The Institute of Cancer Research and The Royal Marsden NHS Foundation Trust

London, United Kingdom



NCT05124795

Financial and Operating Results

Consolidated Statements of Operations

(In thousands, except share and per share amounts, unaudited)

		Months June 30,	Six Months Ended June 30,		
	2022	2021	2022	2021	
Operating expenses:					
Research and development	\$ 16,538	\$ 15,738	\$ 33,983	\$ 27,257	
General and administrative	4,072	3,432	8,062	7,050	
4SC royalty settlement	—	-	_	17,250	
Total operating expenses	20,610	19,170	42,045	51,557	
Loss from operations	(20,610)	(19,170)	(42,045)	(51,557)	
Other income (expense):					
Interest income	106	13	113	41	
Other income (expense), net	(1,397)	1,223	(777)	(952)	
Total other income (expense)	(1,291)	1,236	(664)	(911)	
Net loss	\$ (21,901)	\$ (17,934)	\$ (42,709)	\$ (52,468)	
Net loss per share, basic and diluted	\$ (0.72)	\$ (0.82)	\$ (1.49)	\$ (2.44)	
Weighted-average common shares outstanding, basic and diluted	30,248,767	21,749,439	28,686,910	21,463,656	

\$88.1 million in cash and cash equivalents as of June 30, 2022 are expected to **fund operations into the fourth quarter of 2023**



Anticipated Clinical Milestones

Vidofludimus Calcium in Multiple Sclerosis Executive Summary

Phase 3 program of vidofludimus calcium in RMS ongoing based on **excellent clinical data** package

New third-party data clearly highlights the unmet need of **preventing disability progression**, which is seen across the spectrum of patients with MS

Vidofludimus calcium selectively manages all three components needed to **quell smoldering MS**

Large market opportunity exists for a therapy that can holistically and sustainably address patients' needs

- Demonstrated effect on all relevant endpoints in 268 RRMS patients, including anti-inflammatory & neuroprotective effects
- Unrivaled safety to date, with over 1,100 individuals treated
- The understanding of MS has evolved, with evidence showing a smoldering disease that is connected to Epstein-Barr virus and subsequent inflammation & neurodegeneration
- Anti-viral effect
- Anti-inflammatory effect
- Neuroprotective impacts
- Even current market leaders only optimize for one feature
- Most treatment options have series of risks / downsides



Key Publications in 2022 Provide Clear Evidence of a Direct Link Between Epstein-Barr Virus and MS

- Epidemiologic study showed a clear association between EBV infection and occurrence of MS^[1]:
 - 32-fold increased risk in EBV-infected patients
 - Serum levels of neurofilament light chain increased only after EBV seroconversion
- Cross-reactive antibodies between EBV antigen EBNA1 and CNS protein GlialCAM found in the CSF of MS patients^[2,3]
 - Proof of mechanistic link between EBV and MS
 - Anti-CD20 antibodies deplete B cells, but do not deplete their progeny (antibody-producing plasma cells, which are CD20 negative).

RESEARCH					_	Science		PERSPECTIVE
REPORT			dates of collection	nch of military service, a of blood samples who w a duty when the case w	rere			Cite as: W. H. Robinson, L. Steinman Science 10.1126/science.abm7930 (2022
Longitudinal analysis reveals high prevale		valence of diagnosed (Fig. 1A and fig. S1). There were 801 MS cases and 1566 controls with samples available to assess EBV infection status. Most		rere ples fost	Epstein-Barr virus and multiple sclerosis			
Epstein-Barr virus associated w	with mult	tiple sclerosis	of age at the time o	n our study were <20 ye f their first blood collect	tion	William H. Robinson ¹	^{,2} and Lawrence Steinman ³	
Kjetil Bjornevik ¹ †, Marianna Cortese ¹ †, Brian C. Healy ^{2,3,4} , Yumei Leng ⁶ , Stephen J. Elledge ⁶ , David W. Niebuhr ² , Ann I Kassandra L. Munger ¹ ‡, Alberto Ascherio ^{1,10,33} ‡	, Jens Kuhle ⁵ , M I. Scher ⁹ ,	ichael J. Mina ^{6,28} ,		e who developed MS I nedian of 10 years after ti ;; 1C).		¹ Division of Immunology and Rheu ³ Department of Neurology and Ne	eurological Sciences, Stanford University, Stanford, O	sity, Stanford, CA, USA. ?VA Palo Alto Health Care System, Palo Alto, CA, USA. 24, USA. Email: w.robinson@stanford.edu; steinman@stanford.edu
Multiple sclerosis (MS) is a chronic inflammatory demyeli	linating dis	Article					barr virus is the trigger for the	development of multiple sclerosis
system of unknown etiology. We tested the hypothesis th virus (EBV) in a cohort comprising more than 10 million y	hat MS is c		ovnon	dodDod	llcinmultin		in-Barr virus (EBV) has long been tiple sclerosis (MS) (1). Prior anal-	plasmablasts and plasma cells, which are CD20 ⁻ . The mechanism (or mechanisms) of EBV-mediated Mi
US military, 955 of whom were diagnosed with MS during					ells in multip		ased serum antibodies to EBV in	development remains elusive. Possibilities include molecula mimicry, through which EBV viral protein sequences mimi
MS increased 32-fold after infection with EBV but was no viruses, including the similarly transmitted cytomegalovir		colorosi	hind F	BV FRN	A1 and Glial	`A M	mpared with ~94% of healthy indi-	
light chain, a biomarker of neuroaxonal degeneration, incl	creased onl	SCIELOSI	5 DILIU E	DV EDIN	AI and Ghai		X of this issue, Bjornevik et al. (3)	
These findings cannot be explained by any known risk fac	actor for MS						in serum from 801 individuals who	
leading cause of MS.							phort of >10 million people active in	
							year period (1993-2013). Thirty-five initially EBV seronegative, and 34	
	ively rare c	https://doi.org/10.1038/s4			amille Brewer ¹⁴ , Peggy P. Ho ⁵ , Jae-Seur ardo A. Fernandes ⁶ , Aleiandro M. Gome		V before the onset of MS. EBV sero-	
	an invest ear collabe	Received: 6 August 2021			ardo A. Fernandes", Atejandro M. Gome *, Ryan D. Schubert*, Isobel A. Hawes*, :		quitous at the time of MS develop-	
	ave identi	Accepted: 14 January 202	2	Manasi Iyer", J. Bradley	Zuchero ¹¹ , Bianca Teegen ¹² , Jeffrey E. D	unn ¹² , Christopher B. Lock ¹² ,	1 MS cases being EBV seronegative	costimulatory pathway that is important for B cell-T cell ir
brain and spinal cord is an immune- comp	posed of a	Lucas B. Kipp ¹⁰ , Victoria C. Co			a C. Cotham ^{14,15} , Beatrix M. Ueberheide ¹	⁴³⁵ , Blake T. Aftab ³⁶ ,	These findings provide compelling	teraction. Additionally, EBV encodes an interleukin-10-lik
	nel betwee	Check for updates		Mark S. Anderson ¹⁰ , Jos Michael Platton ^{2,320} K	seph L. DeRisi ^{10,18} , Michael R. Wilson [®] , Ra Christopher Garcia ⁶ , Lawrence Steinm	achael J. M. Bashford-Rogers",	s the trigger for the development of	
	rse populat ve-duty me	Check for updates		michaet Flatten , K.	Christopher Garcia , Lawrence Stemm	an a muan A. Robinson		mediate bystander damage to the axon and its surrounding
	e start of r						a tropism for B cells develop into a	
	eafter, and			Multiple sclerosis (MS) is a heterogenous autoimmune disease in which			vous system (CNS)? In MS, there is gainst the myelin sheath and the ax-	
	i (>62 milli ie Departm			autoreactive lymphocytes attack the myelin sheath of the central nervous system.			mately, neurons themselves are in-	
	(DoDSR)			B lymphocytes in the cerebrospinal fluid (CSF) of patients with MS contribute to			s and their activated progeny,	
	he DoDSR			inflammation and secrete oligocional immunoglobulins ^{1,2} . Epstein–Barr virus (EBV)		egrin a4, which has adhesive prop-	There are multiple reports suggesting that molecula	
	of first sar infection			infection has been epidemiologically linked to MS, but its pathological role remains unclear ³ . Here we demonstrate high-affinity molecular mimicry between		body-producing cells to move from		
	of active d			remains unclear ² . Here we demonstrate nign-amnity molecular minicry between the EBV transcription factor EBV nuclear antigen 1 (EBNA1) and the central nervous		peripheral circulation and then	tients to the EBV small capsid protein BFRF3 cross-react wit	
ological studies. Evidence of causality, how- we for	ound that.			system protein glial cell adhesion molecule (GlialCAM) and provide structural and		rrier (BBB), where they take resi-	the cytoplasmic protein septin-9 and are associated with d	
	ative at the onding to				idence for its relevance. A cross-rea		l its internal lining (4). A distinct	myelination (10). Another study showed serum antibodie
	-negative		was initially identified by single-cell sequencing of the paired-chain B cell		esis of immunoglobulins by clonal	from MS patients are cross-reactive between amino acids 411		
	ction and	repertoire of MS blood and CSF, followed by protein microarray-based test			s within the brain. When these im- erebrospinal fluid (CSF) from pa-	440 of the viral protein EBV nuclear antigen 1 (EBNA-1) ar		
	e docume ng active-di		recombinantly expressed CSF-derived antibodies against MS-associated viruses.			ed to an electrophoretic gel, they	the human chloride-channel protein, anoctamin 2 (ANO2 which is associated with electrical conduction in axons (II	
	ng active-d 315 cases fre			Sequence analysis, annity measurements and the crystal structure of the EBNAT-		obility, called oligoclonal immuno-	MS serum antibodies targeting EBNA-1 residues 411–426 that	
nature," a longitudinal investigation of MS For e	each MS ca			peptide epitope in complex with the autoreactive rab tragment enabled tracking of		ting clonal expansions of plas-	cross-react with myelin basic protein have also been ident	
	m samples			the development of the naive EBNAT-restricted antibody to a mature EBNAT-		es target myelin-producing glial	fied (12). Clonally expanded antibodies in the CSF of MS pa	
	onset (the fi re disease				tive antibody. Molecular mimicry is		em (4).	tients targeting EBNA-1 residues 386-405 that cross-read
	s were mat				odification of GlialCAM. EBNA1 imm		identified EBV-infected B cells in	with the CNS cell adhesion molecule, glialCAM, have als
	viduals wit				odel of MS, and anti-EBNA1 and ant with MS. Our results provide a mec		5, 6). Understanding how infection	been described (4). It is intriguing that three contiguous r
					MS and EBV and could guide the de		s the pathology seen in MS is now	gions of mimicry have been reported in a small region of th
Department of Nutrition, Harvard T. H. Chan School of Public Health, Bos Neurology, Harvard Medical School, Boston, MA, USA. ⁴ Biostatistics Cente	ter, Massachus			therapies.	inio una cortana coura garacente ac		nding of the roles of these clonally	EBNA-1 protein; this may arise through immune surveillan
for Clinical Neuroimmunology and Neuroscience Basel (RC2NB), University Hughes Medical Institute, Department of Genetics, and Program in Virology, I Department of Immunology and Infectious Diseases, Harvard T. H. Chan Scho	ity Hospital Ba Harvard Medic						nablasts. Depletion of B cells with geting CD20 has emerged as one of	in a process called epitope spreading. Increased incidence of EBV infection is associated with
Department of Immunology and Infectious Diseases, Harvard T. H. Chan Scho Boston, MA, USA. ⁹ Department of Preventive Medicine and Biostatistics. Unifi	hool of Public H						pies for MS (7). However, because	other autoimmune diseases, including systemic lupus eryth
	int of Medicine,	The presence of oligoclo	nal bands (OCBs) in	CSF and the efficacy			lonal antibody therapies do not	
*Corresponding author. Email: aascheriöhtsph.harvard.edu These authors contributed equally to this work. These authors contributed equally to this work.		of therapies that deple	te B cells emphasize	e the importance of	The B cell repertoire in MS CSF		amounts, and moreover, antibod-	EBV serum antibodies after resolution of acute infection)
These authors contributed equally to this work.		B cells in the pathobiolo mumps, measles, varice			CSF and blood samples were obtained for onset of disease (clinically isolated synd		their progeny, antibody-producing	
		present in MS ^{4,5} , but their			of relapsing-remitting MS (n = 4). Pat			
Bjornevik et al., Science 375, 296–301 (2022) 21 January 2022	3	titres can be detected in	nearly 100% of pati	ents with MS before	>10 cells µl ⁻¹ were selected (Extended D	ata Table 1, Supplementary Dis-	2 scien	ce.org (Page numbers not final at time of first release)
		the development of clinic			cussion). Single B cells were sorted by			
		an epidemiological link t tious mononucleosis du			Fig. 1a, b). and characteristic phenotyp and CSF were observed ^{13,44} . These includ	ed high plasmablast (PR) counts		
		developing MS7. Molecula	r mimicry between vi	irus and self-antigens	in CSF compared to blood (Extended E	ata Fig. 1c, d), different expres-		
		is a potential mechanis	m that might explai	in this association ⁸ .	sion levels of $\alpha 4$ integrin and HLA-DR	in PBs but not in non-PB B cells		
		Antibodies against cert			 (Extended Data Fig. 1e-j, Supplementa of immunoglobulin G (IgG) in CSF PBs (
		patients with MS, includi (refs, 59-12), which we dese			of immunoglobulin G (IgG) in CSF PBs (We sorted PBs from blood and B cells fr			
		lar mimicry between EB	NA1 and GlialCAM. T	he potential signifi-	cytometry and sequenced their full-len	gth paired heavy-chain (HC) and		
		cance of this mimicry in	the pathophysiolog		light-chain (LC) VDJ regions15. Atotal of 13,			
		in detail.			PBs and 1,689 from CSFB cells passed filt	er thresholds. The CSF repertoire		
		A list of affiliations appears at the e	nd of the paper.					
					Nature	Vol 603 10 March 2022 321	1	

[1] Bjornevik K. et al., Science. 10.1126/science.abj8222 (2022) [2] Lanz, T.V. et al., Nature 603, 321–327 (2022) [3] Robinson WH, Steinman L. Science. 2022 Jan 21;375(6578):264-265 EBV: Epstein-Barr Virus; CNS: central nervous system; CSF: cerebrospinal fluid; CD20: B-lymphocyte antigen



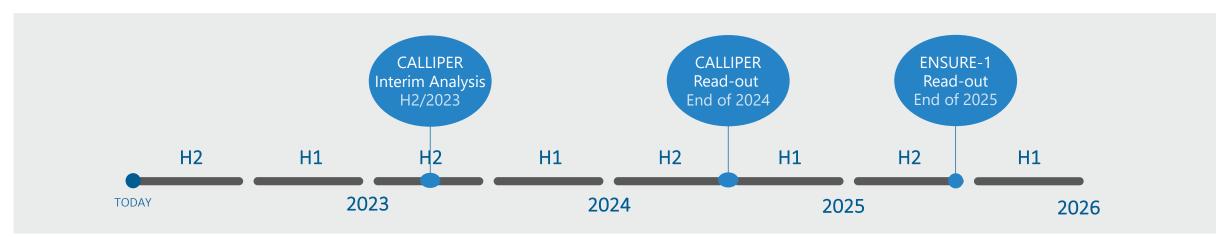
Vidofludimus Calcium in Multiple Sclerosis Straightforward Approval Strategy

Phase 3 ENSURE Program in RMS^[1]

- Two identical pivotal trials in RMS patients
- Goal: Low risk clinical program for regulatory approval of vidofludimus calcium
- Dosage: 30 mg vidofludimus calcium QD

Phase 2 CALLIPER Trial in PMS^[2]

- Phase 2 trial in PMS patients
- Goal: Demonstrate vidofludimus calcium's potential for neuroprotective activity in a nonrelapse setting
- Dosage: 45 mg vidofludimus calcium QD



[1] ClinicalTrials.gov: NCT05134441 & NCT05201638; [2] ClinicalTrials.gov: NCT05054140 RMS: relapsing multiple sclerosis; PMS: progressive multiple sclerosis; QD: quaque die = once-daily



IMU-935 Phase 1 Clinical Trial Part C in Moderate-to-Severe Psoriasis Patients



Recruitment is ongoing in Australia, New Zealand and Bulgaria.

Initial clinical activity results are expected to be available in Q4/2022.



IMU-856 Phase 1 Clinical Trial



Unblinded safety data from the single and multiple ascending dose parts of IMU-856 in healthy human subjects are expected to be available in Q3/2022.



Q&A Session

Summary and Highlights

Advanced Clinical Pipeline

Three Differentiated Programs in Various Phases of Clinical Development

Program	Target	Preclinical	Phase 1	Phase 2	Phase 3	Key Milestones
Vidofludimus Calcium (IMU-838)	DHODH		rosis (RMS) — ENSURE T lerosis (PMS) — CALLIPEI langitis (PSC)	 RMS interim analysis planned after approx. half of the events occurred PMS interim analysis planned after half of the patients completed 24 weeks of treatment 		
IMU-935	IL-17 / RORγt	Psoriasis Castration-Resistant Pr	ostate Cancer (CRPC)			 Q4/2022: initial psoriasis data expected
IMU-856	Intestinal Barrier Function	Celiac Disease				 Q3/2022: SAD/MAD safety data expected

Completed or ongoing In preparation or planned



Thank You!



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