InnoCare Pharma (9969 HK)

Autoimmune therapies as a second growth engine

- **Autoimmune disease pipeline to drive business growth**. Orelabrutinib has seen robust sales growth in FY24 with a YoY increase of 49% to RMB1,001mn, surpassing its +45% YoY target. This robust performance is driven by the rising penetration and growing market share in CLL, MCL and MZL, where orelabrutinib remains the only approved BTKi for MZL. We expect orelabrutinib to continue to deliver robust sales in the oncology market. InnoCare's advancement in autoimmune diseases is equally noteworthy. Such progress includes orelabrutinib in multiple sclerosis (MS), ICP-322 (TYK-2 JH1) in atopic dermatitis (AD), and ICP-488 (TYK-2 JH2) in psoriasis. As of 3Q24, the Company maintained a strong cash balance of RMB7.8bn, providing ample resources to support its R&D efforts.
- **Positive outlook on orelabrutinib's development in multiple sclerosis**. Orelabrutinib has superior plasma exposure and robust CNS penetration compared to other BTK inhibitors. In a global Ph2 study, orelabrutinib showed encouraging efficacy in RRMS. We are optimistic about its potential for treating PPMS and SPMS, where significant unmet medical needs persist due to the lack of effective treatments. In Sep 2024, InnoCare received FDA approval to initiate a Ph3 trial for orelabrutinib in PPMS, with FPI anticipated in 2Q25. Furthermore, the FDA encouraged InnoCare to launch a Ph3 trial targeting the SPMS population, with FPI expected in 3Q25. In addition to advancing these trials, we expect the Company to actively pursue out-licensing opportunities of orelabrutinib. Moreover, given the promising Ph2a results for SLE, we anticipate the release of Ph2b trial results in 4Q25, with planning for a Ph3 trial already underway.
- **Two TYK2 inhibitors showing differentiated efficacy**. The Ph2 results of ICP-322 for the treatment of atopic dermatitis (AD) were remarkable, showing significant improvements in EASI 75, EASI 90, and Investigator's Global Assessment (IGA) scores of 0 or 1, combined with a favourable safety profile. These results position ICP-322 as a highly promising therapeutic option for AD, outperforming other existing treatments such as JAK1/2 inhibitors, IL-4Rα monoclonal antibodies, and IL-13 monoclonal antibodies. ICP-332 has been advanced into a Ph3 trial in China for AD in Nov 2024. We also anticipate out-licensing potential of ICP-332. A Ph1 study was also initiated in the US in 2024. For ICP-488, the Ph2 study in psoriasis demonstrated 50% placebo-adjusted PASI 90 and 62% sPGA 0/1, which are relatively competitive compared to other TYK2 targeted therapies.
- **Maintain BUY.** Supported by a solid cash position and steady cash inflows driven by strong orelabrutinib sales, we remain optimistic about InnoCare's clinical advancement in autoimmune diseases, including the development of key assets such as orelabrutinib, ICP-332, and ICP-488. We derive our DCF-based TP as HK$7.91 (WACC: 12.07%, terminal growth rate: 2.0%).

Earnings Summary

(YE 31 Dec)	FY22A	FY23A	FY24E	FY25E	FY26E
Revenue (RMB mn)	625	739	1,009	1,475	1,966
Net profit (RMB mn)	(886.6)	(631.3)	(442.8)	(360.6)	(235.9)
EPS (Reported) (RMB)	(0.60)	(0.37)	(0.25)	(0.20)	(0.13)
R&D expenses (RMB mn)	(639)	(751)	(886)	(997)	(1,082)
Admin expenses (RMB mn)	(182)	(194)	(202)	(273)	(305)
CAPEX (RMB mn)	(227)	(255)	(100)	(100)	(100)

Source: Company data, Bloomberg, CMBIGM estimates

BUY (Maintain)

Target Price	**HK$7.91**
(Previous TP	HK$9.07)
Up/Downside	**40.5%**
Current Price	**HK$5.63**

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Stock Data

Mkt Cap (HK$ mn)	9.918
Avg 3 mths t/o (HK$ mn)	29.7
52w High/Low (HK$)	7.49/4.06
Total Issued Shares (mn)	1762.6

Source: FactSet

Shareholding Structure

Hillhouse Capital	13.3%
Pang Kee Chan	10.5%

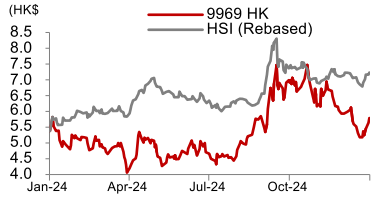
Source: Company data

Share Performance

	Absolute	Relative
1-mth	-2.4%	-4.2%
3-mth	-17.2%	-15.6%
6-mth	22.9%	6.5%

Source: FactSet

12-mth Price Performance



Source: FactSet

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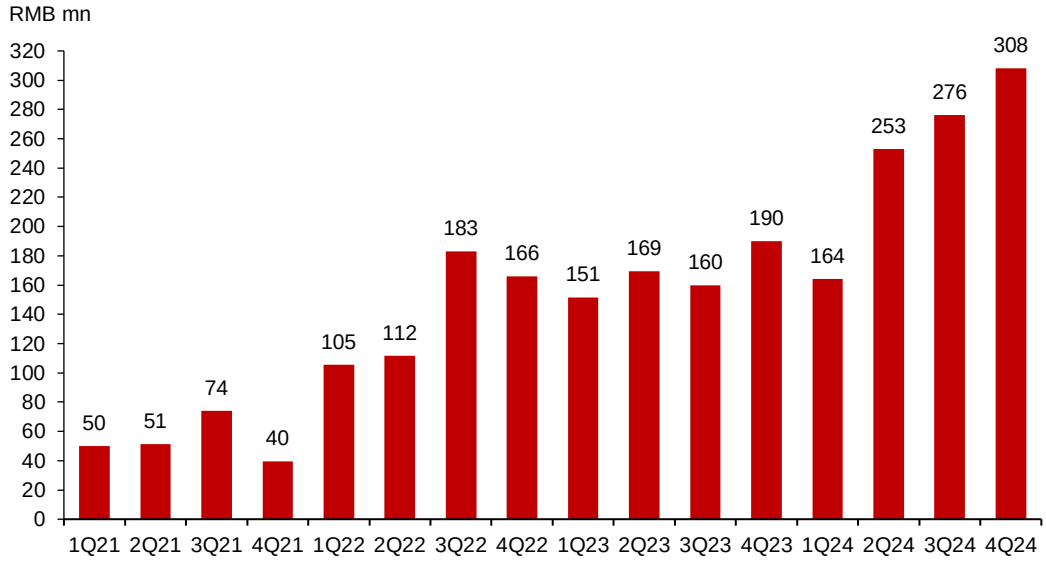
Strong sales of orelabrutinib as a cash cow to support further clincial development

As an oncology product, orelabrutinib has seen robust sales growth in FY24 with a YoY increase of 49% to RMB1,001mn, above the Company's +45% YoY target. This growth is driven by the rising NRDL penetration and market share in CLL, MCL and MZL, where orelabrutinib is the only approved BTKi for MZL. We expect orelabrutinib to serve as a cash cow to support the Company's further clinical development, particularly in assets for auto-immune diseases, including orelabrutinib in multiple sclerosis, ICP-332 (TYK-2 JH1) in atopic dermatitis, ICP-488 (TYK-2 JH2) in psoriasis. As of end-3Q24, the Company had sufficient cash balance of RMB7.8bn.

Figure 1: Quarterly sales of orelabrutinib



Source: Company data, CMBIGM

Expect large global potential for orelabrutinib in multiple sclerosis

Orelabrutinib has superior plasma exposure and robust CNS penetration compared to other BTK inhibitors, such as Sanofi's tolebrutinib and Merck KGaA's evobrutinib. In a global Ph2 study, orelabrutinib showed encouraging efficacy in the treatment of RRMS, demonstrating strong competitiveness compared to other drugs or drug candidates for RRMS. We are optimistic about the further development of orelabrutinib for PPMS and SPMS patients, which are areas with significant unmet medical needs due to lack of effective treatment options. In Sep 2024, InnoCare received approval from the US FDA for a Ph3 trial of orelabrutinib in PPMS, with first patient dosing (FPI) expected in 2Q25. Additionally, the FDA encouraged the Company to initiate a second Ph3 trial targeting the SPMS population, with the FPI to take place in 3Q25. We anticipate the Company to conduct the Ph3 trials while actively seek for BD opportunities.

Figure 2: Strong CNS penetration of orelabrutinib

BTKi	Company	Dose (mg)	CSF Conc. ~2h (ng/mL)
Orelabrutinib	InnoCare	150 QD	31.3
Evobrutinib	Merck KGaA	75 BID	3.21[2]
Tolebrutinib	Sanofi	120 QD	1.87[1]

Source: Company data, CMBIGM

Large unmet medical needs in MS, especially for PPMS and SPMS

Multiple sclerosis (MS) is a chronic disease that affects more than 2.9 million people worldwide, mostly younger adults in the overseas markets. Multiple sclerosis occurs when the immune system abnormally attacks the central nervous system (brain, spinal cord and optic nerves), causing inflammation and consequent damage. This damage can cause a wide range of symptoms, including weakness, fatigue and difficulty in seeing, and may eventually lead to disability.

Relapsing-remitting multiple sclerosis (RRMS) is the most common form of the disease and is characterized by episodes of new or worsening signs or symptoms (relapses) followed by periods of recovery. Approximately 85% of people with multiple sclerosis are initially diagnosed with RRMS. Around 50% of RRMS patients will eventually transition to secondary progressive multiple sclerosis (SPMS), in which they experience steadily worsening disability over time. nrSPMS refers to people with MS who have stopped experiencing confirmed relapses but continue to experience accumulation of disability. Relapsing forms of multiple sclerosis (RMS) include people with RRMS and people with SPMS who continue to experience relapses. Primary progressive multiple sclerosis (PPMS) is a debilitating form of the disease marked by steadily worsening symptoms but typically without distinct relapses or periods of remission. Approximately 15% of people with multiple sclerosis are diagnosed with PPMS.

We value the market potential of PPMS and SPMS due to the lack of effective treatment options. According to guidelines (link), various DMT (Disease-Modifying Therapy) drugs are available for patients with MS at the remission or maintenance phase, mainly for RMS (RRMS and SPMS with relapses), including Teriflunomide (特立氟胺, Aubagio), Fingolimod Hydrochloride (盐酸芬戈莫德, Gilenya), Siponimod (西尼莫德, Mayzent), Ozanimod (奥扎莫德, Zeposia), Dimethyl Fumarate (富马酸二甲酯, Tecfidera), Ofatumumab (奥法妥木单抗, Kesimpta), Glatiramer Acetate (醋酸格拉替雷, Copaxone), etc. The acute phase treatment for multiple sclerosis primarily includes corticosteroids and plasma exchange.

Current MS therapies reduce acute focal inflammation, but are less effective at slowing disability accumulation, as many DMTs primarily focus on modulating the immune system to reduce inflammatory responses, with lesser effects on neuroprotection and repair of damaged nerve tissue in the CNS. Additionally, progressive forms of MS like SPMS and PPMS have proven harder to treat than RMS. There are more than 20 DMTs approved for the treatment of RRMS, while only Roche/Biogen's ocrelizumab (Ocrevus, CD20 mAb) is approved in the US for PPMS. Globally, multiple new drugs are under late-stage clinical development for MS, including BTK inhibitor, CD40L mAb, CD20 mAb, etc.

The only approved CD20 mAb for PPMS has demonstrated significant sales potential. According to Fortune Business Insights (link), the global MS drugs market size was valued at US$21.33bn in 2023 and is projected to grow to US$38.94bn by 2032. Ocrevaus, a CD20 mAb from Roche and Biogen, was approved in the US and EU in 2017 and 2018, respectively, for treatment of RMS and PPMS. Ocrevaus generated a revenue of US$6.90bn in 2023. Notably, it remains the only FDA-approved treatment for PPMS and is priced at an annual retail cost of US$78,858 in the US (link). In Sep 2024, the FDA approved subcutaneous (SC) formulations of Ocrevus Zunovo (ocrelizumab). Roche has projected that Ocrevus Zunovo will contribute an incremental US$2.0bn in revenue to the overall Ocrevus franchise, complementing the existing intravenous (IV) formulation. We anticipate that new treatment options, such as orelabrutinib, will capture significant market value by addressing the substantial yet underserved MS patient population.

Orelabrutinib showed promising dose-dependent efficacy in Ph2 trial for RRMS

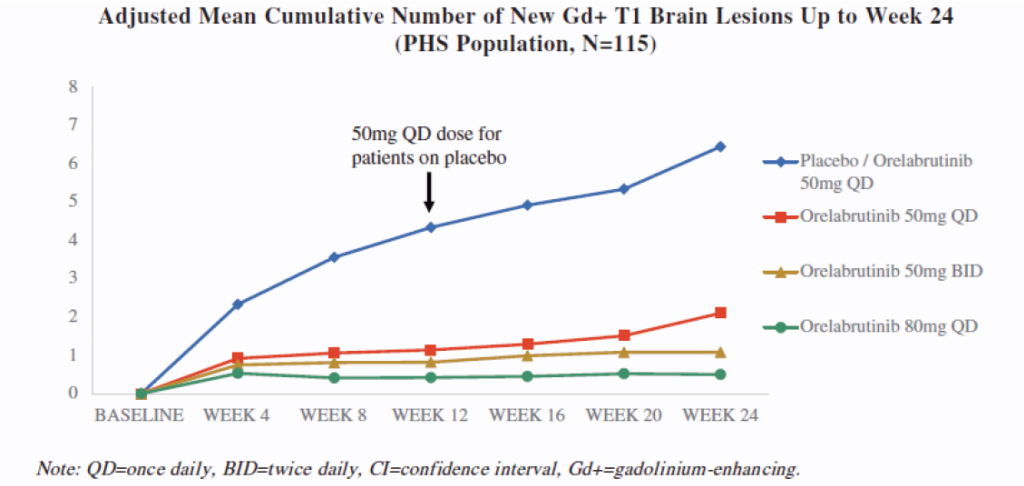
Orelabrutinib's Ph2 trial in RRMS has released promising results for RRMS, which support the drug's further investigation in MS. The Ph2 trial (NCT04711148) was designed to enroll 160 patients globally, who were randomised to three treatment arms (50 mg QD, 50 mg BID, and 80 mg QD) and a placebo control arm. Over a 24-week treatment period (n=115), the trial achieved its primary endpoint dose-dependently across all orelabrutinib treatment groups.

Notably, all orelabrutinib arms demonstrated control of T1 new lesions after just 4 weeks of treatment, with this effect sustained through 24 weeks. A dose-dependent improvement trend was observed. The 80 mg QD group achieved a 92.3% relative reduction in the cumulative number of new gadolinium-enhancing (Gd+) T1 lesions at week 24 compared to the placebo arm, which switched to orelabrutinib 50 mg

QD after week 12. This 92.3% reduction represents superior efficacy compared to other MS therapies, where relative reductions typically range from 61% to 89%. Orelabrutinib's strong performance, particularly at the 80 mg QD dose, underscores its potential as a highly effective therapy for RRMS (Refer to the figure below for further comparison).

Figure 3: Reduction of T1 lesions in orelabrutinib's Ph2 trial for RRMS

Cumulative number of New Gd+ T1 Lesion from Week 4 to Week 24	Placebo / Orelabrutinib 50mg QD (N=27)	Orelabrutinib 50mg QD (N=30)	Orelabrutinib 50mg BID (N=29)	Orelabrutinib 80mg QD (N=29)
Adjusted mean cumulative number (95% CI) of lesions from W4 to W24	6.45 (3.62, 11.52)	2.10 (0.62, 7.11)	1.08 (0.30, 3.81)	0.50 (0.09, 2.74)
Percent reduction		**67.4** (-22.0, 91.3)	**83.3** (33.2, 95.8)	**92.3** (56.5, 98.6)
P-value		**0.0958**	**0.0114**	**0.0037**



Source: Company data, CMBIGM

Figure 4: Comparison of Ph2 results of various therapies for RMS

Therapy	MoA	Company	Trial design	Primary endpoint	Relative reduction% in new Gd+ T1 lesions vs placebo
Orelabrutinib	BTKi	InnoCare	Placebo-controlled (N=136), 24Wk + extension (ext)	Cumulative Gd+ lesions at Wk12	92.3% (week 24)
Tolebrutinib	BTKi	Sanofi	Placebo-controlled for 4Wk, with 12Wk cross-over (N=130), 16Wk + ext	Dose-response for Gd+ lesions at Wk 12	85%
Fenebrutinib	BTKi	Roche	Placebo-controlled (N=106), 12Wk + ext	Cumulative Gd+ lesions at Wk 4, 8, and 12	90% (week 12)
Evobrutinib	BTKi	Merck KGaA	Placebo-controlled + open label DMF as a reference (N=267), 24Wk + ext	Cumulative Gd+ lesions at Wk 12, 16, 20, and 24	70%
Remibrutinib	BTKi	Novartis	Data in MS to be released in 2026	-	-
Ocrelizumab	CD20	Roche	Placebo-controlled + Interferon beta-1a as a reference (N=218), 24Wk + ext	Cumulative Gd+ lesions at Wk 12, 16, 20, and 24	89%
Ofatumumab/Kesimpta	CD20	Novartis	Placebo-controlled (N=231), 24Wk + ext	Cumulative Gd+ lesions at Wk 12	65%; 91%
Frexalimab	CD40L	Sanofi	Placebo-controlled (N=166), 12Wk + ext	number of new Gd+ lesions at Wk 12 relative to Wk8	89%
Siponimod	S1PR	Novartis	Placebo-controlled, adaptive, dose ranging (N = 297), 6m + ext	Dose-response for CUAL at 3 mo	72%

Dimethyl Fumarate (DMF)	Nrf2	Biogen	Placebo-controlled(N=257),24Wk + ext	Cumulative Gd+ lesions at Wk12, 16, 20, and 24	69%
Fingolimod	S1PR	Novartis	Placebo-controlled (N=281), 6m + ext	Cumulative Gd+ lesions monthly for 6 months	61%; 88% at month 6
Teriflunomide	DHODH	Sanofi	Placebo-controlled (N=179), 36Wk + ext	# of CUAL per MRI scan	61%

Source: PharmCube, PubMed, CMBIGM

In orelabrutinib's Ph2 study for RRMS, the 80 mg QD cohort reported the lowest incidence of liver-related TEAEs among all treatment groups. Two cases of ALT/AST >8x ULN were observed, occurring in the 50 mg BID group and the 50 mg QD group, respectively. Notably, the safety profile of the 80 mg QD dose was comparable to that of the placebo group, further supporting its potential as a well-tolerated treatment option. We maintain our positive attitude towards orelabrutinib's safety profile in liver-related TEAEs for MS patients, and look forward to the development of orelabrutinib for PPMS and SPMS in Ph3 studies.

Next-generation therapies for treatment of MS in late stage of development

BTK inhibitors' ability to penetrate the blood-brain barrier allows them to target immune cells on both sides of the barrier, offering the potential to effectively curb disease progression in multiple sclerosis. Currently, several BTK inhibitors, such as orelabrutinib, tolebrutinib, fenebrutinib, among others, are in late-stage development for different types of MS.

Orelabrutinib is advancing into Ph3 trials for PPMS and SPMS. Regarding PPMS, Sanofi's tolebrutinib is conducting a Ph3 trial with data expected in 2H25, while the Ph3 trial of Roche's fenebrutinib has been fully enrolled with data expected by end 2025. With Ph3 FPI to take place in 2Q25, orelabrutinib ranks third globally in the development for PPMS, trailing tolebrutinib and fenebrutinib. For SPMS, Sanofi's tolebrutinib has released positive Ph3 data for nrSPMS patients, with NDA filing in the US expected in the near term, which is a significant milestone for the MS therapeutic market. Orelabrutinib is set to initiate a Ph3 trial in SPMS, following tolebrutinib's success. Additionally, Sanofi's frexalimab (CD40L) has a Ph3 trial in nrSPMS ongoing.

Given orelabrutinib's strong efficacy and safety profile revealed in the Ph2 trial and considering it is one of the most advanced BTK inhibitors for the underserved PPMS and SPMS markets, we anticipate orelabrutinib to become a major player in the global MS market.

Figure 5: Therapeutic candidates for MS

Therapy	MoA	Company	Development stage
Orelabrutinib	BTKi	InnoCare	PPMS global Ph3 initiated (as per Mgmt); **SPMS** global Ph3 to start in 1H25.
Tolebrutinib	BTKi	Sanofi	**nrSPMS** (HERCULES) Ph3 met primary endpoint, to file NDA; PPMS Ph3 (PERSEUS) to release data in 2H25; RMS Ph3 trials (GEMINI 1/2) missed the primary endpoints; Long-term safety Ph3 study (NCT06372145) ongoing.
Fenebrutinib	BTKi	Roche	RMS Ph3 trials (FENhance 1/2) and PPMS Ph3 trial (FENtrepid) fully enrolled; data readout for RMS and PPMS at end-2025.
Remibrutinib	BTKi	Novartis	RMS Ph3 trials (REMODEL-1/2) ongoing, data readout in 2026.
Evobrutinib	BTKi	Merck KGaA	Development terminated in 4Q23; RMS Ph3 trials (volutionRMS 1/2, vs teriflunomide) missed primary endpoint ARR (link).

Frexalimab	CD40L	Sanofi	RMS and **nrSPMS** Ph3 trials ongoing; data readouts anticipated from 2027.

Source: Company slides, press release, CMBIGM

Tolebrutinib's success in nrSPMS marks a major milestone

Tolebrutinib, an oral and brain-penetrant BTK inhibitor developed by Sanofi, is being evaluated in Ph3 trials for the treatment of various forms of MS. In the Ph3 HERCULES study, tolebrutinib achieved its primary endpoint for non-relapsing SPMS (nrSPMS) by significantly delaying disability progression and even improving disability in some patients. Tolebrutinib is the first and only therapy to demonstrate a delay in the time to onset of confirmed disability progression (CDP) in patients with nrSPMS, which represents a strong encouragement for the development of BTK inhibitors in the treatment of MS.

In the HERCULES Ph3 study for nrSPMS (link1, link2), tolebrutinib demonstrated a 31% delay in time to onset of 6-month CDP in patients with nrSPMS, compared to placebo (HR 0.69; p=0.0026). Further analysis of secondary endpoints demonstrated that the number of participants who experienced confirmed disability improvement was nearly double with tolebrutinib (10%) compared to those on placebo (5%) (HR 1.88; nominal p=0.021). In the study, a slight increase in adverse events was observed among patients treated with tolebrutinib. Elevations in liver enzymes (>3x ULN) occurred in 4.1% of participants receiving tolebrutinib, compared to 1.6% in the placebo group. A small proportion (0.5%) of participants in the tolebrutinib treatment arm experienced peak ALT increases >20x ULN, all of which were observed within the first 90 days of treatment. Importantly, nearly all cases of liver enzyme elevations resolved without the need for further medical intervention, except for one. The implementation of more frequent monitoring has mitigated serious liver sequelae.

On the other hand, the GEMINI 1 and 2 Ph3 studies of tolebrutinib vs SoC Aubagio (teriflunomide) in relapsing MS (RMS) did not meet their primary endpoints of improvement in annualized relapse rates (ARR, link). However, in the key secondary endpoint, a pooled analysis of data from GEMINI 1 and 2 revealed that tolebrutinib delayed the time to onset of 6-month confirmed disability worsening (CDW) by 29% (HR 0.71; nominal p=0.023), which are in line with the 31% delay in CDP observed in participants with nrSPMS.

Moreover, the Ph3 trial PERSEUS of tolebrutinib in PPMS is ongoing, with the data to be available in 2H25.

Figure 6: Ph3 results of major drugs and drug candidates for different types of MS

	Tolebrutinib	**Tolebrutinib**	**Ocrevus**
Company	Sanofi	Sanofi	Roche
MoA	BTKi	BTKi	CD20
Trial ID	HERCULES, Ph3	GEMINI1/2, pooled, Ph3	ORATORIO, Ph3
Patients	**nrSPMS** (SPMS absent of relapses in two years)	**RRMS**	**PPMS**
Patients number	754 vs 377	933 vs 940	488 vs 244
Regemen	Tolebrutinib vs placebo	Tolebrutinib vs Teriflunomide	ocrelizumab vs placebo
Primary endpoint	Time to 6-Month CDP	Annualised Relapse Rate	Time to 3-month CDP
Time to 3-month CDP	32.6% vs 41.5%; HR=0.76, 24% risk reduction, p=0.013	14.7% vs 18.5%; HR=0.73, 27% risk reduction, p=0.018	32.9% vs 39.3%; HR=0.76, 24% risk reduction, p=0.03
Time to 6-Month CDP	26.9% vs 37.2%; HR=0.69, 31% risk reduction, p=0.0026	9.9% vs 13.2%; HR=0.71, 29% risk reduction, p=0.023	29.6% vs 35.7%; HR=0.75, 25% risk reduction, p=0.04

Annualised Relapse Rate		Rate ratio=1.03, p=0.80, **missed primary endpoint**	
Annualised Rate of New/Enlarging T2 Lesions	1.8 vs 2.9, rate ratio=0.62, p=0.011	5.6 vs 5.2, rate ratio=1.08 (GEMINI1); 5.1 vs 4.4, rate ratio=1.17 (GEMINI2);	
Rate of New Gd-Enhancing T1 Lesions		0.53 vs 0.29, rate ratio=1.86 (GEMINI1); 0.46 vs 0.22, rate ratio=2.12 (GEMINI2);	
Any serious TEAE	15.0% vs 10.4%	9.8% vs 8.2%	20.4% vs 22.2%
Any TEAE leading to discontinuation	3.9% vs 2.9%	4.5% vs 4.4%	4.1% vs 3.3%
ALT >3×ULN	4.1% vs 1.6%	5.6% vs 6.3%	
ALT >20×ULN	0.5% vs 0	0.5% vs 0.1%	
Source	Link	Link	Link

Source: Pubmed, CMBIGM

Figure 7: Sanofi's Ph3 trials in multiple sclerosis

Disease category	Drug candidate	Stage	Trial ID	Trial description
Neuro-inflammation	tolebrutinub (BTK inhibitor)	Phase 3	NCT04458051 PERSEUS	Study of BTK inhibitor tolebrutinib in PPMS
Neuro-inflammation	tolebrutinub (BTK inhibitor)	Phase 3	NCT04411641 HERCULES	Study of BTK inhibitor tolebrutinib in nrSPMS
Neuro-inflammation	tolebrutinub (BTK inhibitor)	Phase 3	NCT04410991 GEMINI 2	Study of BTK inhibitor tolebrutinib in RMS
Neuro-inflammation	tolebrutinub (BTK inhibitor)	Phase 3	NCT04410978 GEMINI 1	Study of BTK inhibitor tolebrutinib in RMS
Neuro-inflammation	tolebrutinub (BTK inhibitor)	Phase 3	NCT06372145	A Study to investigate long-term safety and tolerability of tolebrutinib in participants with multiple sclerosis
Neuro-inflammation	frexalimab (CD40L mAb)	Phase 3	NCT06141473	Studies of frexalimab in adults with relapsing forms of multiple sclerosis (RRMS)
Neuro-inflammation	frexalimab (CD40L mAb)	Phase 3	NCT06141486	Study of frexalimab in adults with nonrelapsing secondary progressive multiple sclerosis (nrSPMS)

Source: Sanofi, CMBIGM.

As also mentioned in the above figure, Sanofi's frexalimab (CD40L mAb) is in late stage development for MS. Based on its promising Ph2 data, we anticipate frexalimab to be a future competitor to orelabrutinib, particularly in the treatment of SPMS. In a Ph2 study of frexalimab for RMS (link), the relative reduction in Gd+ T1 lesions reached 89% in the frexalimab 1200mg IV group after 12 weeks of treatment. Ph3 studies of frexalimab in RMS and nrSPMS are ongoing, with the data readouts anticipated from 2027.

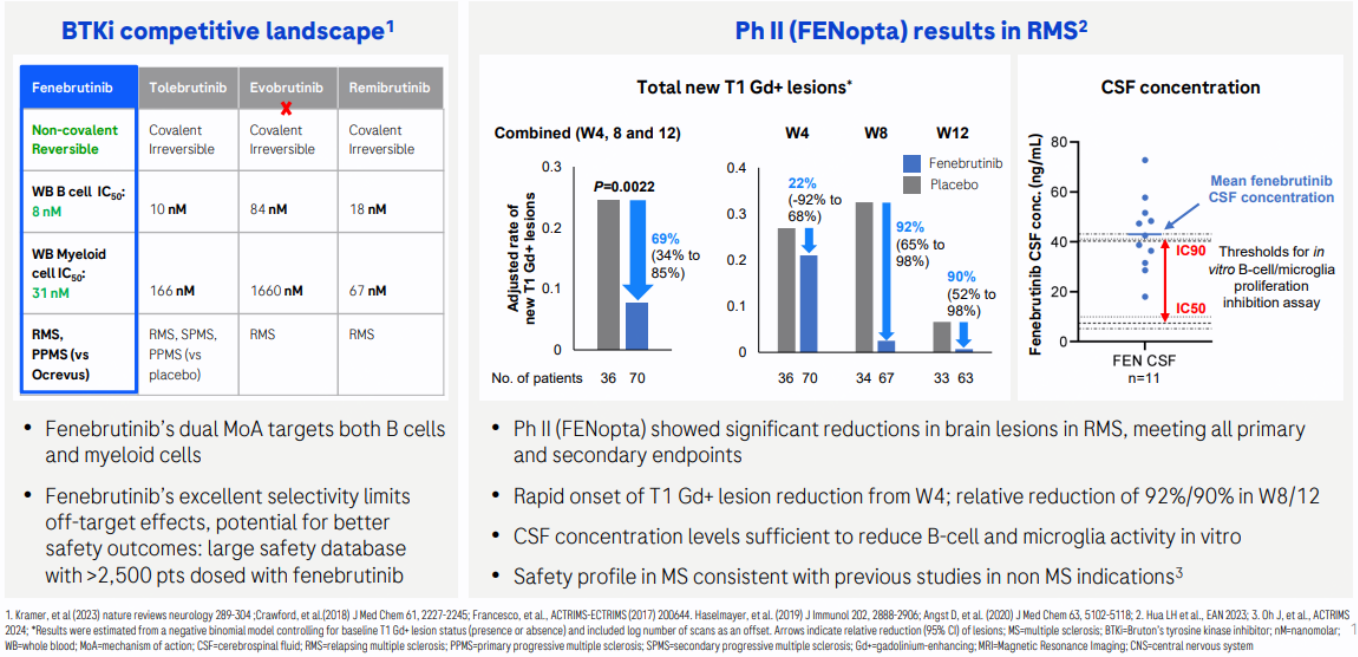
Roche's fenebrutinib has demonstrated promising Ph2 data in RMS, with Ph3 trials in PPMS and RMS ongoing

Roche's fenebrutinib, a BTK inhibitor, also has three Ph3 trials underway in MS: the FENhance 1 and 2 trials in RMS and the FENtrepid trial in PPMS. The two identical RMS trials use teriflunomide as the active comparator, while the PPMS trial is the only study evaluating a BTK inhibitor head-to-head against Ocrevus (ocrelizumab), the current standard of care for PPMS. Data readouts from these pivotal studies are anticipated by the end of 2025.

Strong Ph2 results of fenebrutinib support its further investigation in Ph3 studies. In the Ph2 FENopta study, fenebrutinib demonstrated near-complete suppression of disease activity and disability progression in relapsing MS (RMS) patients, showcasing its strong efficacy potential. 106 RMS patients were randomized 2:1 to receive fenebrutinib (n=70) or placebo (n=36) for 12 weeks in the study. At

Weeks 4, 8 and 12 (combined), fenebrutinib patients had a 69% reduction in total new Gd+ lesions vs placebo patients. Relative reductions in Gd+ lesions were observed at Week 8 (92%) and Week 12 (90%).

Figure 8: IC50 and Ph2 results of fenebrutinib in RMS



Source: Roche, CMBIGM

Two highly diffrentiated TYK2 inhibitors targeting autoimmune diseases

The Janus tyrosine kinases (JAKs) family encompasses four mammalian members: JAK1, JAK2, JAK3, and TYK2. InnoCare has two TYK2 inhibitors targeting the treatment of auto-immune diseases, including ICP-332 (TYK-2 JH1) and ICP-488 (TYK-2 JH2). ICP-332 is designed to be a potent and selective TYK2 JH1 inhibitor with 400 folds of selectivity against JAK2 to avoid the adverse events associated with nonselective JAK inhibitors. ICP-488 is a potent and selective TYK2 allosteric inhibitor of the pseudo kinase domain JH2 of TYK2, without inhibition to any other JAK family members.

Figure 9: Selectivity of ICP-332 and ICP-488 across the JAK family

抑制剂	IC_{50} (nM)	IC_{50} (nM) @1 mM ATP			
	TYK2 JH2	TYK2 JH1	JAK1	JAK2	JAK3
ICP-332	2319	0.5	19	191	930
ICP-488	5	>10,000			

Source: Company data, CMBIGM

In late 2023, the Ph2 results for ICP-322 in the treatment of atopic dermatitis (AD) was revealed, showing impressive outcomes, with remarkable improvements in EASI 75, EASI 90, and Investigator's Global Assessment (IGA) scores of 0 or 1, accompanied by a favourable safety profile. These findings positioned ICP-322 as a highly promising therapeutic option for AD, outperforming other existing treatments such as JAK1/2 inhibitors, IL-4Rα monoclonal antibodies, and IL-13 monoclonal antibodies (refer data comparison in Figure 12). The positive Ph2 outcomes have paved the way for continued exploration. ICP-332, has advanced into Ph3 trial in China (NCT06775860) for AD and has also initiated Ph1 clinical investigations in the US since Jun 2024. We anticipate ICP-332 to become an important player in the therapeutic landscape for AD.

For ICP-488, the topline results of Ph2 study in psoriasis was released in Oct 2024, with 50% placebo-adjusted PASI 90 and the 62% placebo-adjusted sPGA 0/1, which were quite competitive compared to other TYK2 targeted innovative drugs. Despite the fact that monoclonal antibodies (mAbs) targeting IL-23p19 and IL-17 (i.e. guselkumab and secukinumab) represent promising injectable therapies for psoriasis, ICP-488 presents a potential robust convenient oral alternative for the treatment of psoriasis, in our view. Innocare is preparing to initiate a Ph3 trial of ICP-488 in psoriasis.

Global development of TYK2 inhibitors: BMS's Sotyktu leads the way

BMS's Sotyktu (deucravacitinib, 氘可来昔替尼), a highly selective and first-in-class TYK2 inhibitor, has achieved regulatory approval from both the US FDA and

China's NMPA for the treatment of adult patients with moderate-to-severe plaque psoriasis. This approval was based on the results of the POETYK PSO-1 and POETYK PSO-2 Ph3 studies, which demonstrated the superior efficacy of once-daily deucravacitinib in improving skin clearance compared to placebo and twice-daily apremilast (PDE4 inhibitor). In its early market rollout, deucravacitinib delivered global sales of US$170mn in 2023 and US$163mn (+52% YoY) in 9M24.

Beyond Sotyktu, the TYK2 inhibitor field is rapidly expanding, with several other candidates progressing through Ph3 trials, including zasocitinib, ESK-001, ICP-332, and HS-10374, for the treatment of various autoimmune diseases, i.e. psoriasis, Sjögren's syndrome (SS), systemic lupus erythematosus (SLE), atopic dermatitis (AD). The competitive pipeline underscores the increasing interest in TYK2 inhibitors as a transformative therapy class for autoimmune diseases.

In the field of atopic dermatitis, several antibody injectable have been approved, i.e. dupilumab (IL-4Rα), CM310 (IL-4Rα), and tralokinumab (IL-13), alongside several oral JAK1 inhibitors, such as upadacitinib and abrocitinib. Notably, ICP-332 stands as the only TYK2 inhibitor drug candidate currently in Ph3 study for atopic dermatitis. Given the release of its competitive Ph2 efficacy data and favourable safety results, we anticipate the potential of ICP-332 to be a first-in-class TYK2 inhibitor for atopic dermatitis. The psoriasis market is relatively more competitive – as of now, several TYK2 inhibitors have either been approved or are in Ph3 trials for psoriasis, including deucravacitinib, zasocitinib, ESK-001, ICP-332 and HS-10374.

Figure 10: Global development landscape of TYK2 inhibitors

Drug Name	MoA	Company	US development phase	China development phase	Note
deucravacitinib	TYK2 allosteric inhibitor	Bristol-Myers Squibb	Approved (2023.09, psoriasis)	Approved (2023.10, psoriasis)	Sjögren's Syndrome (SS), SLE, PsA in Ph3
zasocitinib	TYK2 allosteric inhibitor	Nimbus(Takeda); Schrödinge	Ph3	Ph3	Psoriasis, PsA in Ph3
ESK-001	TYK2 allosteric inhibitor	Alumis	Ph3	-	Psoriasis in Ph3
ICP-332	**TYK2 inhibitor**	**InnoCare Pharma**	**Ph1**	**Ph3**	**AD in Ph3 in China**
HS-10374	TYK2 allosteric inhibitor	Hansoh Pharmaceutical	-	Ph3	Psoriasis in Ph3 in China
ropsacitinib	TYK2 inhibitor	Pfizer; Priovant	Ph2	IND	
VTX-958	TYK2 allosteric inhibitor	Ventyx Biosciences	Ph2	-	
GLPG3667	TYK2 inhibitor	Gilead; Galapagos	Ph2	-	
lomedeucitinib	TYK2 inhibitor	Bristol-Myers Squibb	Ph2	-	
ICP-488	**TYK2 allosteric inhibitor**	**InnoCare Pharma**	**-**	**Ph2**	**Psoriasis in Ph2**
D-2570	TYK2 allosteric inhibitor	InventisBio	-	Ph2	
TQH3906	TYK2 allosteric inhibitor	Chia Tai Tianqing Pharma	-	Ph2	
WD-890	TYK2 inhibitor	Wenda Pharma	-	Ph2	
BGB-23339	TYK2 allosteric inhibitor	BeOne Medicines	Ph1	Ph1	
UA021	TYK2 inhibitor	Usynova	-	Ph1	
ARTS-011	TYK2 allosteric inhibitor	Allorion Therapeutics	-	Ph1	

BMS-986465	TYK2 inhibitor	Bristol-Myers Squibb	Ph1	-
CS32582	TYK2 allosteric inhibitor	Chipscreen Biosciences	-	Ph1
FZ007	TYK2 inhibitor	Fermion	-	Ph1
ZG-002	TYK2 allosteric inhibitor	Warrant Pharmaceutical	-	Ph1
CMS-D001	TYK2 inhibitor	China Medical System	-	Ph1

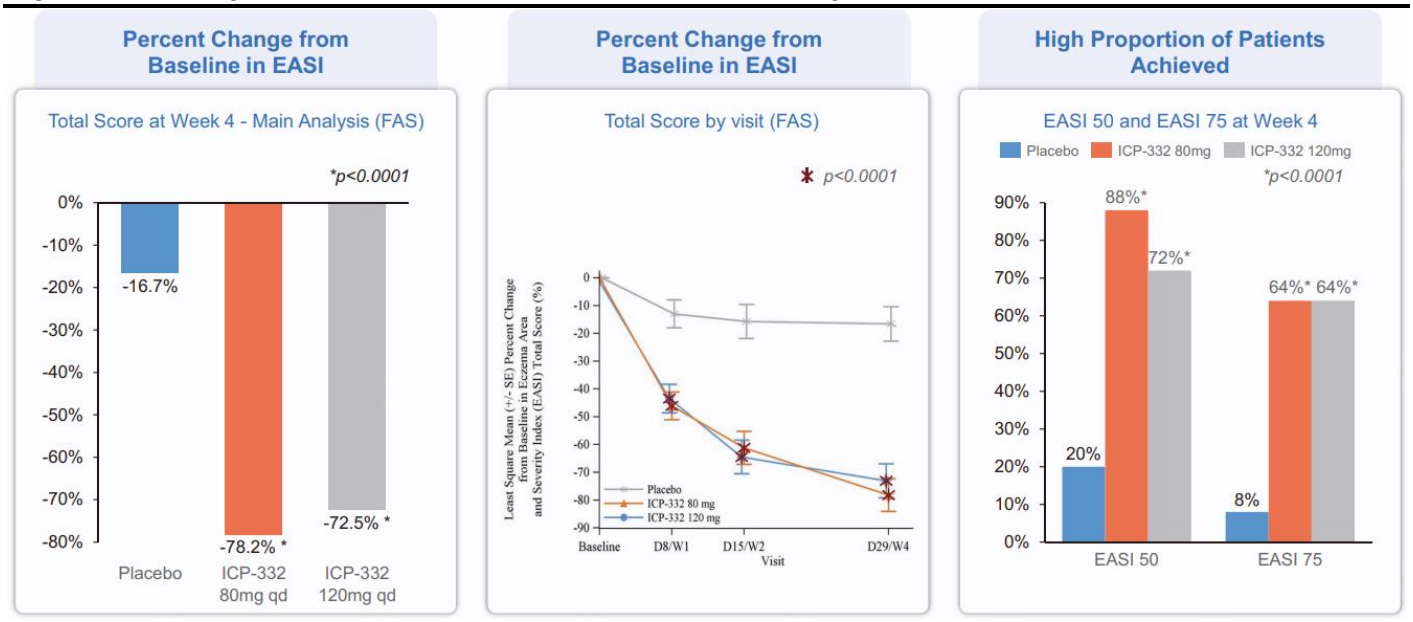
Source: PharmCube, CMBIGM. Note: As of Jan 2025

ICP-332 demonstrated strong efficacy in AD with a favourable safety profile

Atopic dermatitis (AD) is one of the most prevalent forms of eczema, characterized by itching, redness, and inflammation. The global market potential for AD is projected to reach US$10bn by 2030. In China alone, there were 65.7mn AD patients in 2019, a number expected to grow to 81.7mn by 2030. For moderate-to-severe cases, AD significantly impacts patients' quality of life, with recurring itching often leading to sleep disturbances. Consequently, reducing itching remains an urgent unmet need for most patients with moderate-to-severe AD.

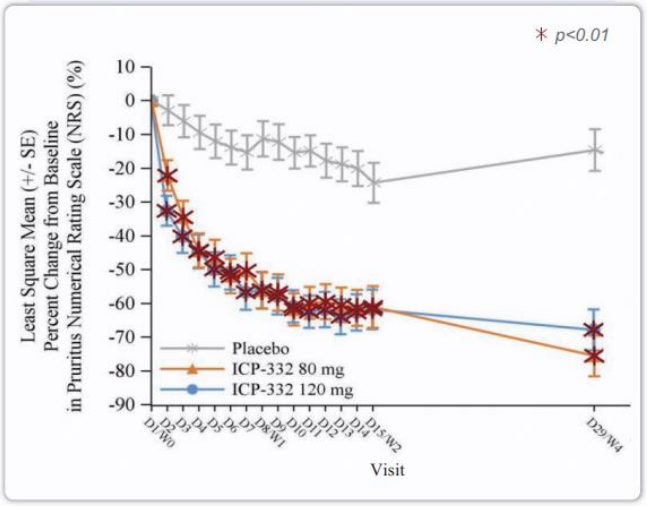
In Dec 2023, positive Ph2 proof-of-concept data for ICP-332 in AD were announced (link1, link2). The trial was a 4-week randomized study evaluating the efficacy and safety of ICP-332 in patients with moderate-to-severe AD. A total of 75 adult subjects were enrolled, with 25 patients in each of the 80mg QD, 120mg QD, and placebo groups. Subjects underwent four weeks of treatment, followed by a 28-day safety observation period. ICP-332 achieved multiple efficacy endpoints, including percentage reductions from baseline in EASI score, as well as EASI 50, EASI 75, EASI 90, and IGA 0/1 response rates in the 80mg and/or 120mg treatment groups.

Figure 11: Efficacy of ICP-332 for the treatment of AD in Ph2 study



Quick and Statistically Significant Response from Day 2

Pruritus Numerical Rating Scale (NRS)



Improvement of Patient Quality of Life

Dermatology Life Quality Index (DLQI) Score Change from Baseline by Visits (Full Analysis Set)

	Placebo (N=25)	ICP-332 80mg (N=25)	ICP-332 120mg (N=25)
D8/W1	-3.3(-4.8,-1.9)	-6.5(-8.0,-5.1)	-6.8(-8.4,-5.3)
	p-value	**0.0027**	**0.0018**
D15/W2	-2.2(-4.2,-0.2)	-8.7(-10.7,-6.7)	-7.9(-9.9,-5.9)
	p-value	**<0.0001**	**0.0002**
D29/W4	-1.2(-3.3,0.9)	-10.8(-12.8,-8.8)	-8.9(-11.0,-6.8)
	p-value	**<0.0001**	**<0.0001**

Source: Company data, CMBIGM

The mean percentage change from baseline in the EASI score reached 78.2% and 72.5% for the 80mg and 120mg, respectively, both showing highly statistically significant improvements compared to 16.7% in the placebo arm. The proportion of patients achieving EASI 75 was 64% in both the 80mg and 120mg groups, compared to 8% in the placebo group ($p < 0.0001$). These results exceed the reported efficacies of several approved drugs following 12- or 16-week treatment regimens (not a head-to-head comparison, refer to the figure below).

In the 80mg QD treatment group, the difference from placebo reached 56% in EASI 75, 40% in EASI 90, 32% in (IGA) 0/1 and 56% in NRS ≥4 Improvement ($p<0.01$). Additionally, significant improvements in pruritus (itch) were observed. Patients treated with ICP-332 experienced a rapid reduction in pruritus severity and frequency as early as day 2, as measured by the pruritus numerical rating scale (NRS) ($p < 0.01$) across both the 80mg and 120mg dosing groups. This improvement in pruritus was accompanied by a notable enhancement in quality of life. Starting from day 7, the DLQI (Dermatology Life Quality Index) scores in the treatment groups showed statistically significant improvements compared to the placebo group, with sustained benefits observed through the end of the treatment period.

ICP-332 was safe and well tolerated in AD patients. In this study, all TRAEs were mild or moderate. The overall incidence rates of TRAEs and TRAEs related to infections and infestations in the two treatment groups were comparable to the placebo group.

Figure 12: Efficacy comparison of various therapies for AD

Drug name	ICP-332	Upadacitinib		Abrocitinib		Baricitinib		Dupilumab		Tralokinumab		Lebrikizumab		CM310
MoA	TYK2/ JAK1	JAK1		JAK1		JAK1/2		IL-4Rα		IL-13		IL-13		IL-4Rα
Company	InnoCare	AbbVie		Pfizer		Eli Lilly, Incyte		Regeneron, Sanofi		Leo Pharma		Roche, Eli Lilly, Almirall		Keymed
Trial	NCT05702268, 80mg	Measure Up 1) 15mg	Measure Up 2) 15mg	JADE MONO-1, 100mg	JADE MONO-2, 100mg	BREEZE AD1, 2mg	BREEZE AD2, 2mg	SOLO-1	SOLO-2	ECZTRA 1	ECZTRA 2	ADvocate1	ADvocate2	NCT05265923
Treatment duration (weeks)	4w	16w	16w	12w	12w	16w	16w	16w	16w	16w	16w	16w	16w	16w
Placebo-adjusted results														
EASI 75	56%	53%	47%	28%	34%	10%	12%	36%	32%	12%	22%	42%	33%	41%
EASI 90	40%	45%	37%	13%	20%	-	-	28%	32%	10%	13%	29%	21%	26%
IGA 0/1 with >=2 points from baseline	32%	40%	34%	16%	19%	7%	6%	28%	27%	9%	12%	30%	22%	28%
Source	Link	Link	Link	Link	Link	Link	Link	Link	Link	Link	Link	Link	Link	Link

Source: Pubmed, FDA label, CMBIGM

In a cross-trial comparison, the 56% EASI 75, 40% EASI 90, and 32% IGA 0/1 improvements observed with ICP-332 versus placebo appear highly promising compared to other therapies, including JAK1/2 inhibitors, IL-4Rα mAbs, and IL-13 mAbs. Notably, upadacitinib (a JAK1 inhibitor from AbbVie) demonstrated strong efficacy in its Measure Up 1 study at week 16, achieving 53% EASI 75, 45% EASI 90, and 40% IGA 0/1, which are comparable to the results observed with ICP-332 at week 4, in our view. However, from a safety perspective, upadacitinib carries an FDA boxed warning for risks including serious infections, mortality, malignancy, major adverse cardiovascular events (MACE), and thrombosis. In light of efficacy and safety profiles, we believe ICP-332 could be a competitive treatment option in the AD market once confirmed in Ph3 studies. A Ph3 trial for atopic dermatitis has been ongoing since Nov 2024, and a Ph1 trial in the US is currently underway.

ICP-488, a promising oral therapy for psoriasis

Psoriasis is a chronic immune-mediated disease characterized by raised, scaly patches on the skin, driven by systemic inflammation. Recent data indicates that approximately 7 million people in China are currently living with psoriasis, and this number continues to rise. For moderate-to-severe plaque psoriasis, biologic therapies and small molecule targeted drugs are complementary, guideline-recommended treatment options. Traditional biologic therapies, such as TNF-α mAbs, have long been a cornerstone of psoriasis management. However, the emergence of next-generation systemic therapies, including newer biologics targeting IL-12/IL-23, IL-17, IL-23p19, and IL-36, as well as small molecules targeting PDE4, JAK1/2/3, and TYK2, is expanding the landscape of effective treatment options.

Figure 13: Next generation therapies for psoriasis and other autoimmune diseases

Target	Drug name	Chinese name	China approved indications	Year of initial China approval	US approved indications	Year of initial US approval	Initial NRDL coverage date
Biologics							
IL-12/IL-23	ustekinumab	乌司奴单抗	PP, CD	2017	PP, PsA, CD, UC	2009	2022.01
IL-17	secukinumab	司库奇尤单抗	PP, AS	2019	PP, PsA, AS, etc	2015	2021.03
	ixekizumab	依奇珠单抗	PP, AS	2019	PP, PsA, AS, etc	2016	2022.01
	brodalumab	布罗利尤单抗	PP	2020	PP	2017	-
	bimekizumab	比吉利珠单抗	AS	2024	PP, AS, PsA	2023	-
IL-23p19	guselkumab	古塞奇尤单抗	PP	2019	PP, PsA	2017	2023.03
	tildrakizumab	替拉珠单抗	PP	2023	PP	2018	2024.01
	risankizumab	利生奇珠单抗	-	2023.07 BLA	PP, PsA, DC, UC	2019	-
	mirikizumab	Omvoh	-	-	UC	2023	-
IL-36	spesolimab	佩索利单抗	GPP	2022	GPP	2022	2024.01
Small molecules							
PDE4	apremilast	阿普米司特	PP	2021	PP, PsA, etc	2014	2023.03
JAK1-3	tofacitinib	托法替布	PsA, RA, AS	2017	PsA, AS, UC, RA, etc	2012	2023.03
	upadacitinib	乌帕替尼	PsA, RA, AD	2022	PsA, AS, UC, RA, etc	2019	2023.03
TYK2	deucravacitinib	氘可来昔替尼	PP	2023	PP	2022	2025.01

Source: PharmCube, CMBIGM. Notes: PP - plaque psoriasis, CD - Crohn disease, PsA - psoriatic arthritis, UC - ulcerative colitis, AS - ankylosing spondylitis, RA - rheumatoid arthritis, GPP - generalized pustular psoriasis, HS - hidradenitis suppurativa.

Among biologics, IL-23p19 antibodies represent a highly promising therapeutic option for psoriasis, offering superior skin clearance with PASI 90 rates exceeding 80%. For example, guselkumab (IL-23p19) demonstrated superiority over secukinumab (IL-17) in a head-to-head study for psoriasis, achieving a PASI 90 at week 48 of 84% vs 70% ($p < 0.0001$, link). Secukinumab (IL-17) outperformed ustekinumab (IL-12/IL-23) in another head-to-head study, achieving a PASI 90 at week 52 of 73% vs. 60% ($p < 0.0001$, link). In China, secukinumab (IL-17 mAb) currently holds a significant share of the psoriasis market, partly due to its early inclusion in the NRDL in 2021.

While next-generation monoclonal antibodies have demonstrated excellent efficacy in treating psoriasis, TYK2 inhibitors are emerging as a compelling alternative, offering the convenience of an oral therapy. Bristol-Myers Squibb's (BMS) TYK2 inhibitor deucravacitinib has already been approved in the US for psoriasis, and several other TYK2 inhibitors are in Ph3 trials, including zasocitinib, ESK-001, ICP-488 and HS-10374.

In Oct 2024, InnoCare announced that the Ph2 study of ICP-488 for moderate-to-severe plaque psoriasis met its primary endpoint (link). The study enrolled 129 patients, randomized in a 1:1:1 ratio to three treatment groups receiving 6mg once-daily, 9mg once-daily, or placebo for 12 weeks. ICP-488 achieved multiple efficacy endpoints, including PASI 75, PASI 90, PASI 100, and sPGA 0/1, in both the 6mg and 9mg dosing groups. The results were as follows:

- PASI 75: 77.3% (6mg) and **78.6%** (9mg) vs. 11.6% (placebo) ($p < 0.0001$).
- PASI 90: 36.4% (6mg) and **50.0%** (9mg) vs. 0% (placebo) ($p < 0.0001$).

- PASI 100: 11.4% (6mg) and **11.9%** (9mg) vs. 0% (placebo) ($p < 0.05$).
- sPGA 0/1: 70.5% (6mg) and **71.4%** (9mg) vs. 9.3% (placebo) ($p < 0.0001$).

Figure 14: Cross-comparison of various psoriasis therapies

Drugs	ICP-488	BMS-986165/ deucravacitinib	ESK-001	TAK-279 /zasocitinib	HS-10374	ropsacitinib	D-2570	ustekinumab	guselkumab vs secukinumab
MoA	TYK2	TYK2	TYK2	TYK2	TYK2	TYK2	TYK2	IL-12/IL-23	IL-23p19 vs IL-17
Company	InnoCare	BMS	Alumis	Nimbus (Takeda)	Hansoh	Pfizer; Priovant	Inventisbio	J&J	J&J/Novartis vs Novartis
Trial	Ph2	Ph3, Asia trial, POETYK PSO-3	Ph2	Ph2	Ph2	Ph2	Ph2	Ph3	Ph3, head-to-head
Patient No.	129	146 vs 74	39 vs 38	259	125	45 vs 43 vs 45	161	255 vs 256 vs 255 (PHOENIX1) 409 vs 411 vs 410 (PHOENIX2)	534 vs 514
Dose	6mg or 9mg vs placebo, QD	6mg vs placebo, QD	40mg vs placebo, BID	1:1:1:1:1 in 2, 5, 15, or 30 mg or placebo	1:1:1 in 6mg vs 12mg vs placebo	200mg vs 400mg vs placebo	low, mid, high dose vs placebo, QD	45mg vs 90mg vs placebo	-
Treatment duration	12 weeks	16 weeks	12 weeks	12 weeks	12 weeks	16 weeks	12 weeks	12 weeks	12 weeks, 48 weeks
PASI 75	77.3% or 78.6% vs 11.6%	68.8% vs 8.1%	64.1% vs 0	18% vs 44% vs 68% vs 67% vs 6%	28.6% vs 72.1% vs 7.5%	46% vs 72% vs 9% (estimate)	85.0% - 90.0% vs 12.5%	67.1% vs 66.4% vs 3.1% (PHOENIX1); 66.7% vs 75.7% vs 3.7% (PHOENIX2);	85% vs 80% (week 48)
PASI 75 (best results, placebo adj)	67%	61%	64%	62%	65%	63%	78%	72%	85% vs 80% (week 48)
PASI 90	36.4% or 50.0% vs 0	38.2% vs 1.4%	38.5% vs 0	8% vs 21% vs 45% vs 46% vs 0%	-	33.0% vs 46.5% vs around 5%	70.7% - 77.5% vs 5.0%	41.6% vs 36.7% vs 2.0% (PHOENIX1); 42.3% vs 50.9% vs 0.7% (PHOENIX2);	75% vs 68% (week 12, estimate) 84% vs 70% (week 48)
PASI 90 (best results, placebo adj)	**50%**	**37%**	**39%**	**46%**	-	**42%**	**73%**	**50%**	**75% vs 68% (week 12, estimate)**
sPGA 0/1	70.5% or 71.4% vs 9.3%	55.6% vs 6.8%	59.0% vs around 8%	10% vs 27% vs 49% vs 52% vs 0%	33.3% vs 65.1% vs 10.0%	45% vs 70% vs 16%	80.5% - 87.5% vs 20.0%	59% vs 61% vs 4% (PHOENIX1); 68% vs 73% vs 4% (PHOENIX2);	-
sPGA 0/1 (best results, placebo adj)	**62%**	**49%**	**51%**	**52%**	**55%**	**54%**	**68%**	**69%**	**-**
Source	Link	Link	Link	Link	Link	Link	Link	Link	Link

Source: PharmCube, Pubmed, CMBIGM

In a cross-trial comparison, the 50% placebo-adjusted PASI 90 and 62% placebo-adjusted sPGA 0/1 achieved by ICP-488 for psoriasis demonstrate strong competitiveness compared to other TYK2-targeted innovative therapies, such as

deucravacitinib, ESK-001, and zasocitinib. The efficacy of ICP-488 was even comparable to certain biologics, such as ustekinumab, which delivered a PASI 90 of 50% and sPGA 0/1 of 69% in clinical studies.

While injectable biologics targeting IL-23p19 and IL-17 represent highly effective therapies for psoriasis—such as guselkumab and secukinumab, which achieved PASI 90 scores of 75% and 68%, respectively, at week 12 in the ECLIPSE study—ICP-488 nonetheless offers a compelling oral alternative. ICP-488's oral route of administration provides significant convenience and accessibility for patients, in our view.

Notably, D-2570, a TYK2 inhibitor developed by InventisBio (益方生物), has emerged as a formidable contender in the TYK2 inhibitor market. In its Ph2 study, D-2570 demonstrated impressive efficacy, achieving placebo-adjusted PASI 90 and sPGA 0/1 response rates of 73% and 68%, respectively, further highlighting the potential of TYK2 inhibitors as a competitive class of therapies for psoriasis.

Valuation

We expect orelabrutinib to continue to deliver robust sales in the oncology market, driven by the rising penetration and growing market share in CLL, MCL and MZL, where orelabrutinib remains the only approved BTKi for MZL. Supported by a solid cash position and steady cash inflows driven by strong orelabrutinib sales, we remain optimistic about InnoCare's further clinical advancement in autoimmune diseases, including the development of key assets such as orelabrutinib, ICP-332, and ICP-488. We derive our DCF-based TP as HK$7.91 (WACC: 12.07%, terminal growth rate: 2.0%).

Figure 15: Risk-adjusted DCF valuation

DCF Valuation (RMB mn)	2024E	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E	2034E	2035E
EBIT	(610)	(522)	(392)	(206)	12	371	782	1,302	1,623	1,889	2,024	2,169
Tax rate	0%	0%	0%	0%	15%	15%	15%	15%	15%	15%	15%	15%
EBIT*(1-tax rate)	(610)	(522)	(392)	(206)	11	316	665	1,107	1,379	1,606	1,721	1,844
+ D&A	62	62	62	62	62	62	62	62	62	62	62	62
- Change in working capital	130	105	188	161	170	176	41	(121)	(200)	(210)	(257)	(281)
- Capex	(100)	(100)	(100)	(100)	(100)	(100)	(100)	(100)	(100)	(100)	(100)	(100)
FCFF	(518)	(455)	(243)	(84)	142	453	667	947	1,141	1,357	1,426	1,524
Terminal value												**15,443**
FCF + Terminal value	(518)	(455)	(243)	(84)	142	453	667	947	1,141	1,357	1,426	16,967

PV of enterprise (RMB mn)	6,698
Net debt (RMB mn)	-6,269
Minorities	4
Equity value (RMB mn)	12,963
Corporate value (HK$mn)	**13,939**
No. of shares (mn)	1,763
DCF per share (HK$)	**7.91**
Terminal growth rate	**2.00%**
WACC	**12.07%**
Cost of Equity	15.6%
Cost of Debt	4.50%
Equity Beta	1.20
Risk Free Rate	3.00%
Market Risk Premium	10.50%
Target Debt to Asset ratio	30.00%
Effective Corporate Tax Rate	15.00%

Source: CMBIGM estimates

Figure 16: Sensitivity analysis (HK$)

	WACC				
Terminal growth rate	**11.07%**	**11.57%**	**12.07%**	**12.57%**	**13.07%**
3.0%	9.09	8.64	8.23	7.88	7.56
2.5%	8.86	8.44	8.06	7.73	7.43
2.0%	8.65	8.26	**7.91**	7.60	7.32
1.5%	8.46	8.09	7.77	7.48	7.21
1.0%	8.29	7.95	7.64	7.36	7.11

Source: CMBIGM estimates

Figure 17: CMBIGM estimates vs consensus

	CMBIGM			Consensus			Diff (%)		
RMB mn	**FY24E**	**FY25E**	**FY26E**	**FY24E**	**FY25E**	**FY26E**	**FY24E**	**FY25E**	**FY26E**
Revenue	1,009	1,475	1,966	983	1,313	1,708	3%	12%	15%
Gross profit	859	1,263	1,671	842	1,131	1,476	2%	12%	13%
Operating profit	(452)	(360)	(244)	(645)	(586)	(445)	N/A	N/A	N/A
Net profit	(457)	(375)	(250)	(520)	(445)	(323)	N/A	N/A	N/A
EPS (RMB)	(0.25)	(0.20)	(0.13)	(0.29)	(0.24)	(0.14)	N/A	N/A	N/A
Gross margin	85.12%	85.64%	85.00%	85.63%	86.13%	86.43%	-0.51 ppt	-0.49 ppt	-1.43 ppt

Source: Company data, Bloomberg, CMBIGM estimates

Financial Summary

INCOME STATEMENT	2021A	2022A	2023A	2024E	2025E	2026E
YE 31 Dec (RMB mn)						
Revenue	**1,043**	**625**	**739**	**1,009**	**1,475**	**1,966**
Cost of goods sold	(66)	(143)	(128)	(150)	(212)	(295)
Gross profit	**977**	**482**	**610**	**859**	**1,263**	**1,671**
Operating expenses	**(1,212)**	**(1,547)**	**(1,458)**	**(1,539)**	**(1,835)**	**(2,114)**
Selling expense	(298)	(439)	(367)	(450)	(565)	(728)
Admin expense	(140)	(182)	(194)	(202)	(273)	(305)
R&D expense	(722)	(639)	(751)	(886)	(997)	(1,082)
Others	(52)	(288)	(147)	0	0	0
Other income	218	198	244	228	212	199
Other expense	(3)	(17)	(35)	(26)	(15)	(7)
Gain/loss on financial assets at FVTPL	0	0	0	20	0	0
Share of (losses)/profits of associates/JV	(1)	(10)	(5)	0	0	0
Pre-tax profit	**(20)**	**(894)**	**(644)**	**(457)**	**(375)**	**(250)**
Income tax	(47)	0	(1)	0	0	0
After tax profit	**(67)**	**(894)**	**(646)**	**(457)**	**(375)**	**(250)**
Minority interest	2	7	14	14	14	14
Net profit	**(65)**	**(887)**	**(631)**	**(443)**	**(361)**	**(236)**

BALANCE SHEET	2021A	2022A	2023A	2024E	2025E	2026E
YE 31 Dec (RMB mn)						
Current assets	**6,417**	**9,300**	**8,765**	**8,010**	**7,555**	**7,437**
Cash & equivalents	5,929	8,698	8,225	7,459	6,751	6,351
Account receivables	45	128	308	300	443	596
Inventories	10	65	119	108	152	212
Financial assets at FVTPL	317	313	0	0	0	0
Other current assets	116	95	114	143	209	279
Non-current assets	**980**	**1,021**	**1,154**	**1,192**	**1,231**	**1,269**
PP&E	430	653	760	801	842	883
Right-of-use assets	136	284	294	292	290	288
Investment in JVs & assos	21	12	6	6	6	6
Intangibles	34	41	39	39	38	38
Goodwill	3	3	3	3	3	3
Other non-current assets	356	28	52	52	52	52
Total assets	**7,398**	**10,321**	**9,919**	**9,203**	**8,786**	**8,706**
Current liabilities	**329**	**2,075**	**2,094**	**1,835**	**1,793**	**1,963**
Short-term borrowings	0	1,197	1,256	856	456	156
Account payables	85	119	135	148	209	291
Other current liabilities	218	735	680	807	1,104	1,493
Lease liabilities	20	20	23	23	23	23
Contract liabilities	7	4	0	0	0	0
Non-current liabilities	**1,409**	**601**	**644**	**644**	**644**	**644**
Long-term borrowings	0	0	26	26	26	26
Convertible bonds	1,201	0	0	0	0	0
Deferred income	124	278	269	269	269	269
Other non-current liabilities	85	323	349	349	349	349
Total liabilities	**1,739**	**2,677**	**2,738**	**2,479**	**2,437**	**2,608**
Share capital	0	0	0	0	0	0
Other reserves	5,605	7,597	7,148	6,705	6,344	6,109
Total shareholders equity	**5,605**	**7,597**	**7,148**	**6,705**	**6,345**	**6,109**
Minority interest	54	47	33	18	4	(10)
Total equity and liabilities	**7,398**	**10,321**	**9,919**	**9,203**	**8,786**	**8,706**

CASH FLOW	2021A	2022A	2023A	2024E	2025E	2026E
YE 31 Dec (RMB mn)						
Operating						
Profit before taxation	**(20)**	**(894)**	**(644)**	**(457)**	**(375)**	**(250)**
Depreciation & amortization	10	35	59	59	59	59
Tax paid	0	0	(0)	0	0	0
Change in working capital	120	(75)	(244)	130	105	188
Others	57	368	104	(170)	(144)	(140)
Net cash from operations	**167**	**(565)**	**(726)**	**(438)**	**(355)**	**(143)**
Investing						
Capital expenditure	(166)	(227)	(255)	(100)	(100)	(100)
Net proceeds from disposal of short-term investments	(1,637)	(1,509)	796	0	0	0
Others	73	10	125	178	162	149
Net cash from investing	**(1,730)**	**(1,726)**	**667**	**78**	**62**	**49**
Financing						
Dividend paid	0	0	0	0	0	0
Net borrowings	0	0	31	(400)	(400)	(300)
Proceeds from share issues	2,562	3,120	4	20	0	0
Others	(17)	(25)	(28)	(26)	(15)	(7)
Net cash from financing	**2,545**	**3,095**	**8**	**(406)**	**(415)**	**(307)**
Net change in cash						
Cash at the beginning of the year	2,301	5,929	8,698	8,225	7,459	6,751
Exchange difference	(45)	140	26	0	0	0
Cash at the end of the year	**5,929**	**8,698**	**8,721**	**7,459**	**6,751**	**6,351**

GROWTH	2021A	2022A	2023A	2024E	2025E	2026E
YE 31 Dec						
Revenue	76,368.7%	(40.0%)	18.1%	36.6%	46.2%	33.3%
Gross profit	71,554.4%	(50.7%)	26.6%	40.8%	47.1%	32.3%

PROFITABILITY	2021A	2022A	2023A	2024E	2025E	2026E
YE 31 Dec						
Gross profit margin	93.7%	77.1%	82.6%	85.1%	85.6%	85.0%
Return on equity (ROE)	(1.5%)	(13.4%)	(8.6%)	(6.4%)	(5.5%)	(3.8%)

GEARING/LIQUIDITY/ACTIVITIES	2021A	2022A	2023A	2024E	2025E	2026E
YE 31 Dec						
Current ratio (x)	19.5	4.5	4.2	4.4	4.2	3.8
Receivable turnover days	107.6	110.6	111.6	112.6	113.6	114.6
Inventory turnover days	262.0	262.0	262.0	262.0	262.0	262.0
Payable turnover days	360.2	360.2	360.2	360.2	360.2	360.2

VALUATION	2021A	2022A	2023A	2024E	2025E	2026E
YE 31 Dec						
P/E	ns	ns	ns	ns	ns	ns

Source: Company data, CMBIGM estimates. Note: The calculation of net cash includes financial assets.

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