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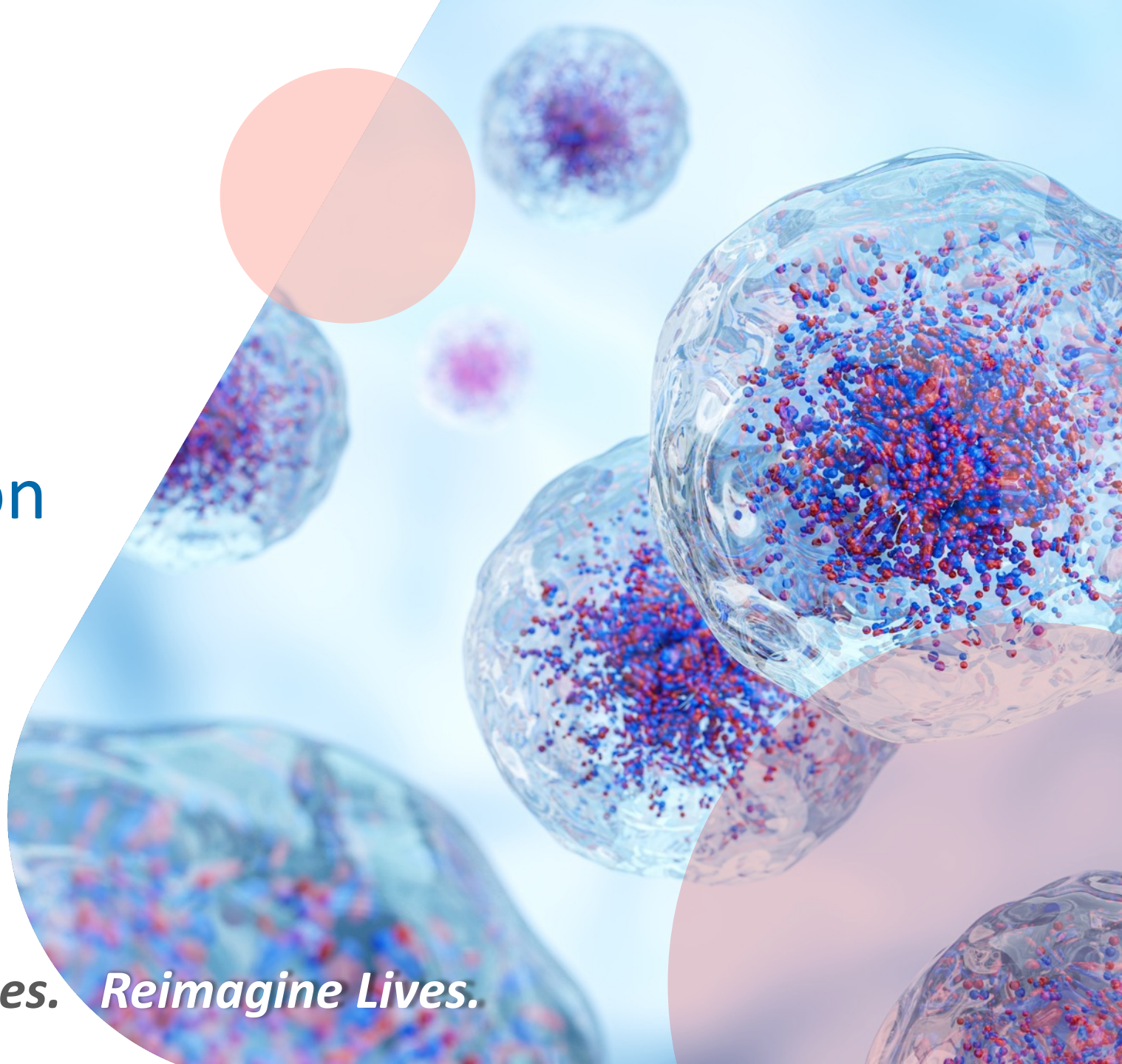
Corporate Presentation

September 2024



Transform Therapies. Reimagine Lives.

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Corporate Overview

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Developing Oral Therapies To Transform Lives of Patients With Immune-Mediated Diseases

ESK-001

ESK-001, potentially the first and only allosteric TYK2 inhibitor well-tolerated at maximal target inhibition

- Demonstrated maximal target inhibition, potential best-in-class tolerability profile in plaque psoriasis (PsO)
- Multibillion dollar market opportunity[1] in a broad set of indications, including systemic lupus erythematosus (SLE)
- Ongoing ONWARD Phase 3 clinical trials in PsO and LUMUS Phase 2b trial in SLE

A-005

A-005, a Phase 1 CNS penetrant allosteric TYK2 inhibitor for the treatment of neuroinflammatory and neurodegenerative diseases

- Potential first- and best-in-class opportunity with blood-to-brain ratio of approximately 1:1

APPROACH

Precision approach to replace broad immuno-suppression with targeted therapies

- Precision data analytics platform generating genetic, genomic, proteomic, and biological and clinical disease insights
- Accelerate research and development and increase the probability of clinical success

CATALYSTS

Anticipated value-creating near-term catalysts

- Multiple milestones in 2024 / 2025, including readout for A-005 Phase 1 trial in healthy volunteers
- Catalyst rich 2026 with topline data for Phase 3 trials in PsO, Phase 2b trial in SLE and Phase 2 trial in multiple sclerosis (MS)

LEADERSHIP

Experienced team with strong track record in value creation

- Strong financial position and backed by established blue-chip life science investors

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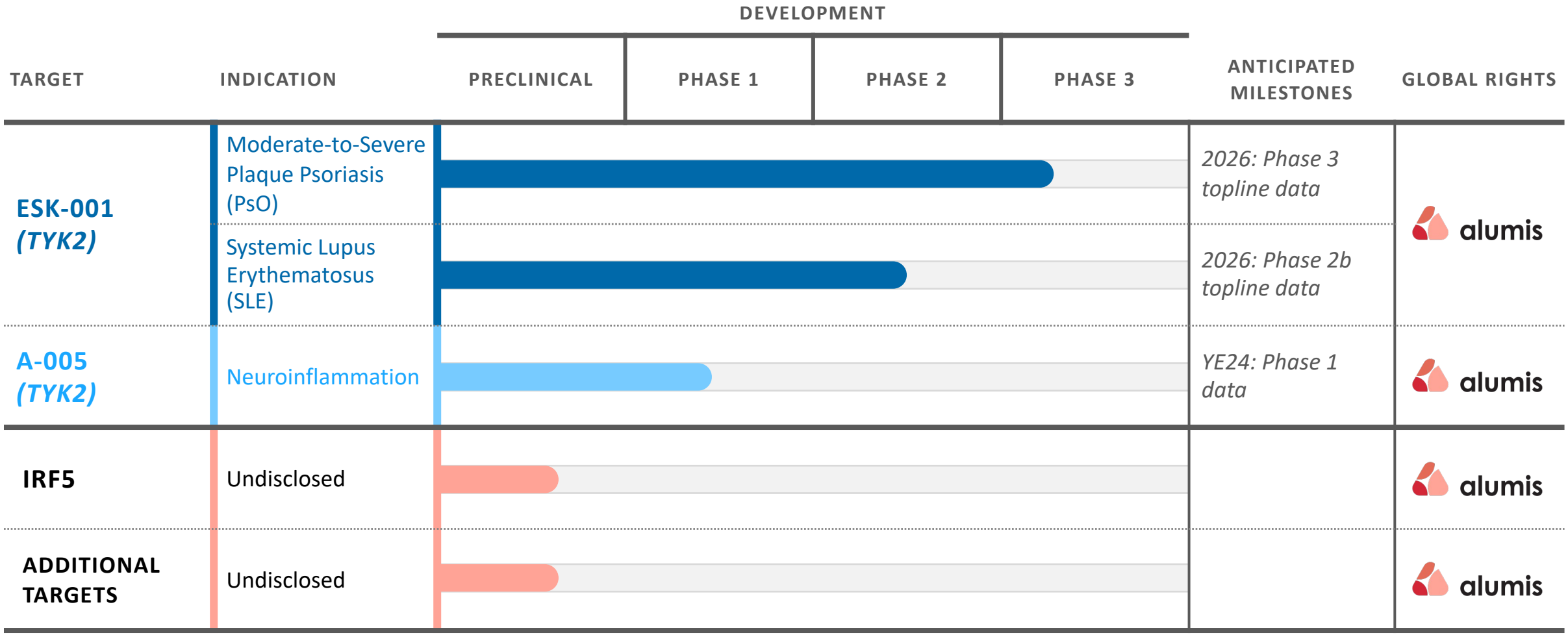
1. Based on current internal estimates

Alumis is Building a Wholly Owned, Diverse Precision Therapeutics Portfolio



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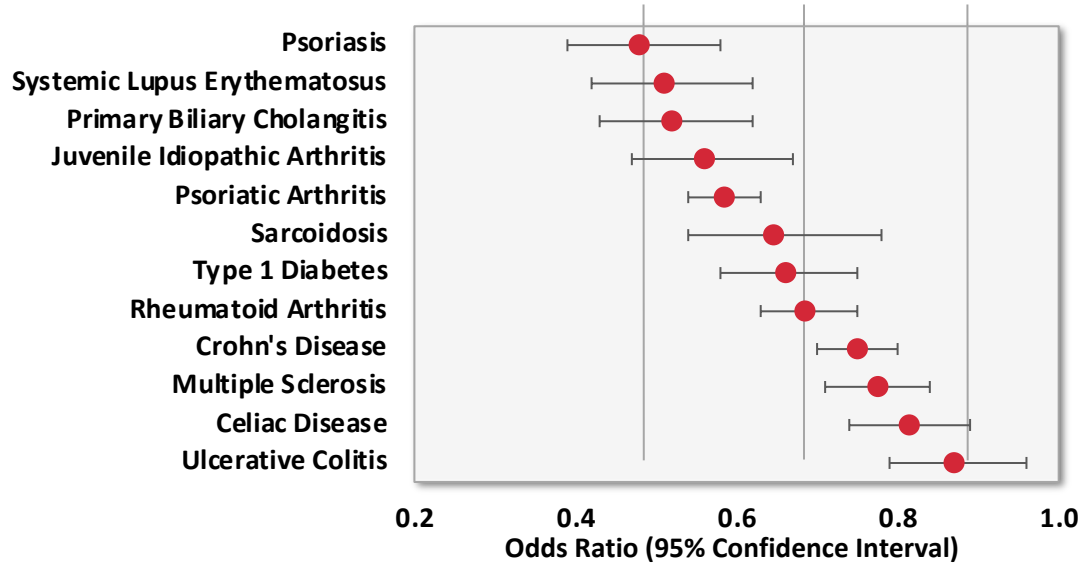
Alumis' Precision Approach and Capabilities

Precision Approach

- Drug targets selected with strong human genetic evidence or with human clinical validation
- Proprietary genetic database
- Comprehensive biomarker collection and profiling
- Data platform with large multi modal data sets utilizing modern AI- and ML-based methods
- Differentiated molecules designed to achieve maximal target engagement

Example of Application: The "Right Indication"

Association of TYK2 P1104A Loss of Function Variant with Immune Mediated Diseases



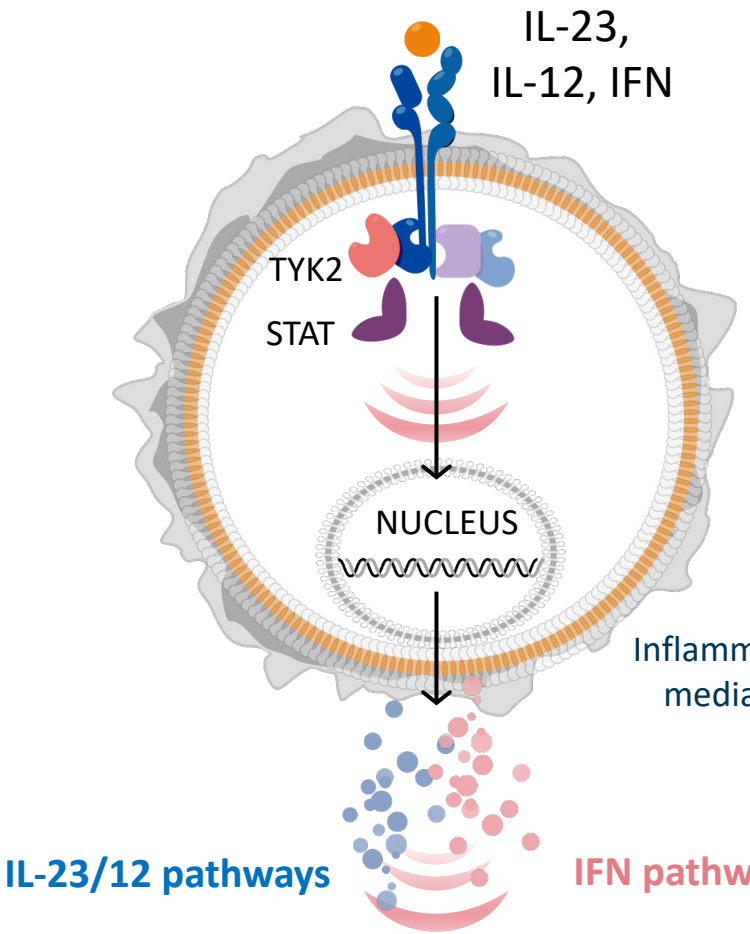
Potential to increase speed of development, probability of success and precision of therapy

Genetic associations established by proprietary comprehensive immune-focused database and advanced multi-trait statistical methods

Note: Forest plot of immune-mediated indications associated with TYK2 loss-of-function variant P1104A. Point estimates below 1 suggest protection against disease while point estimates above 1 suggest susceptibility to disease, with points furthest away from "1.0" indicating highest level of association

TYK2 Pipeline-in-a-Product Opportunity

- **Pathogenic TYK2 signaling** is associated with the development of **immune mediated diseases** in both the periphery and CNS
- TYK2 mediates signaling from key validated proinflammatory cytokines, including **IL-23, IL-12 and Type I IFN**
- We believe allosteric inhibition of TYK2 has potential to treat a **wide array of diseases** with a **benign safety profile**
- Orals offer opportunity for **greater tissue penetration, immediate treatment cessation** and **convenience**



- Psoriasis
- SLE

- Multiple Sclerosis
- IBD
- Psoriatic Arthritis
- Other I&I Diseases

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ESK-001: Our Allosteric TYK2 Inhibitor

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ESK-001: Potent and Highly Selective Allosteric TYK2 Inhibitor Designed to Achieve Maximal Target Inhibition

Designed to Deliver Potentially Best-in-class Pharmacokinetic Properties

- Dose-dependent exposure with very low variability
- Excellent penetration into relevant tissues
- Robust PK/PD achieves maximal target inhibition

No Clinically Limiting Findings

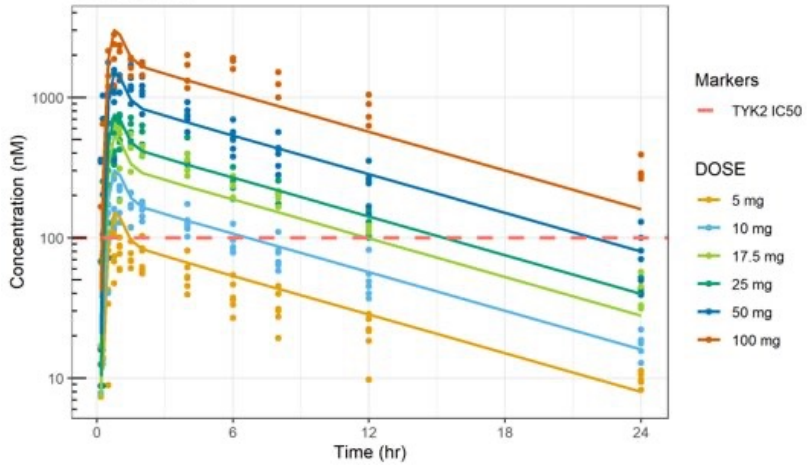
- Highly selective for TYK2 with no off-target JAK pharmacology
- Enabling clinical pharmacology profile including no drug-drug interactions

Only Clinical TYK2 to Safely Achieve Maximal Target Inhibition

		Steady State Time Above (hr)	
Drug	**Dose**	**IC50**	**IC90**
ESK-001	40 mg QD	19	7
	40 mg BID	>24	>24
Sotyktu	6mg QD	9	0
TAK-279	30mg QD	>24	5

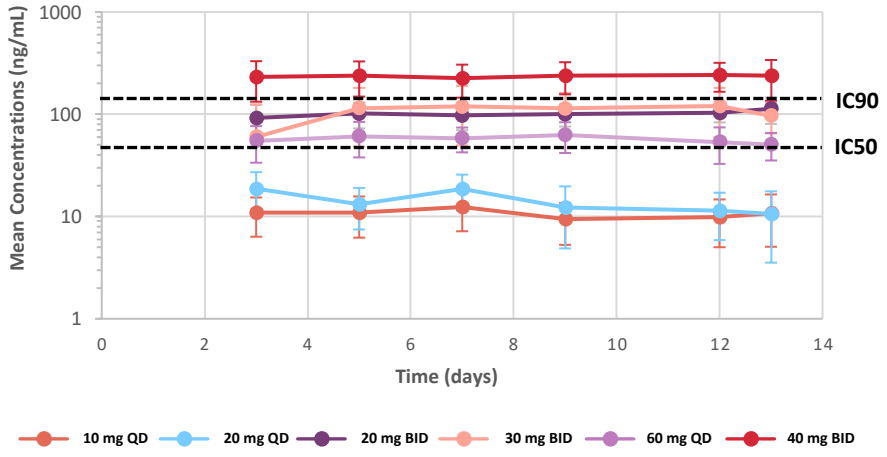
Dose-dependent Exposure, Very Low Variability

ESK-001 Phase 1 SAD PK

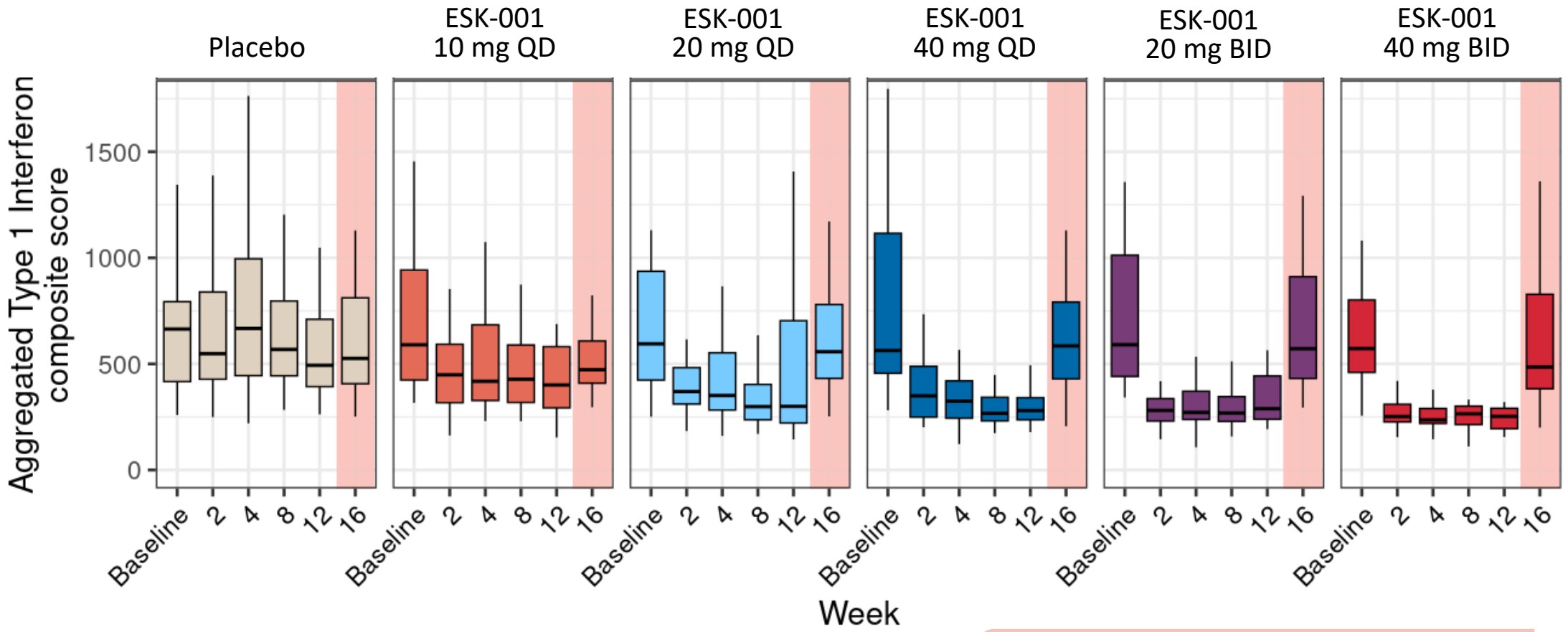


Coverage of IC90 at Trough with 40mg BID Dose

ESK-001 Phase 1 Multidose, Trough PK



Maximal Inhibition of Type I IFN Gene Signature Shown in RNA-seq of Phase 2 Blood Samples



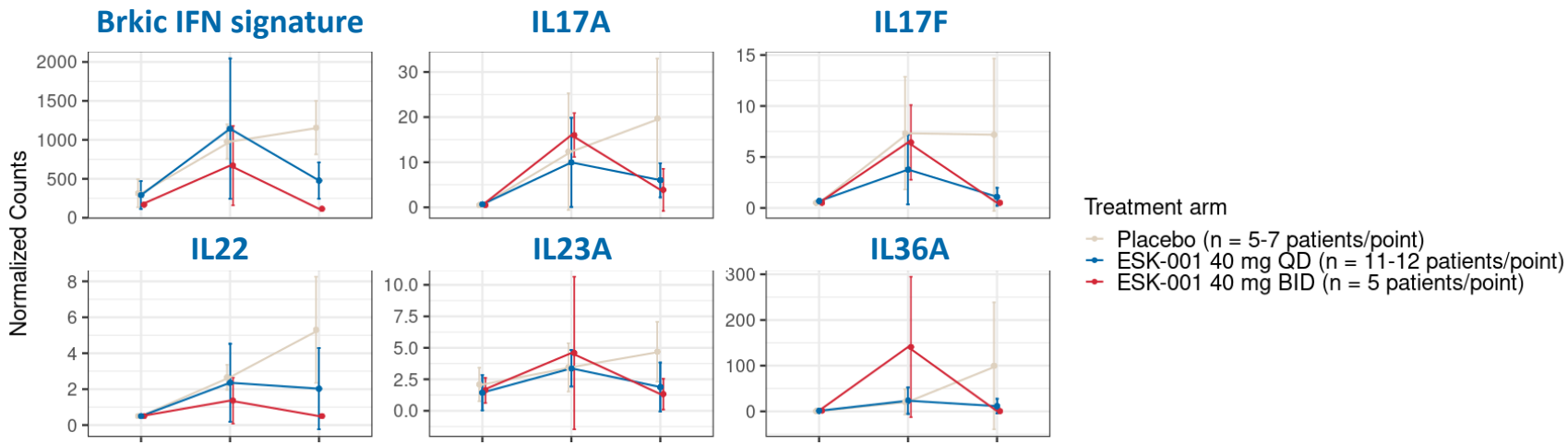
Note: Pink Bar Reflects Samples from End of Drug Washout Period (Week 12 to Week 16)

Note: Blood samples collected at trough

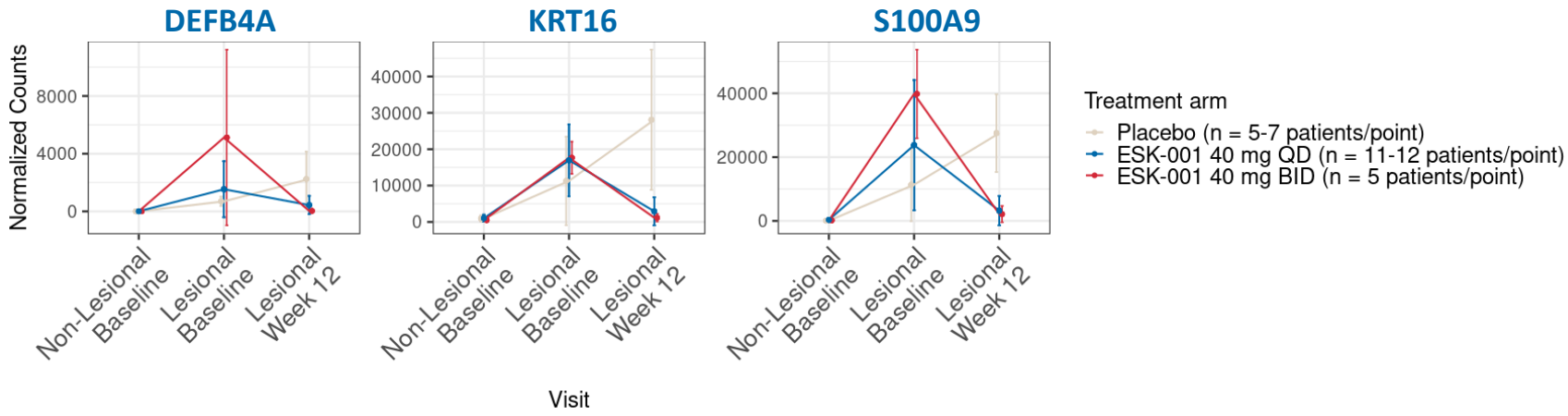
Phase 2 Skin Biopsy RNA-seq Confirms Maximal Inhibition

Lesional Skin Levels of Key Cytokines & Disease Related Biomarkers Return to Non-lesional Levels

TYK2 Pathway Key Cytokines

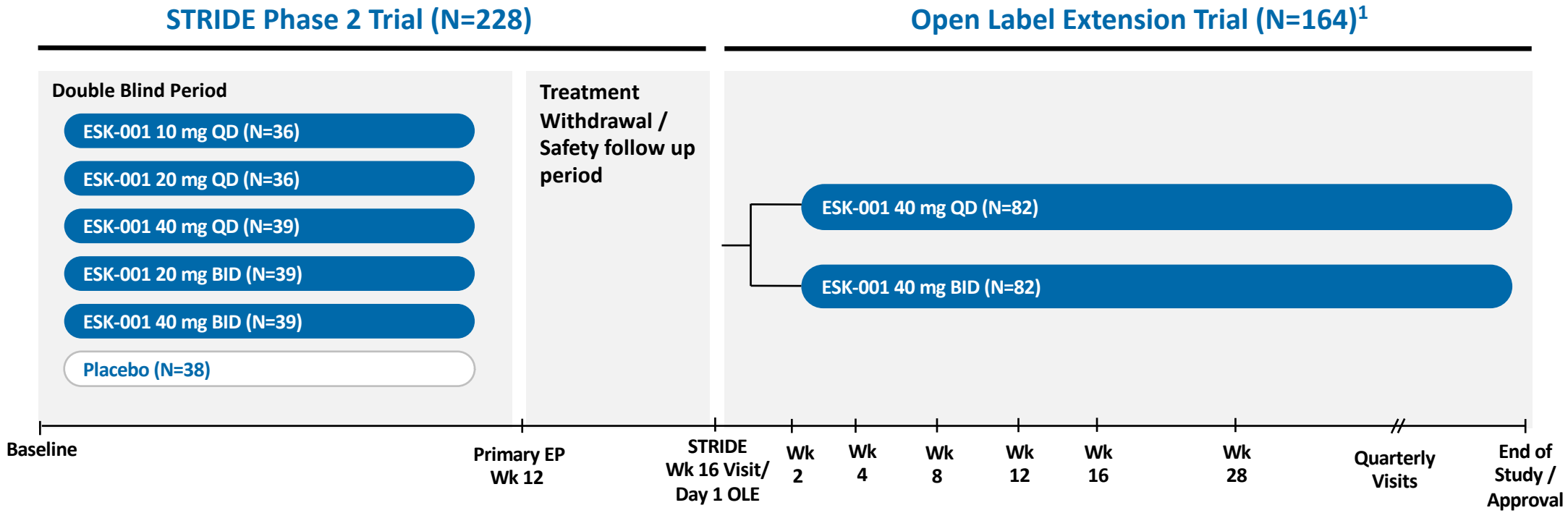


Disease Biomarkers



ESK-001 Psoriasis Phase 2 Skin biopsy RNA-seq, 12/05/2023 dataset.
Skin biopsies collected in subset of patients, all available skin biopsy samples with valid RNAseq data included in analysis.

ESK-001 Phase 2 STRIDE and OLE Studies Designed to Assess Both Short- and Long-Term Efficacy, Safety and Tolerability



Stride Phase 2 Study

- **Key Inclusion Criteria:** adults 18-75 years with plaque psoriasis
  - PASI ≥ 12, sPGA ≥ 3, BSA ≥ 10%
- **1°EP:** PASI 75 Response at Week 12
- **Key 2°EPs at Week 12**: PASI 90, PASI 100, sPGA 0/1, and sPGA 0

Open Label Extension Study

- **OLE Dose Assignment:** same or higher dose as in parent study
- **Safety EPs**: Incidence of TEAEs and SAEs over time
- **Key Efficacy EPs:** PASI-75, PASI-90 and PASI-100; sPGA 0/1 and sPGA 0

TEAE: Treatment emergent adverse event. SAE: Serious adverse event.

1. 165 eligible patients were randomized; of these, 164 patients were assigned to receive either 40 mg BID or 40 mg QD (one patient was not dosed and was not included in the population analysis)

STRIDE: Demographics and Baseline Disease Characteristics Were Well-Balanced Across Study Arms

	Placebo (N=38)	10 mg QD (N=36)	20 mg QD (N=36)	20 mg BID (N=40)	40 mg QD (N=39)	40 mg BID (N=39)	Overall (N=228)
Age, mean (SD)	49.1 (11.7)	48.8 (12.7)	43.9 (12.0)	47.7 (12.5)	49.5 (10.5)	47.9 (14.2)	47.8 (12.3)
Male, n (%)	31 (81.6)	24 (66.7)	24 (66.7)	23 (57.5)	26 (66.7)	26 (66.7)	154 (67.5)
Race, n (%) White Asian Black/African American Other	 27 (71.1) 4 (10.5) 3 (7.9) 4 (10.5)	 30 (83.3) 1 (2.8) 4 (11.1) 1 (2.8)	 31 (86.1) 2 (5.6) 0 3 (8.3)	 34 (85.0) 2 (5.0) 1 (2.5) 3 (7.5)	 33 (84.6) 2 (5.1) 1 (2.6) 3 (7.7)	 33 (84.6) 2 (5.1) 1 (2.6) 3 (7.7)	 188 (82.5) 13 (5.7) 10 (4.4) 17 (7.5)
BMI (kg/m^2), mean (SD)	31.9 (6.8)	30.5 (5.9)	34.9 (12.1)	31.7 (7.4)	30.4 (6.4)	31.6 (7.1)	31.8 (7.9)
Psoriasis Duration (years), mean (SD)	19.8 (11.6)	19.3 (13.4)	17.3 (8.3)	21.8 (12.2)	16.7 (12.4)	21.5 (15.5)	19.4 (12.5)
PASI score, mean (SD)	18.0 (4.5)	16.5 (3.9)	18.9 (6.6)	18.3 (6.5)	17.4 (6.5)	17.5 (4.9)	17.8 (5.6)
PGA score, n (%) 3 (moderate) 4 (marked) 5 (severe)	 22 (57.9) 15 (39.5) 1 (2.6)	 24 (66.7) 9 (25.0) 3 (8.3)	 17 (47.2) 17 (47.2) 2 (5.6)	 23 (57.5) 16 (40.0) 1 (2.5)	 25 (64.1) 14 (35.9) 0	 23 (59.0) 16 (41.0) 0	 134 (58.8) 87 (38.2) 7 (3.1)
BSA, mean	22.9 (12.1)	20.6 (12.2)	19.9 (12.6)	21.4 (15.0)	20.1 (12.9)	21.5 (15.1)	21.1 (13.3)
Bioexperienced (biologics or JAKi), n(%)	13 (34.2)	13 (36.1)	14 (38.9)	16 (40.0)	13 (33.3)	13 (33.3)	82 (36.0)

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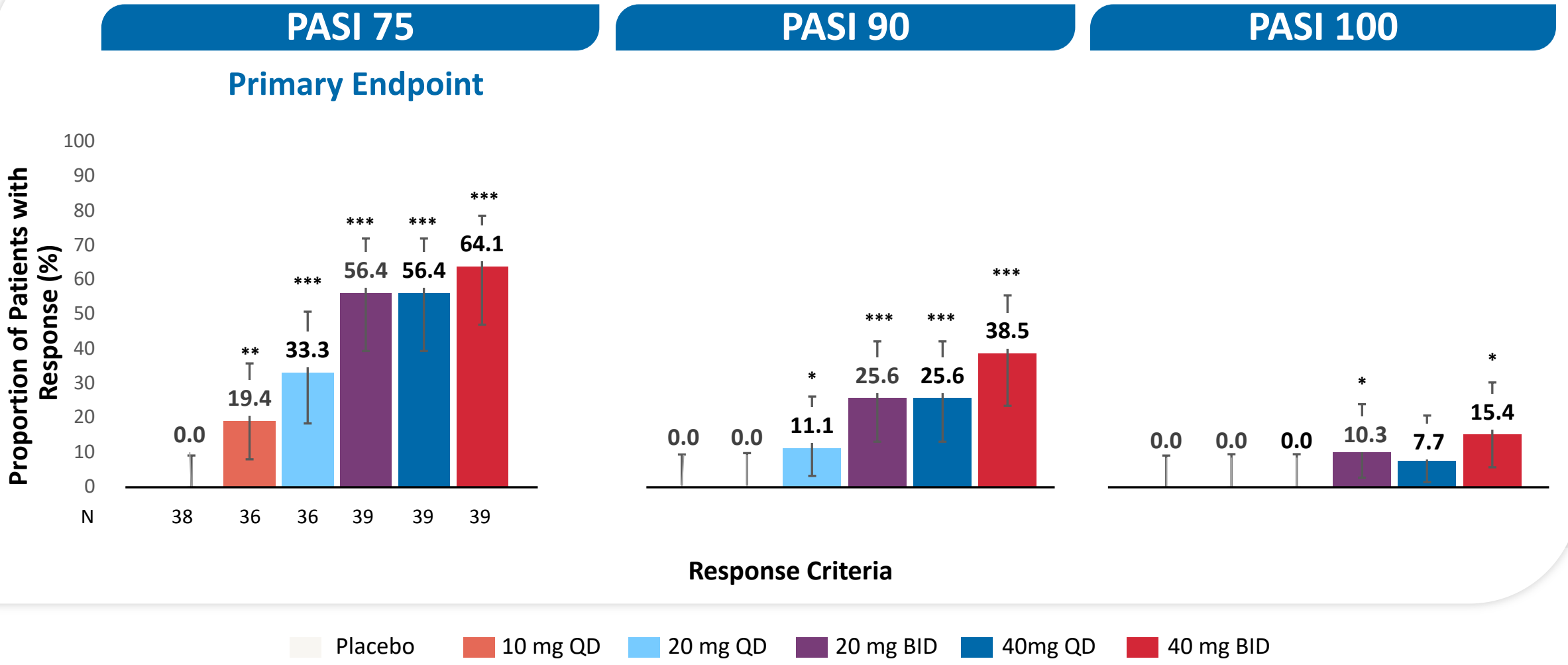
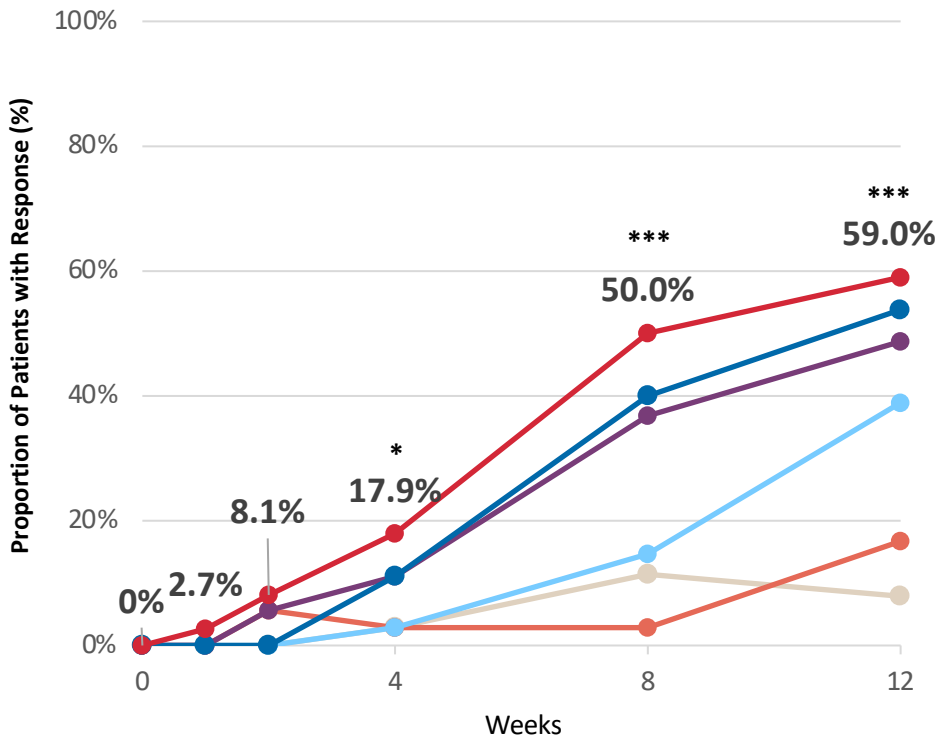
STRIDE: Primary and Secondary PASI Endpoints Achieved at Week 12 With Dose-Dependent Increase in Response Rates



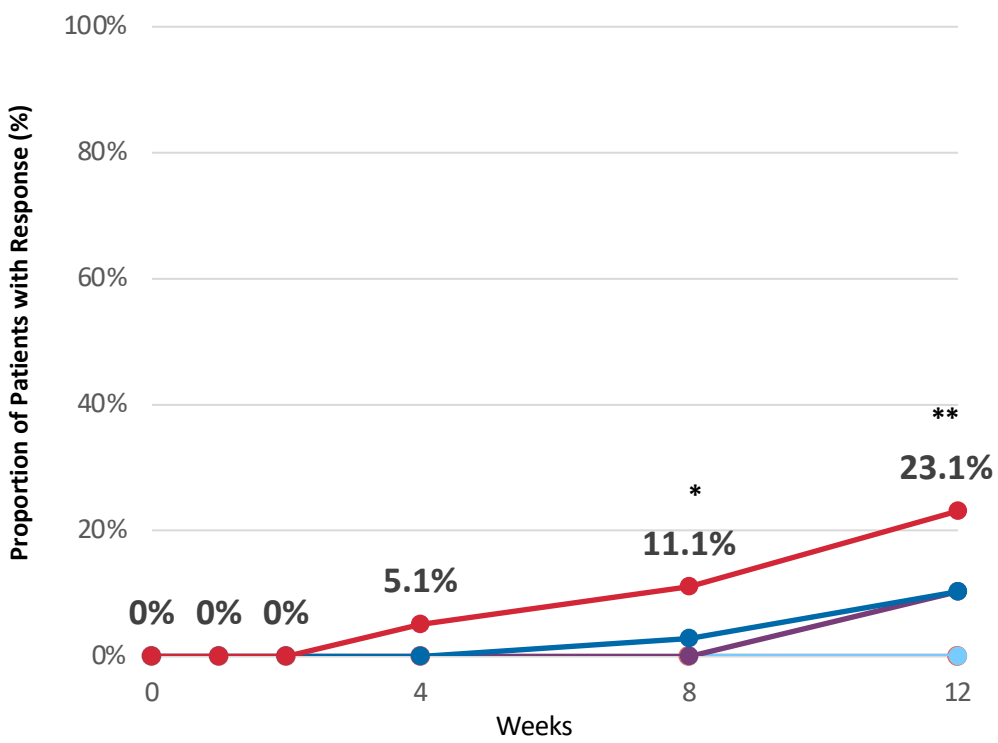
*p<0.05; **p< 0.005; ***p<0.001 . P-value is comparing proportion in each active arm vs placebo using the Cochran-Mantel-Haenszel test adjusted for stratification factors (prior use of biologics and geographic region (North American vs. ROW)). NRI imputation was applied for subjects who discontinued study.

STRIDE: Secondary sPGA Endpoints Achieved at Week 12 With Increasing Response Observed over Time

sPGA 0/1



sPGA 0



Placebo | 10 mg QD | 20 mg QD | 20 mg BID | 40mg QD | 40 mg BID

*p<0.05; **p< 0.005; ***p<0.001 . P-value is comparing proportion in each active arm vs placebo using the Cochran-Mantel-Haenszel test adjusted for stratification factors (prior use of biologics and geographic region (North American vs. ROW)). NRI imputation was applied for subjects who discontinued study.

STRIDE Safety Summary at Week 16

	Placebo (N=38)	10 mg QD (N=36)	20 mg QD (N=36)	20 mg BID (N=39)	40 mg QD (N=39)	40 mg BID (N=39)	Overall (N=227)
Subjects with ≥1 TEAE	15 (39.5)	19 (52.8)	14 (38.9)	18 (46.2)	19 (48.7)	25 (64.1)	110 (48.5)
Subjects with ≥1 SAE	0	1 (2.8)	0	3 (7.7)	1 (2.6)	0	5 (2.2)
Subjects with treatment related SAEs	0	0	0	0	0	0	0
Deaths	0	0	0	0	0	0	0
Subjects with TEAE leading to treatment discontinuation	0	0	2 (5.6)	0	2 (5.1)	1 (2.6)	5 (2.2)
Most frequent TEAEs							
Headache	2 (5.3)	0	2 (5.6)	3 (7.7)	4 (10.3)	3 (7.7)	14 (6.2)
Upper resp. tract infection	0	2 (5.6)	2 (5.6)	1 (2.6)	2 (5.1)	3 (7.7)	10 (4.4)
Nasopharyngitis	3 (7.9)	2 (5.6)	0	1 (2.6)	1 (2.6)	3 (7.7)	10 (4.4)

Note: No Major Adverse Cardiac Events (MACE), serious infections, cytopenias, treatment related thromboses or concerning lab/ECG trends were observed.
TEAE: treatment emergent adverse event.
Most frequent TEAEs: ≥3 patients where occurrence greater in active group vs. placebo.

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95% of Eligible STRIDE Subjects Continued in OLE Study

228
Randomized Phase 2 STRIDE Subjects

204
Subjects Completing STRIDE

165*
Eligible Subjects Entering OLE

9
Eligible for OLE but chose not to participate

30
Ineligible and not participating in OLE

25: OLE not initiated in Czech Republic**
5: Did not meet entry criteria

Open Label Extension Trial

- ESK-001 40 mg QD (N=82)
- ESK-001 40 mg BID (N=82)

* 1 Subject randomized into OLE but not dosed and not included in mITT population analyses

**Patients in the Czech Republic were not eligible to participate in the OLE because local regulatory requirements would not have been consistent with the global protocol

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OLE Safety Summary

	ESK-001 40 mg QD (N=82)	ESK-001 40 mg BID (N=82)	Overall (N=164)
Subjects with ≥ 1 TEAE	41 (50.0)	45 (54.9)	86 (52.4)
Subjects with ≥ 1 TE SAE	1 (1.2)	3 (3.7)	4 (2.4)
Deaths	0	0	0
Subjects with TEAE leading to treatment discontinuation	0	4 (4.9)	4 (2.4)
Subjects with TEAE ≥ Grade 3	1 (1.2)	4 (4.9)	5 (3.0)
Most frequent TEAEs			
Nasopharyngitis	10 (12.2)	3 (3.7)	13 (7.9)
Upper Respiratory Tract Infection	2 (2.4)	9 (11.0)	11 (6.7)
Folliculitis	0	3 (3.7)	3 (1.8)
Gastroenteritis	0	3 (3.7)	3 (1.8)
Urinary Tract Infection	0	3 (3.7)	3 (1.8)
Acne	2 (2.4)	3 (3.7)	5 (3.0)
Arthralgia	1 (1.2)	3 (3.7)	4 (2.4)
Headache	5 (6.1)	3 (3.7)	8 (4.9)
Cough	0	3 (3.7)	3 (1.8)

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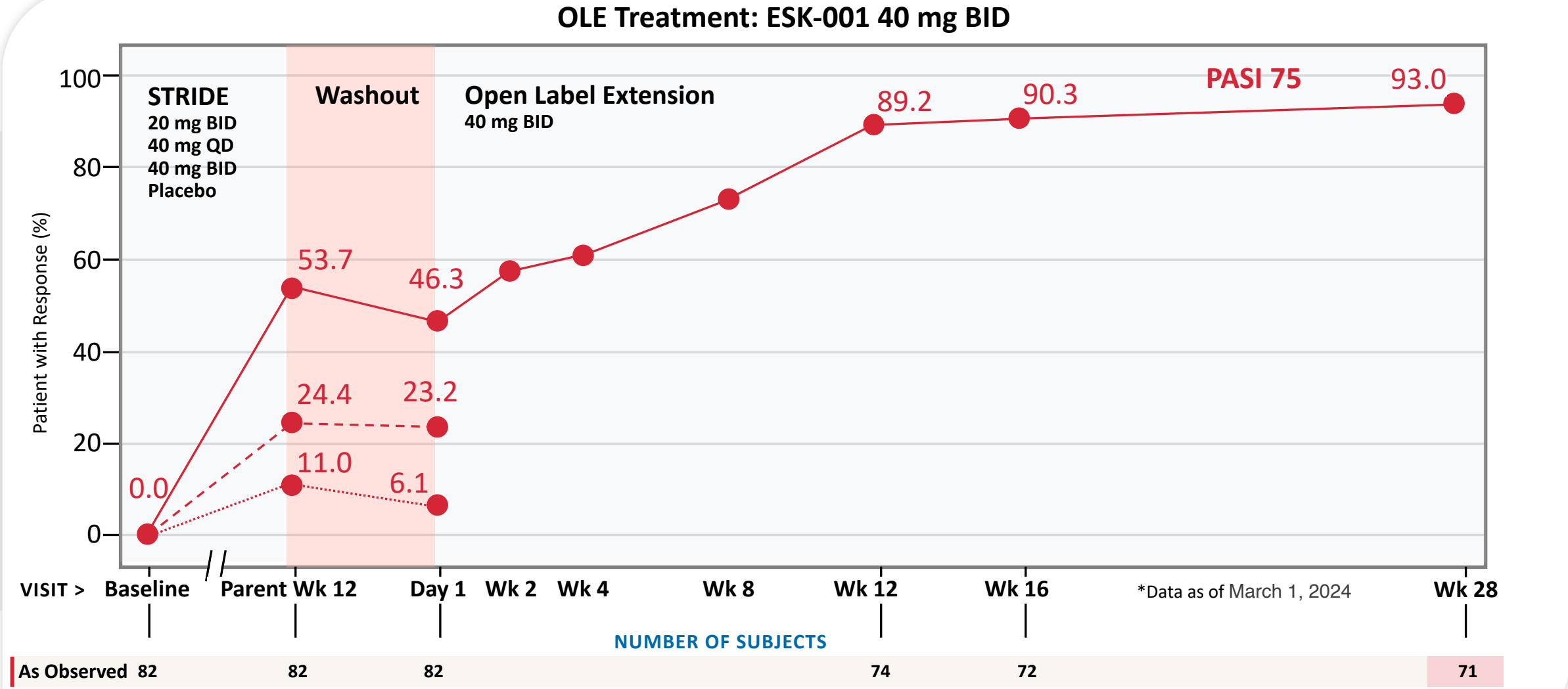
Based on the Safety Analysis Population (all treated patients). Safety data displayed based on 1 March 2024 data cut of ongoing OLE study. For the most recent OLE study safety information, please see the Company's filings with the SEC. TEAE: treatment emergent adverse event.

STRIDE and OLE: Continued ESK-001 Exposure Achieves Significant Increases in PASI Responses



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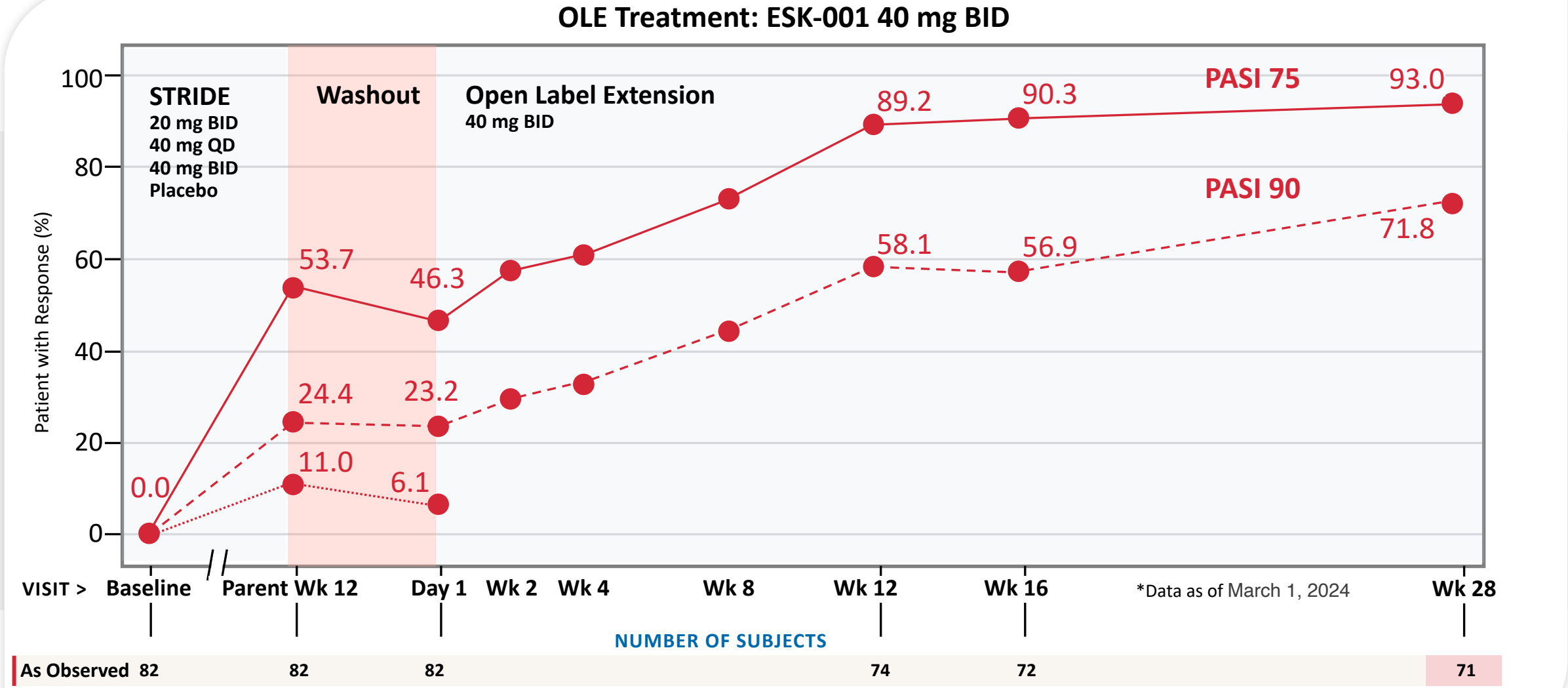

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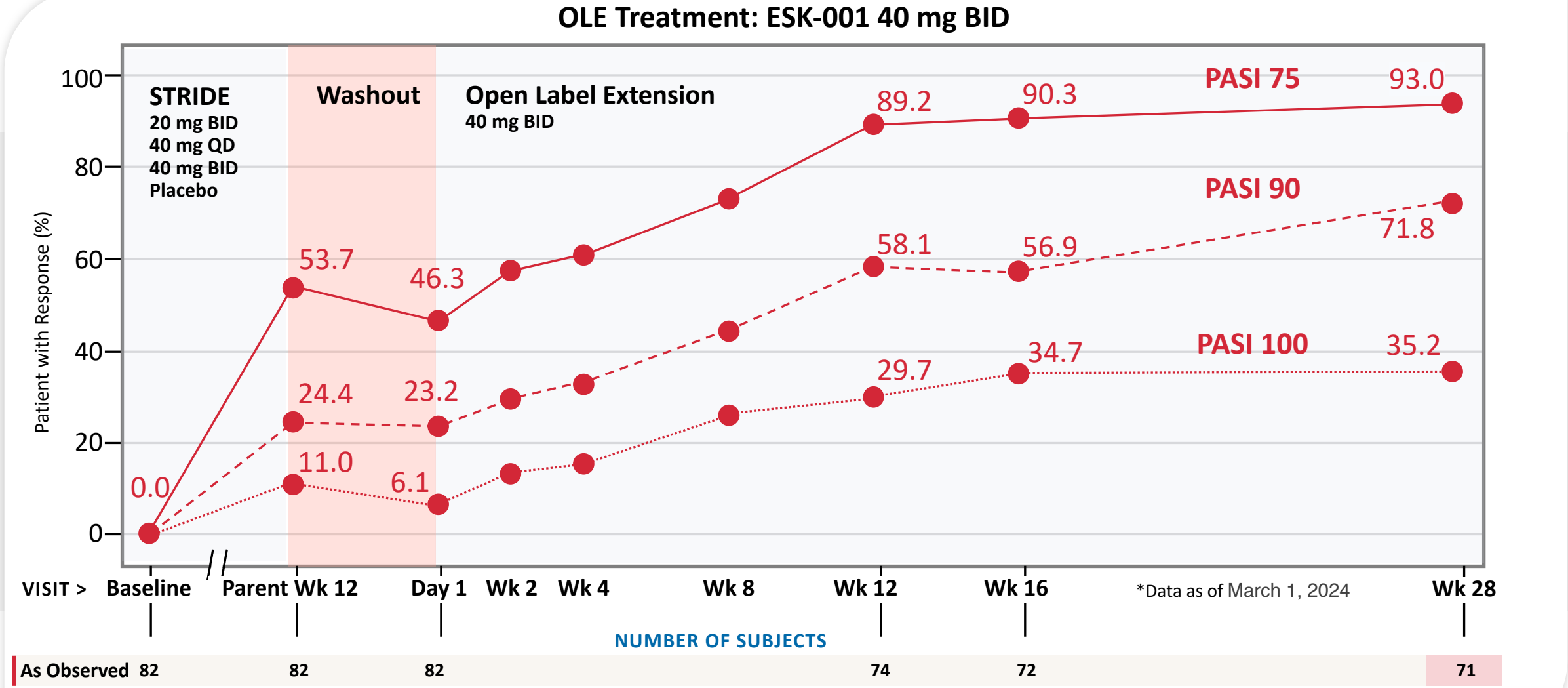

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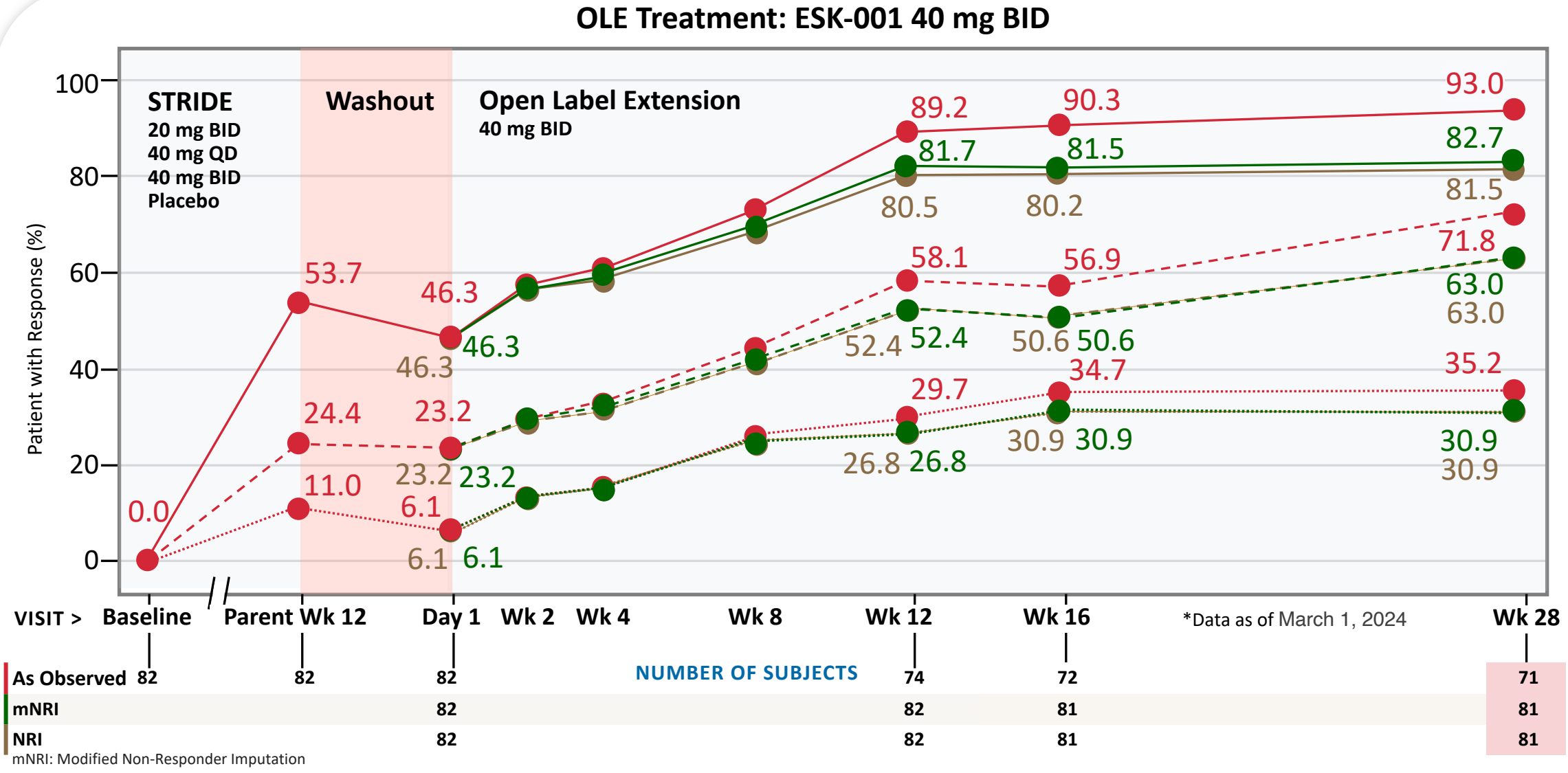

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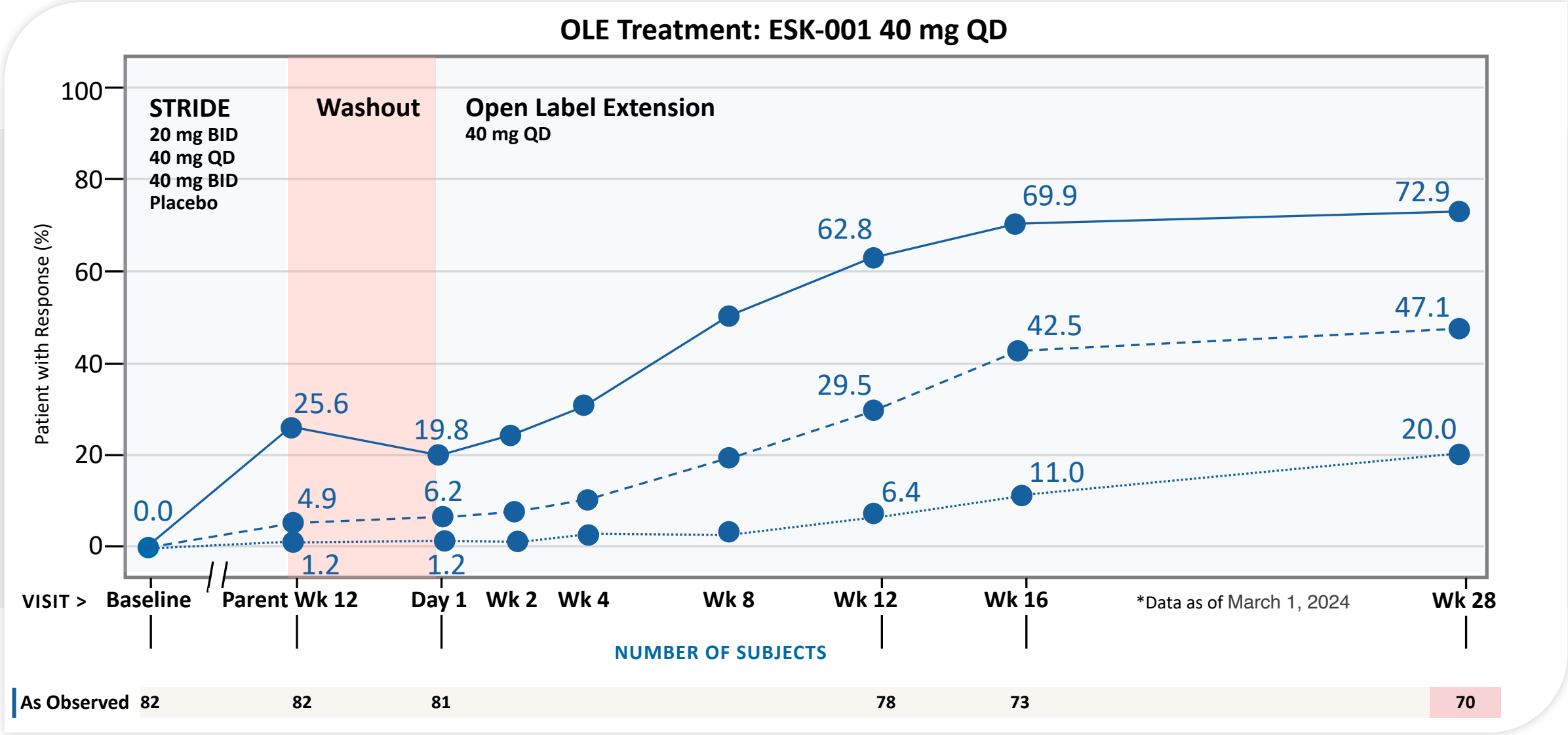
Incomplete Target Inhibition Results in a Substantially Lower PASI Response Compared to Maximal Target Inhibition



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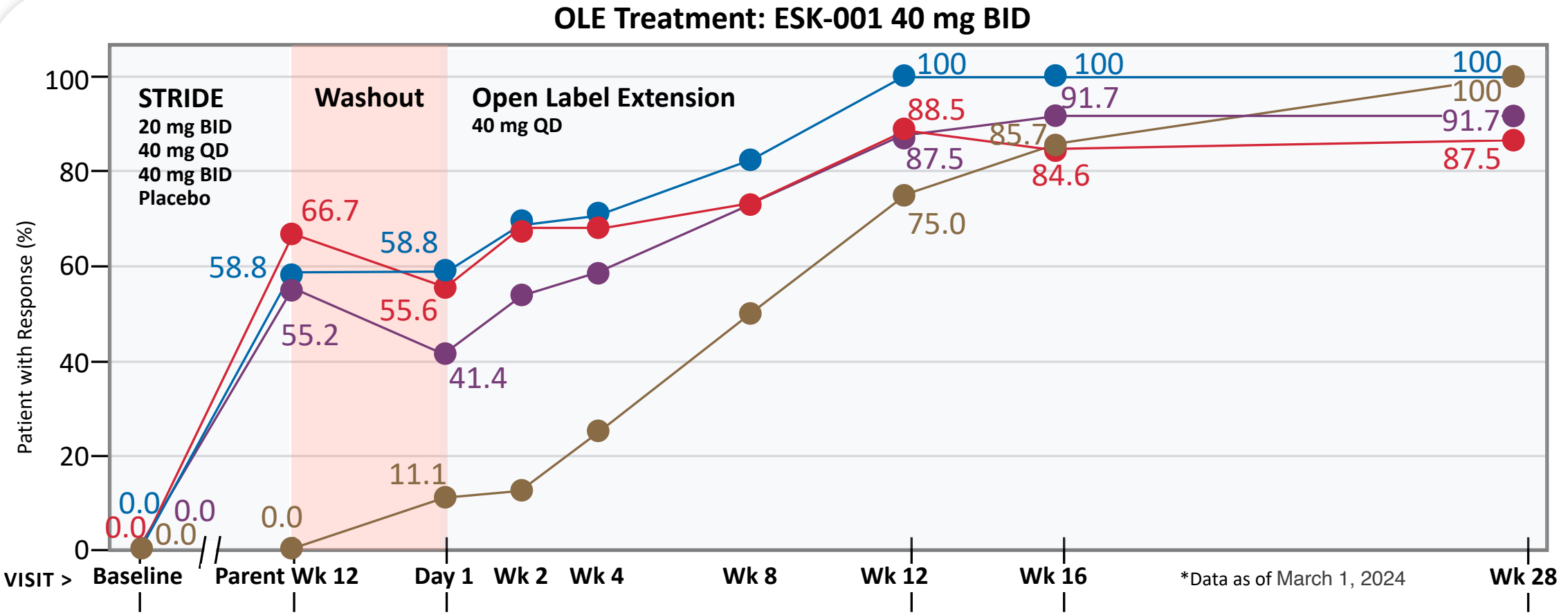
PASI-75 Over Time for OLE 40 mg BID Cohort by Parent Study Dose

ESK-001

Includes** Patients from STRIDE **Placebo, 20 mg BID, 40 mg QD and 40 mg BID Cohorts



NUMBER OF SUBJECTS							
40mg QD	17	17	17		16	15	16
20mg BID	29	29	29		24	24	24
40mg BID	27	27	27		26	26	24
Placebo	9	9	9		8	7	7

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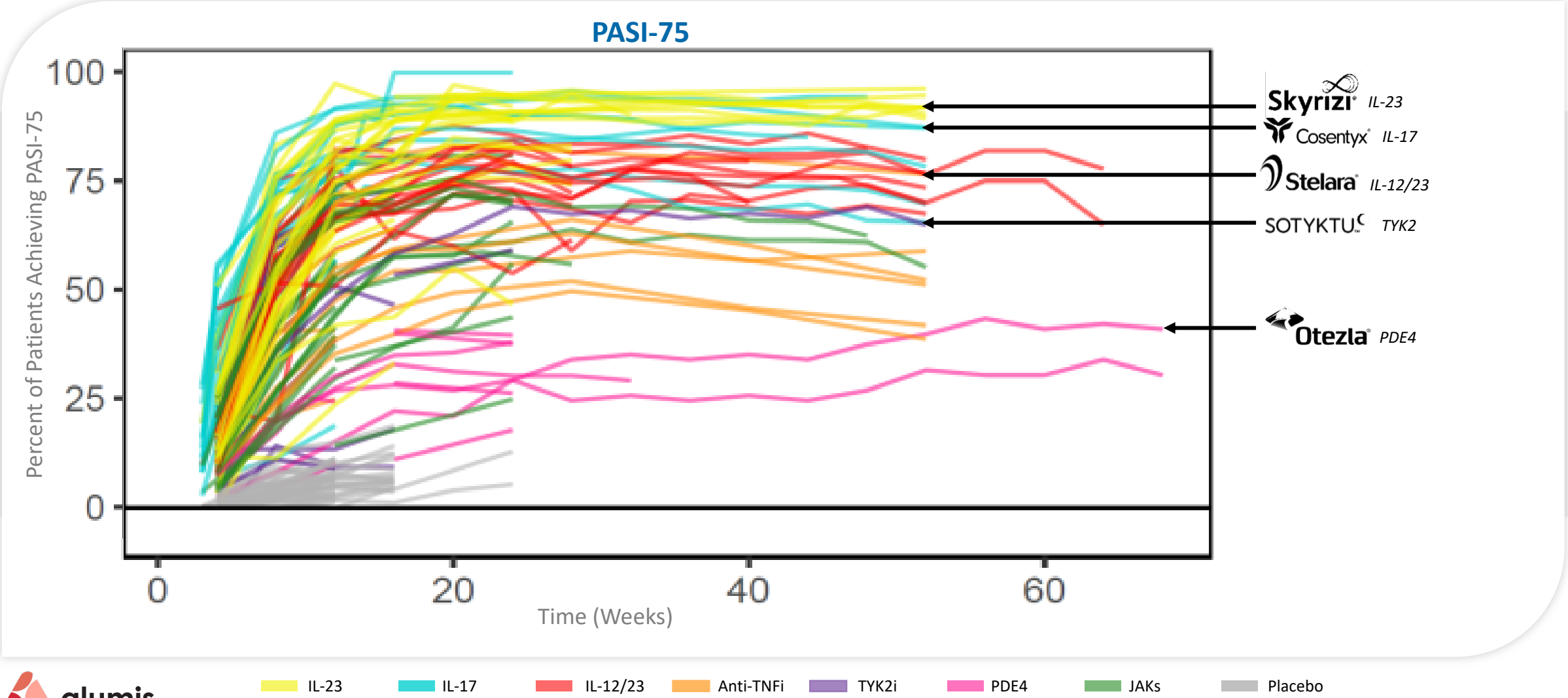
ESK-001 40mg QD ESK-001 20mg BID ESK-001 40mg BID Placebo

PASI-75 Outcomes for Recent Psoriasis Studies (69 studies, 11 molecules)

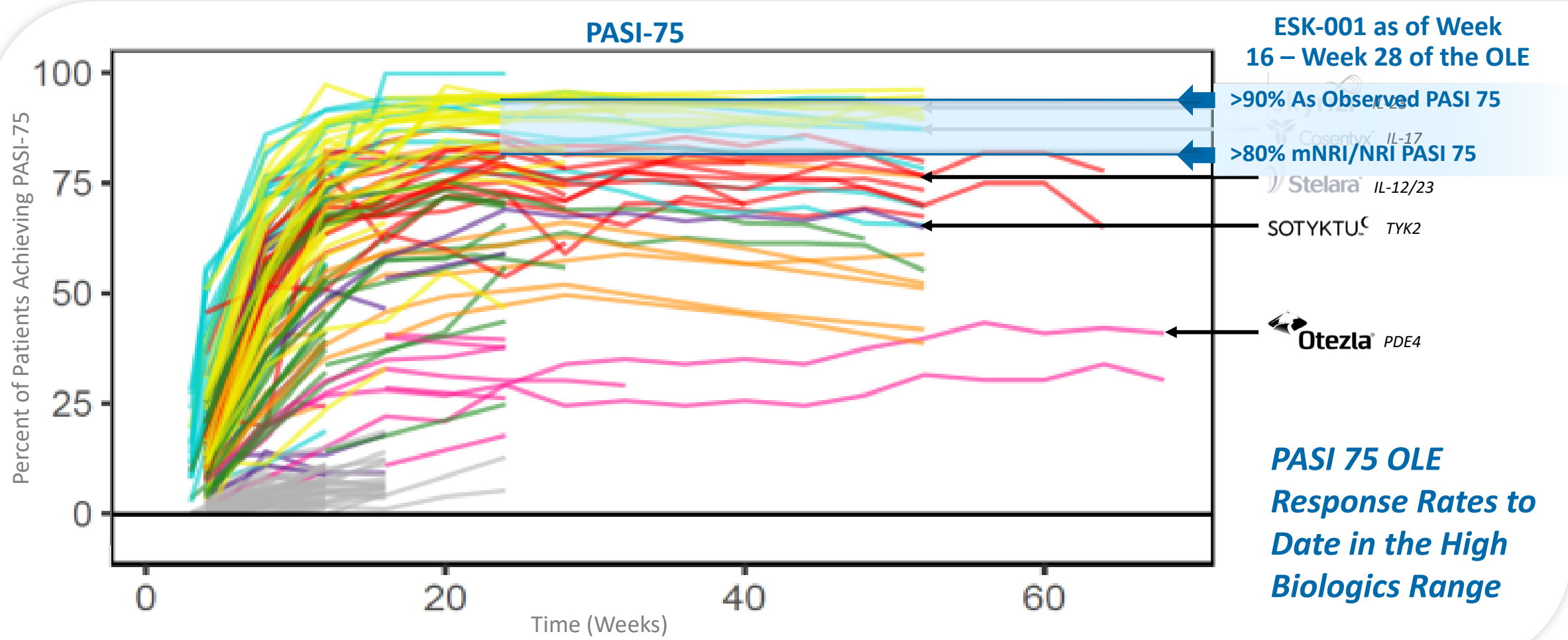
Maximal Response Is Achieved at Week 24 and Beyond



These data are not based on head-to-head or comparator studies. Differences exist between study designs and subject characteristics, and caution should be exercised when comparing data across studies.

PASI-75 Outcomes for Recent Psoriasis Studies (69 studies, 11 molecules)

Maximal Response Is Achieved at Week 24 and Beyond

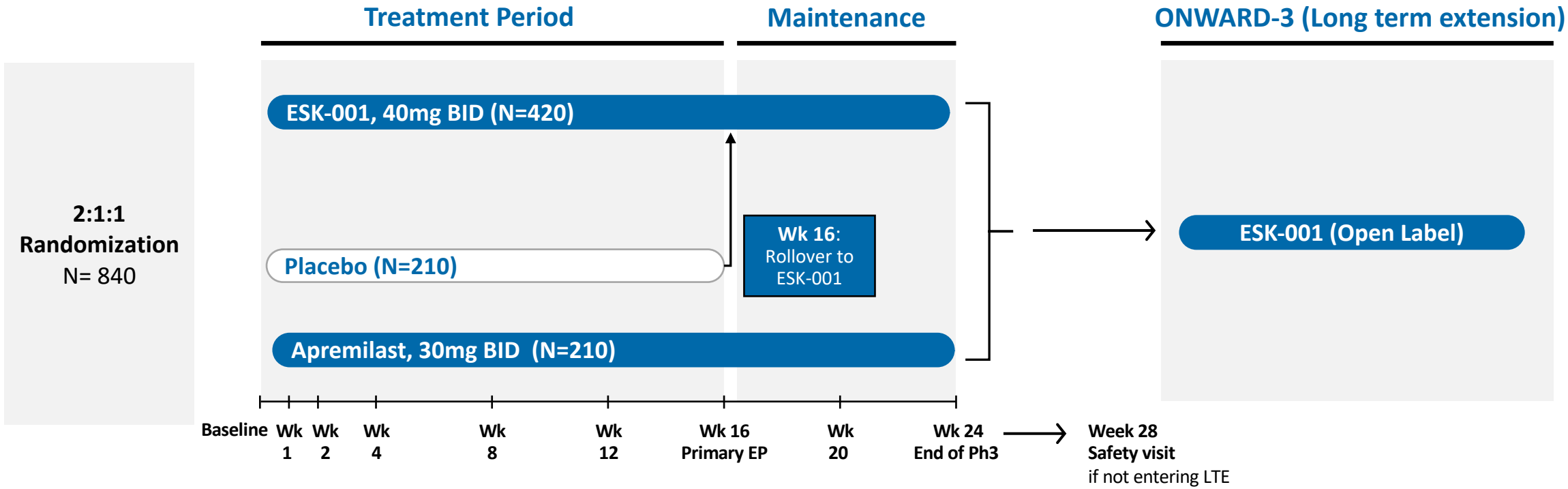


PASI 75 OLE Response Rates to Date in the High Biologics Range

These data are not based on head-to-head or comparator studies. Differences exists between study designs and subject characteristics and caution should be exercised when comparing data across studies.

ESK-001 Psoriasis Phase 3 ONWARD Program

Three Studies: Two parallel Phase 3 studies and a long term extension (LTE) study



ESK-001-016 (ONWARD1) & ESK-001-017 (ONWARD2):

- 24-week duration, apremilast active comparator

ESK-001-018 (ONWARD3)

- Long term extension (LTE) study, includes treatment withdrawal period

- In parallel, Alumis is developing a once-a-day modified release formulation for ESK-001

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Accelerated Phase 3 Plan Designed to Enable Speed to Market Without Compromising Essential Label Elements at Launch

	alumis ESK-001 24-Wk Ph3's Plus LTE	Bristol Myers Squibb™ Sotyktu Ph3	Takeda TAK-279* Current Ph3 Trials	Johnson&Johnson JNJ-2113* Current Ph3 Trials
16-wk Efficacy & Safety vs. Pb0 1° Endpoint	✓	✓	✓	✓
Efficacy & Safety vs. Comparator	Otezla ✓	Otezla ✓	Otezla ✓	Sotyku ✓
24 & 52-wk Efficacy & Safety	Via Ph3 LTE ✓	Via Ph3 ✓	Via Ph3 ✓	Via Ph3 ✓
Treatment Durability (Descriptive)	Ph3 LTE ✓	Ph3 pivotal ✓	Ph3 pivotal ✓	Ph3 pivotal ✓
2-Year Efficacy & Safety	Via Ph2 OLE & Ph3 LTE ✓	Via Ph3 ✓	Via Ph3 ✓	Via Ph3 ✓
3-Year Efficacy & Safety	Via Ph2 OLE ✓	Not in NDA ✗	Not in NDA ✗	Not in NDA ✗

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* Label expectations based on assumptions of clinical data that will be available at time of NDA submission based on published clinical trial plans.

SLE: ESK-001's Potential Ability to Maximally Inhibit Type I Interferon Offers Promise as an Oral Treatment Option for SLE

SYSTEMIC LUPUS ERYTHEMATOSUS

~3.4M

PATIENTS WORLDWIDE[1]

$4B+

GLOBAL MARKET[3]

- **>240K people have SLE in the US**, 68% with moderate-to-severe disease[2]
- **Strong unmet need persists in the SLE treatment space**, with only two approved treatments available; biologics are effective in a subset of patients
- **Opportunity to expand into lupus nephritis and cutaneous lupus erythematosus (CLE)**

1. Current patient estimates from Tian J, Zhang D, Yao X, Huang Y, Lu Q. Global epidemiology of systemic lupus erythematosus: a comprehensive systematic analysis and modelling study. Ann Rheum Dis. 2023 Mar;82(3):351-356. doi: 10.1136/ard-2022-223035. Epub 2022 Oct 14. PMID: 36241363; PMCID: PMC9933169.
2. Current patient estimate per GlobalData report
3. 2030 estimates from GlobalData report

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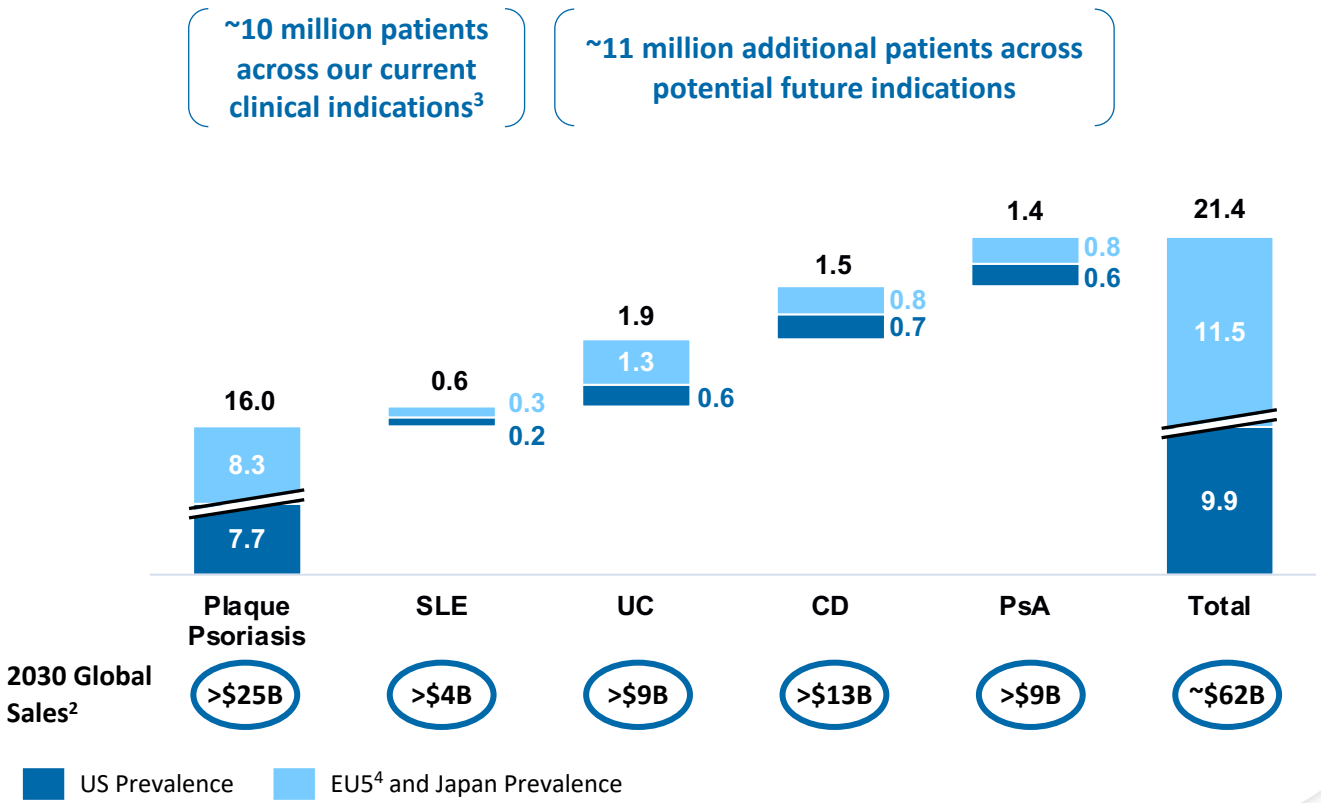
Ongoing Phase 2b SLE Program (LUMUS) Is Designed for Speed to Market and Probability of Clinical Success

- **Multiple points of validation for TYK2 and associated pathways in SLE**
  - Strong genetic rationale from P1104A loss of function mutation
  - Strong scientific rationale for inhibition of Type I Interferon pre-clinically and from Saphnelo®
  - Positive Phase 2 results from competitive TYK2 molecule
- **Saphnelo® data supports the need for maximal Type I IFN inhibition to achieve optimal patient benefit**
- **Ongoing Global Phase 2b LUMUS trial, expected topline readout in 2026**
  - Designed as pivotal trial
  - Primary endpoint BICLA at week 48, target enrollment: n=388 patients
  - Includes OLE for faster enrollment and building of safety database
  - Operationally designed to minimize placebo effect
- **Potential for accelerated regulatory pathway with one additional Phase 3 trial**

ESK-001 Is Designed to Unlock Multi-Billion Dollar Markets With High Unmet Need

Cumulative Prevalence Across Our Potential Indications[1] (M)

(Million patients)



- **~21 million cumulative prevalence** across our current and considered indications, representing an estimated **~$62Bn cumulative annual market** by 2030
- Large indications dominated by injectable biologics or sub-effective orals, driving **high demand for safe and effective oral agents**
- **Strong rationale for TYK2** across all these indications

1. Current estimates from GlobalData reports
2. 2030 estimates from GlobalData reports
3. Includes moderate-to-severe psoriasis and SLE
4. Includes France, Germany, Italy, Spain and the United Kingdom

ESK-001 Profile Creates Significant Opportunity to Address Additional Indications

TYK2 Class Has Extensive Validation with Substantial Market Potential Across Immune-mediated Diseases

Indication	Market Size[1]	Clinical POC	Ongoing Trial	Genetic Evidence	Biologic Rationale
Plaque Psoriasis	>$25B	✓	✓	✓	✓
Psoriatic Arthritis	>$9B	✓	✓	✓	✓
Systemic Lupus	>$4B	✓	✓	✓	✓
Ulcerative Colitis	>$9B		✓	✓	✓
Crohn's Disease	>$13B		✓	✓	✓
Alopecia Areata	>$1.7B		✓	✓	✓
Cutaneous Lupus	>$2B		✓	✓	✓
Ankylosing Spondylitis	>$6B			✓	✓
Multiple Sclerosis	>$30B			✓	✓
Rheumatoid Arthritis	>$33B			✓	✓
Juvenile RA	>$8B			✓	✓
Others	>$20B		✓	✓	✓
Market Size Total	**>$160B**				

Publicly disclosed indications for TYK2, Market size estimates for 2030 worldwide

1. GlobalData, market research reports

ESK-001: Potent, Highly Selective Allosteric TYK2 Inhibitor with Differentiated Clinical Profile

Potentially Best-in-Class Efficacy and Safety Profile for Moderate-to-Severe Plaque Psoriasis

- Well-behaved molecule achieves maximal target inhibition
- Maximal TYK2 inhibition results in high biologic-like efficacy with PASI 75 up to 90% and PASI 90 up to 70%
- Favorable risk-benefit profile to date

Effect of target inhibition on efficacy is significant

- Incomplete target inhibition results in sub-optimal efficacy
- Underscores importance of potent and sustained target inhibition

Significant opportunity in psoriasis and additional immune-mediated diseases

- Ongoing clinical trial in SLE and broad opportunities in additional immune-mediated diseases
- Combination potential in immunology

A-005: Our CNS Penetrant Allosteric TYK2 Inhibitor

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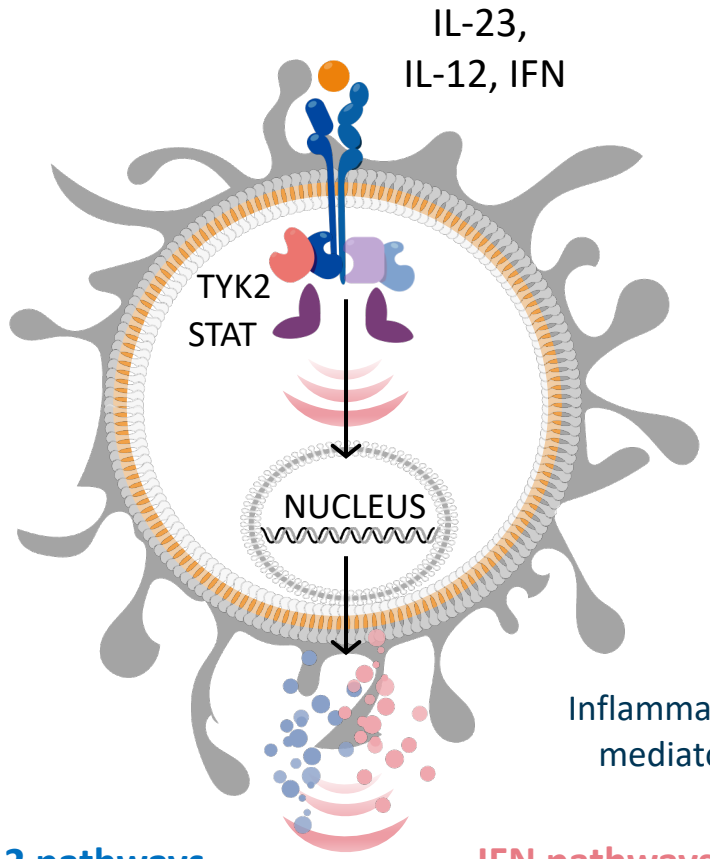
Inhibition of TYK2 Provides Potential for Immunomodulation in Neuroinflammatory and Neurodegenerative Diseases

Targeting TYK2 in CNS Disorders



- Strong biological rationale for the involvement of TYK2 in neuroinflammatory and neurodegenerative diseases .
- Genome-wide association studies have shown the loss-of function TYK2 genetic variant, P1104A, has a protective effect for the development of MS.
- TYK2 is known to be expressed and functionally active in CNS-resident microglia. TYK2 pathway cytokines are active in CNS resident immune cells.

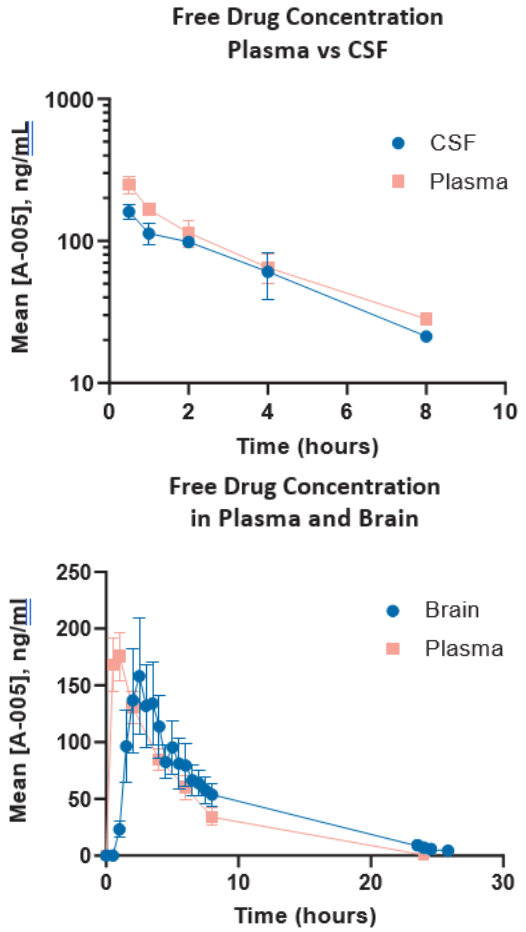
TYK2 inhibition has potential utility in various neuroinflammatory and neurodegenerative diseases

Multiple Sclerosis	Alzheimer's Disease
Parkinson's Disease	Neuroinflammation

A-005 Is a Potential First-in-Class, CNS-Penetrant, Allosteric TYK2 Inhibitor for Neuro-Inflammation

- Highly potent and intrinsically selective for TYK2 with no off-target JAK pharmacology
- Inhibits human whole blood and microglial activation
- A-005 achieves ~1:1 ratio CNS penetration *in vivo*
- Projected low QD dose with ~12h projected half-life
- Phase 1 initiated, with MS Phase 2 as fast-to-POC for neuro-inflammation

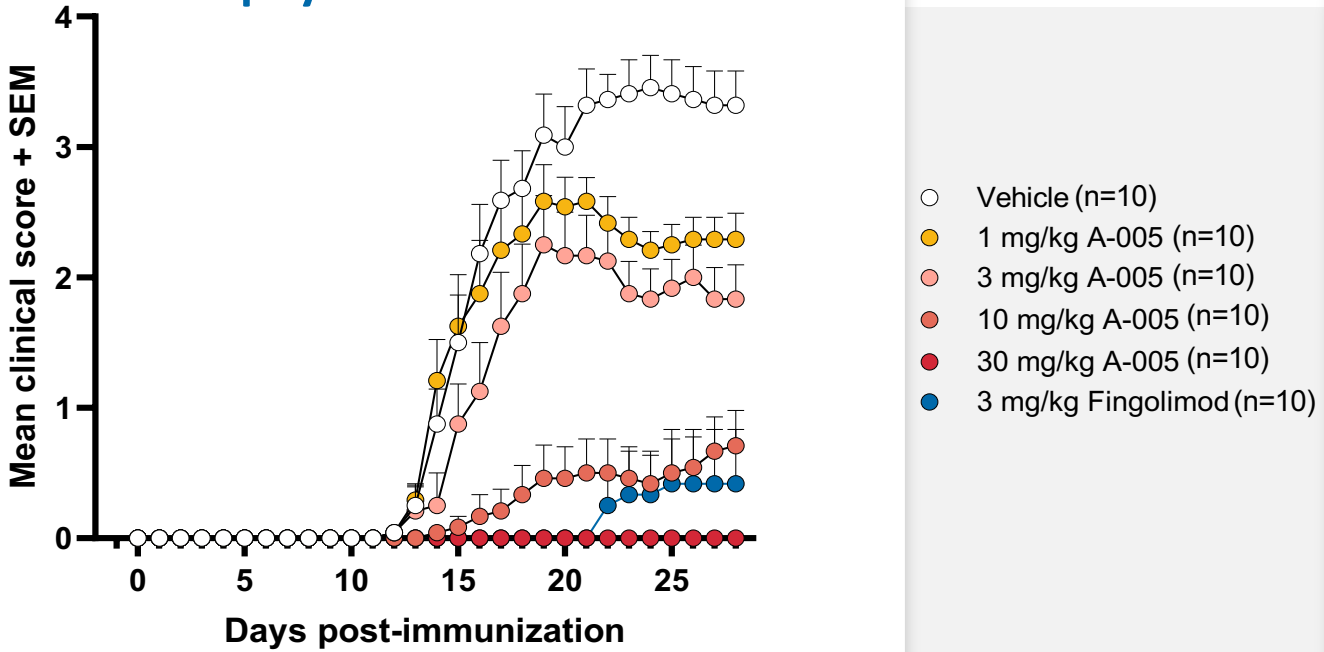
High CNS Penetration Confirmed With Oral Dosing of A-005 *In Vivo*



A-005 Achieves Significant Dose-Dependent Response Preclinically

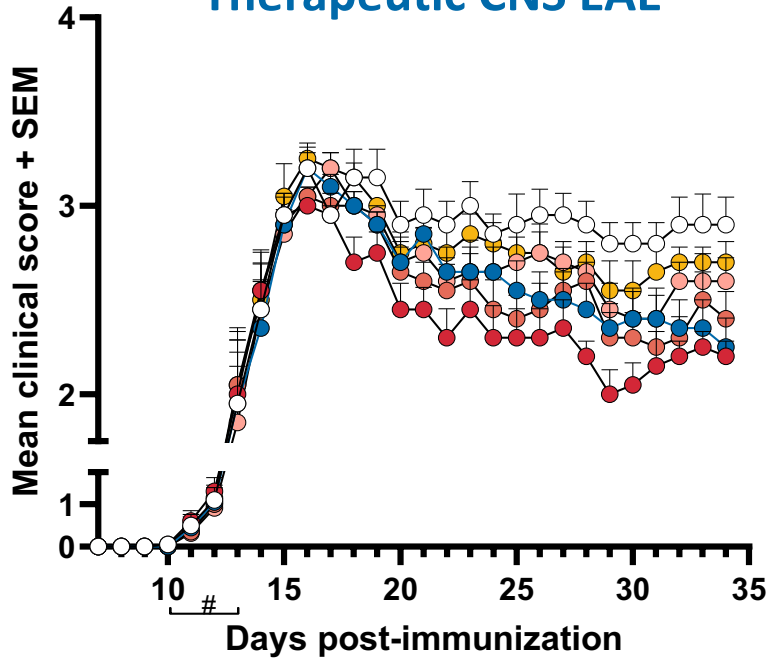
In Both Prophylactic and Therapeutic EAE Models with Once Daily Oral Dosing

Prophylactic CNS EAE



Once daily oral dosing initiated 1 day prior to EAE induction; Mann-Whitney U test vs vehicle, p<0.05 for 1 mg/kg (day 20-28), 3 mg/kg (day 20-28), 10 mg/kg (day 14-28), 30 mg/kg (day 14-28)

Therapeutic CNS EAE



Once daily oral dosing initiated the onset of EAE clinical signs (# enrollment period, days 10-13). Experience continued until each mouse had been dosed for at least 21 days (day 34). Mann-Whitney U-test vs vehicle, p<0.05 for 10 mg/kg (day 21, 25-27, 29-32, 34), 30 mg/kg (day 18-34)

- **Complete suppression** of EAE achieved in prophylactic EAE model, and significantly effective in a therapeutic EAE model
- A-005 **recapitulates** TYK2 human loss of function variant knock-in mouse EAE data

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(Gorman, J. A., Hundhausen, C., Kinsman, M., Arkatkar, T., Allenspach, E. J., Clough, C., West, S. E., Thomas, K., Eken, A., Khim, S., Hale, M., Oukka, M., Jackson, S. W., Cerosaletti, K., Buckner, J. H., & Rawlings, D. J. (2019). The TYK2-P1104A Autoimmune Protective Variant Limits Coordinate Signals Required to Generate Specialized T Cell Subsets. Frontiers in immunology, 10, 44. https://doi.org/10.3389/fimmu.2019.00044)

Clinical Development Strategy for A-005

Goal: Establish Clinical Proof-of-Concept in First Neuroinflammatory Indication by 2026

Ongoing Phase 1 Trial in Healthy Volunteers

- Trial initiated in April 2024, readout expected by year-end 2024
- Assess the safety, PK, and PD of single ascending doses (SAD) and multiple ascending doses (MAD) of orally-administered A-005 in healthy volunteers
- Longer term pre-clinical toxicology program ongoing

Future Clinical Development

- Initial development in Multiple Sclerosis
- Phase 2 study in MS patients currently expected to be initiated in 2025 with readout in 2026
- Potential expansion into neurodegenerative diseases

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Conclusion

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Anticipated Value-Creating Near-Term Catalysts

2024

MAR24 ESK-001 – Present Phase 2 STRIDE and OLE Data at AAD ✓

APR24 A-005 – Phase 1 Initiation ✓

JUL24 ESK-001 – Initiate Phase 3 in PsO ✓

3Q24 ESK-001 – PsO OLE Data Update

YE24 A-005 – Phase 1 Data

2025

2025 A-005 – MS Phase 2 Initiation

2025 IND Filing for 3rd Program

2025 ESK-001 – PsO OLE Data Update

2026

2026 ESK-001 – PsO Phase 3 Topline Data

2026 ESK-001 – SLE Phase 2b Topline Data

2026 A-005 – MS Phase 2 Topline Data

Experienced Team & Strong Financial Position to Execute on Milestones

Management Team

Martin Babler
President and CEO, Chairman

Mark Bradley
Chief Development Officer

Jörn Drappa
Chief Medical Officer

David Goldstein
Chief Scientific Officer

Roy Hardiman
Chief Business and Legal Officer

John Schroer
Chief Financial Officer

Sara Klein
General Counsel

Derrick Richardson
Senior Vice President of People and Culture

Board of Directors

Martin Babler
President and CEO, Chairman

Alan Colowick, M.D., M.P.H.
Managing Member
AyurMaya, an affiliate of Matrix Capital Management

Sapna Srivastava, Ph.D.
Independent Board Member

Zhengbin (Bing) Yao, Ph.D.
CEO
ArriVent Biopharma

Srinivas Akkaraju, M.D., Ph.D.
Founder and Managing General Partner
Samsara BioCapital

Patrick Machado, J.D.
Independent Board Member

Jim Tananbaum, M.D.
Founder and CEO
Foresite Capital

Cash and cash equivalents and marketable securities
Expected to fund operations into 2026

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Developing Oral Therapies To Transform Lives of Patients With Immune-Mediated Diseases

ESK-001

ESK-001, potentially the first and only allosteric TYK2 inhibitor well-tolerated at maximal target inhibition

- Demonstrated maximal target inhibition, potential best-in-class tolerability profile in plaque psoriasis (PsO)
- Multibillion dollar market opportunity[1] in a broad set of indications, including systemic lupus erythematosus (SLE)
- Ongoing ONWARD Phase 3 clinical trials in PsO and LUMUS Phase 2b trial in SLE

A-005

A-005, a Phase 1 CNS penetrant allosteric TYK2 inhibitor for the treatment of neuroinflammatory and neurodegenerative diseases

- Potential first- and best-in-class opportunity with blood-to-brain ratio of approximately 1:1

APPROACH

Precision approach to replace broad immuno-suppression with targeted therapies

- Precision data analytics platform generating genetic, genomic, proteomic, and biological and clinical disease insights
- Accelerate research and development and increase the probability of clinical success

CATALYSTS

Anticipated value-creating near-term catalysts

- Multiple milestones in 2024 / 2025, including readout for A-005 Phase 1 trial in healthy volunteers
- Catalyst rich 2026 with topline data for Phase 3 trials in PsO, Phase 2b trial in SLE and Phase 2 trial in multiple sclerosis (MS)

LEADERSHIP

Experienced team with strong track record in value creation

- Strong financial position and backed by established blue-chip life science investors

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1. Based on current internal estimates

Thank you!

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