South Beach Symposium
medical + aesthetic dermatology
CELEBRATING 20 YEARS
OF PREMIER MEDICAL & AESTHETIC DERMATOLOGY EDUCATION

REIMAGINING
MEDICAL AND
AESTHETIC
DERMATOLOGY

New Targets for Treatment of Psoriasis

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DermResearch, PLLC, Louisville, KY
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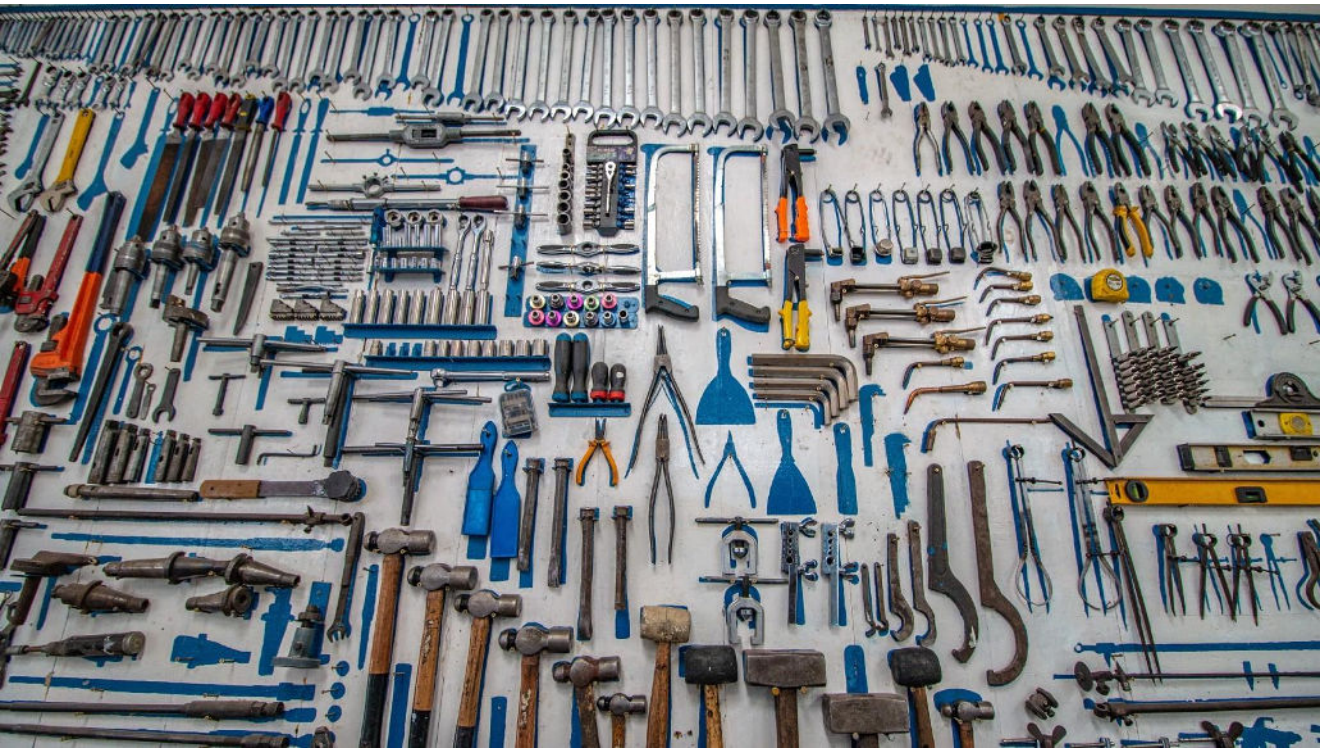
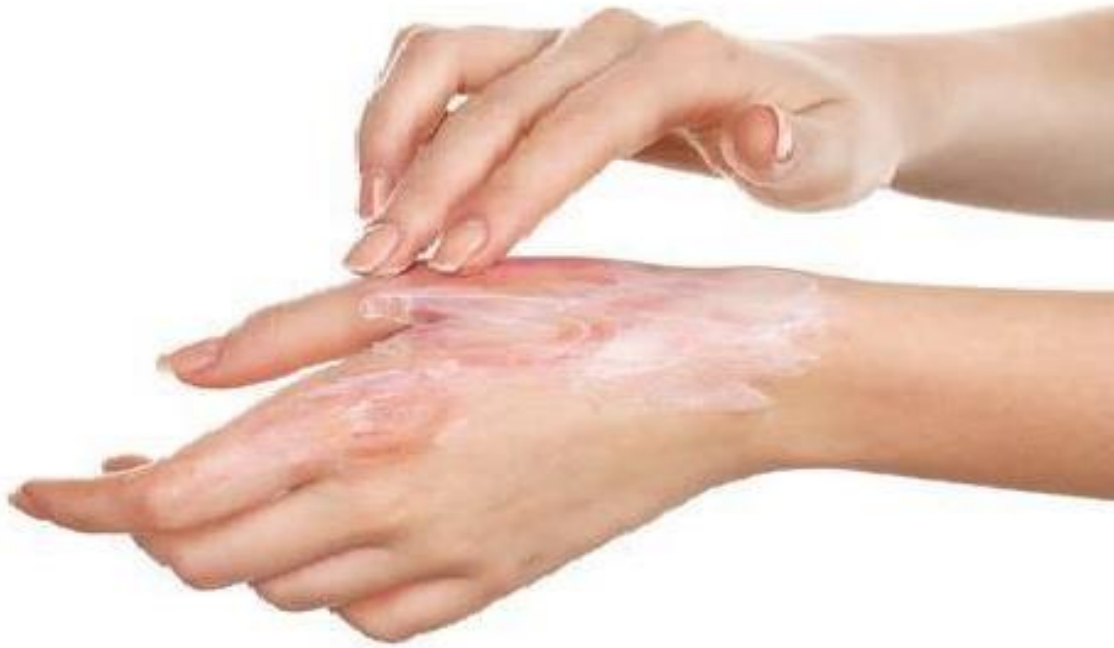
On the horizon…

EMERGING THERAPIES

PSORIASIS

TOPICAL TREATMENT IS THE FOUNDATION OF DERMATOLOGIC THERAPY





WHAT DO I DO?

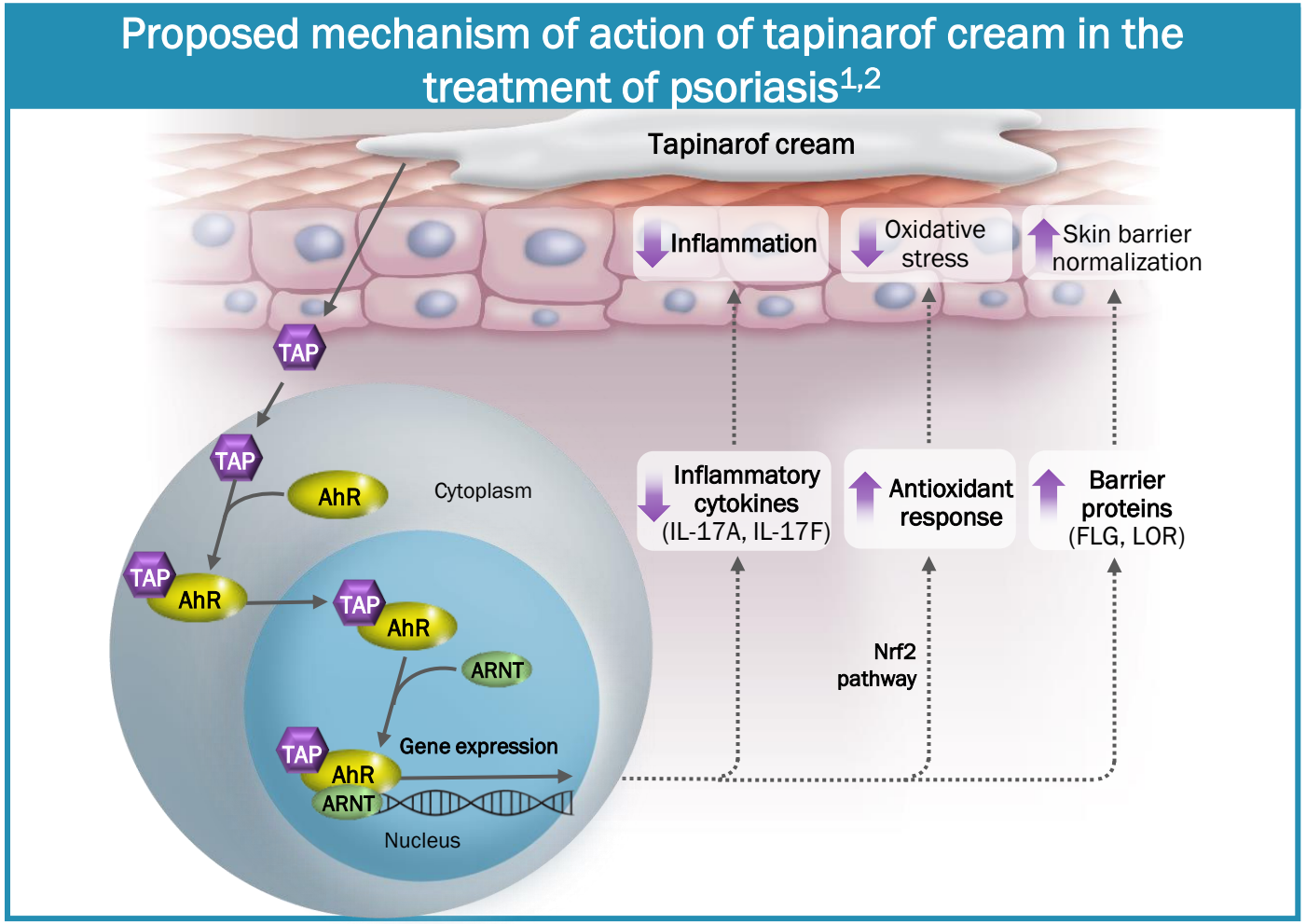

BIOLOGICS TOPICALS
SYSTEMIC TOPICALS
LIGHT TOPICALS
TOPICALS

I DON’T WANT TO BE ON STEROIDS

UNMET NEED

NEW CHEMICAL ENTITIES

Tapinarof is a First-in-Class, Topical Therapeutic Aryl Hydrocarbon Receptor Modulating Agent (TAMA)



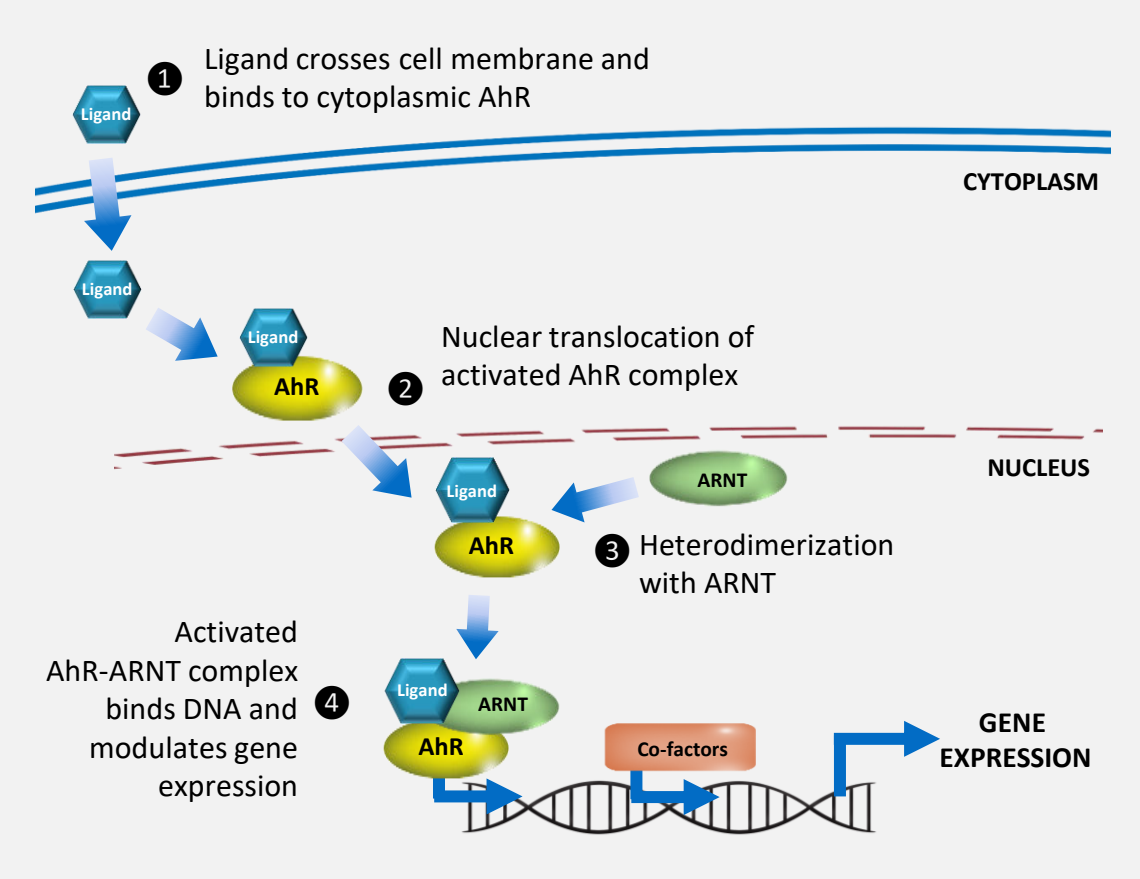
AhR, aryl hydrocarbon receptor; ARNT, aryl hydrocarbon receptor nuclear translocator; FLG, filaggrin; IL, interleukin; LOR, loricrin; Nrf2, nuclear factor erythroid 2-related factor 2; TAP, tapinarof.

1. Smith SH, et al. *J Inv Dermatol.* 2017;137:2110–2119; 2. Furue M, et al. *J Dermatological Sci.* 2015;80:83–88.

Tapinarof: Therapeutic AhR Modulating Agent (TAMA)

- Tapinarof is a topical, small molecule TAMA that directly binds to and activates AhR transcription factor[1]
- AhR activation via tapinarof *in vitro* and animal models leads to:
  - Reduction of Th17 cytokine expression[1]
  - Reduction of Th2 cytokine expression[1,2]
  - Decreased oxidative stress[1]
  - Increased skin barrier proteins[1]

AhR pathway[3]



AhR, aryl hydrocarbon receptor; ARNT, aryl hydrocarbon receptor nuclear translocator; TAMA, therapeutic aryl hydrocarbon receptor modulating agent; Th, T helper cell.

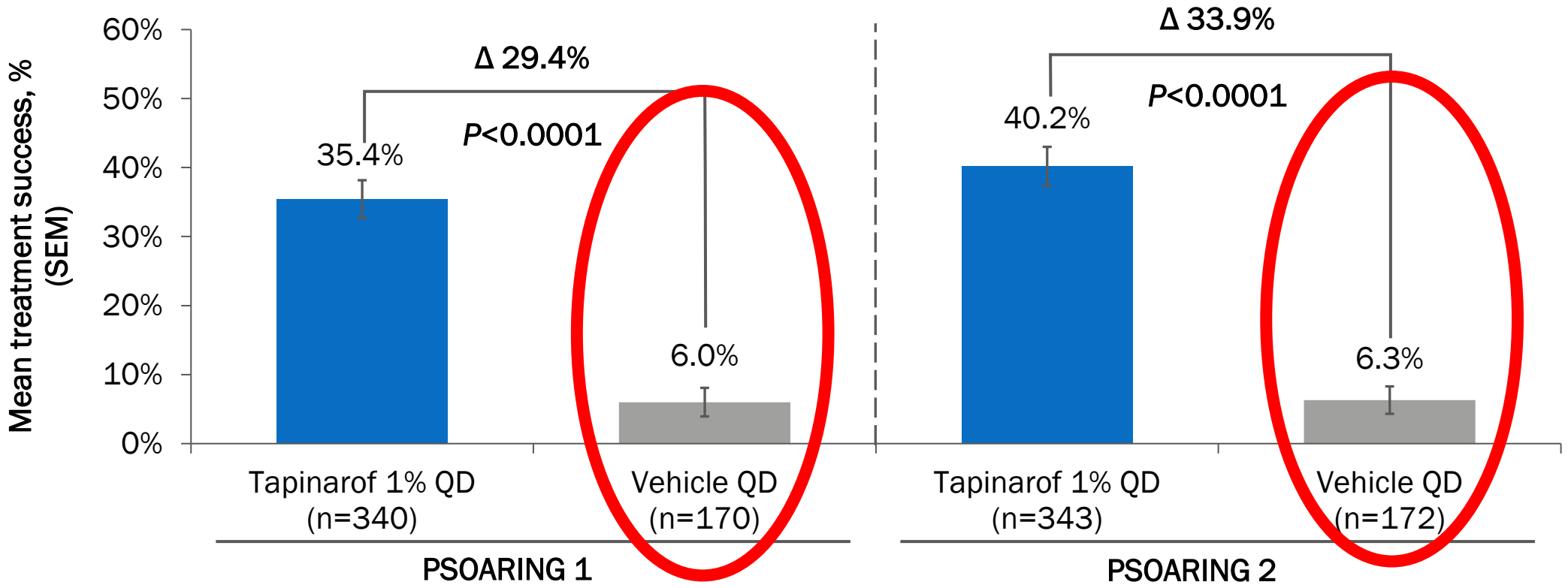
1. Smith SH et al. *J Inv Dermatol* 2017;137:2110–2119; 2. Negishi T et al. *J Immunol.*2005;175;7348–7356; 3. Furue M et al. *J Dermatological Sci*. 2015;80:83–88.

Tapinarof Cream 1% QD for the Treatment of Plaque Psoriasis: Efficacy and Safety in Two Pivotal Phase 3 Trials

Mark Lebwohl,[1] Linda Stein Gold,[2] Bruce Strober,[3] Kim Papp,[4] April Armstrong,[5] Jerry Bagel,[6] Leon Kircik,[1,7] Benjamin Ehst,[8] H Chih-ho Hong,[9] Jennifer Soung,[10] Jeff Fromowitz,[11] Scott Guenthner,[12] Stephen C Piscitelli,[13] David S Rubenstein,[13] Philip M Brown,[13] Anna M Tallman,[13] Robert Bissonnette[14]

[1]Icahn School of Medicine, Mount Sinai, New York, NY, USA; [2]Henry Ford Health System, Detroit, MI, USA; [3]Yale University, New Haven and Central Connecticut Dermatology Research, Cromwell, CT, USA; [4]Probity Medical Research, Waterloo, ON, Canada; [5]Keck School of Medicine University of Southern California, Los Angeles, CA, USA; [6]Psoriasis Treatment Center of Central New Jersey, NJ, USA; [7]Skin Sciences, PLLC, Louisville, KY, USA; [8]Oregon Medical Research Center, Portland, OR, USA; [9]University of British Columbia and Probity Medical Research, Surrey, BC, Canada; [10]Southern California Dermatology, Santa Ana, CA, USA; [11]Dermatology of Boca, Boca Raton, FL, USA; [12]The Indiana Clinical Trials Center, PC, Plainfield, IN, USA; [13]Dermavant Sciences, Inc., Durham, NC, USA; [14]Innovaderm Research Inc., Montreal, QC, Canada

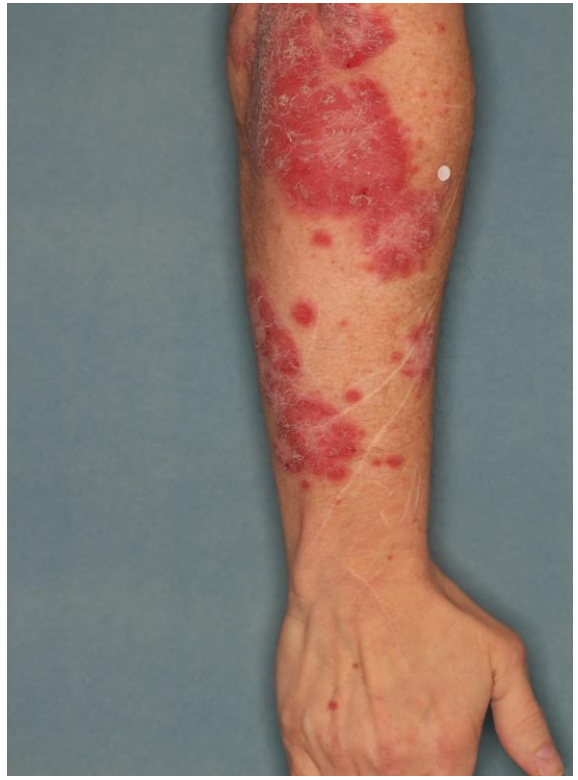
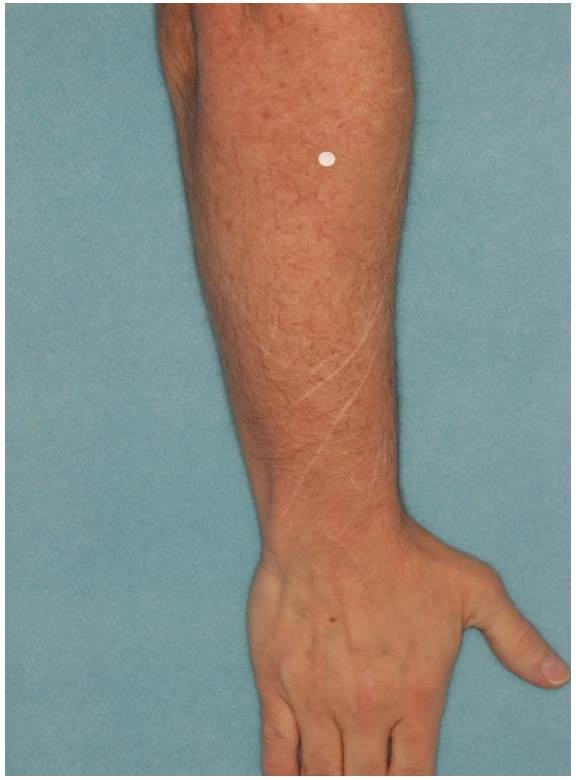
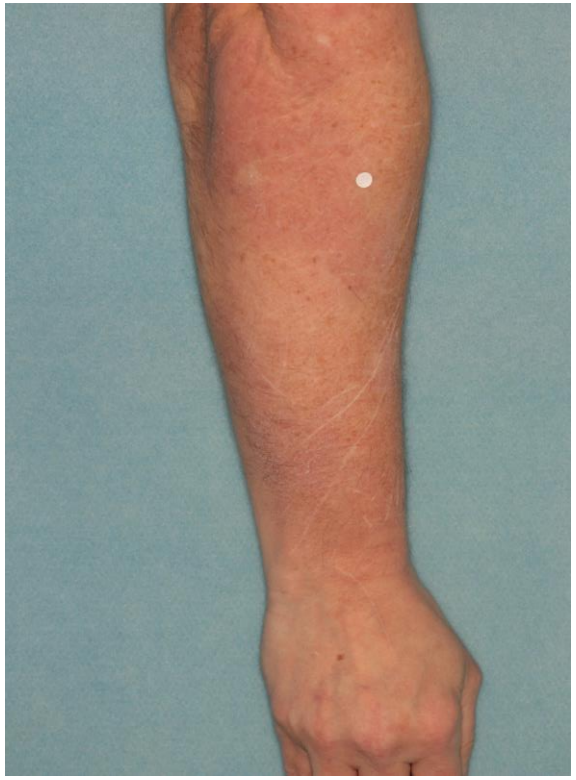
Tapinarof 1% QD: Primary Endpoint of PGA Response at Week 12 was Achieved in Both Studies



PGA response rate* was highly statistically significant in the tapinarof cream 1% QD group versus vehicle in both PSOARING 1 and 2: 35.4% vs 6.0% (*P*<0.0001) and 40.2% vs 6.3% (*P*<0.0001), respectively

*PGA of 0 or 1 and ≥2-grade improvement at Week 12.
ITT population. *P* value based upon Cochran-Mantel-Haenszel analysis stratified by baseline PGA score.
ITT, intent-to-treat; PGA, Physician Global Assessment; QD, once daily; SEM, standard error of mean

Tapinarof 1% QD Clinical Response of Patient with Plaque Psoriasis who Achieved Primary and Secondary Efficacy Endpoints at Week 12

Baseline	Week 4	Week 12
		
• PGA = 3 • PASI = 17.6	• PGA = 2 • PASI = 4	• PGA = 0 • PASI = 0

PGA and PASI are global efficacy assessments. Example of one representative target lesion of a patient treated with tapinarof 1% QD; individual results may vary. Photographs demonstrate improvement in PGA and PASI at Week 4 and 12.
PASI, Psoriasis Area and Severity Index; PGA, Physician Global Assessment; QD, once daily.

Roflumilast Cream, a Once-Daily, Potent Phosphodiesterase-4 Inhibitor, in Chronic Plaque Psoriasis Patients: Efficacy and Safety From DERMIS-1 and DERMIS-2 Phase 3 Trials

- Mark Lebwohl,[1] Leon H. Kircik,[2] Angela Moore,[3] Linda Stein Gold,[4] Zoe D. Draelos,[5] Melinda J. Gooderham,[6] Kim A. Papp,[7] Jerry Bagel,[8] Neal Bhatia,[9] James Del Rosso,[10] Laura K. Ferris,[11] Lawrence J. Green,[12] Adelaide A. Hebert,[13] Terry Jones,[14] Steven E. Kempers,[15] David M. Pariser,[16] Paul S. Yamauchi,[17] Matthew Zirwas,[18] Patrick Burnett,[19] Robert C. Higham,[19] Lynn Navale,[19] David R. Berk[19]

- [1]Icahn School of Medicine at Mount Sinai, New York, NY, USA; [2]Icahn School of Medicine at Mount Sinai, New York, NY, Indiana Medical Center, Indianapolis, IN, Physicians Skin Care, PLLC, Louisville, KY, and Skin Sciences, PLLC, Louisville, KY, USA; [3]Arlington Research Center, Arlington, TX, USA, Baylor University Medical Center, Dallas, TX, USA; [4]Henry Ford Medical Center, Detroit, MI, USA; [5]Dermatology Consulting Services, High Point, NC, USA; [6]SkiN Centre for Dermatology, Probity Medical Research and Queen's University, Peterborough, ON, Canada; [7]Probity Medical Research and K Papp Clinical Research, Waterloo, ON, Canada; [8]Psoriasis Treatment Center of Central New Jersey, Windsor, NJ, USA; [9]Therapeutics Clinical Research, San Diego, CA, USA; [10]JDR Dermatology Research Center, LLC, Las Vegas, NV, USA; [11]University of Pittsburgh, Department of Dermatology, Pittsburgh, PA, USA; [12]George Washington University School of Medicine, Rockville, MD, USA; [13]UT Health McGovern Medical School, Houston, TX, USA; [14]U.S. Dermatology Partners Bryan, Bryan, TX, USA; [15]Minnesota Clinical Study Center, Fridley, MN, USA; [16]Eastern Virginia Medical School and Virginia Clinical Research, Inc., Norfolk, VA, USA; [17]David Geffen School of Medicine at UCLA, Los Angeles, and Dermatology Institute & Skin Care Center, Inc., Santa Monica, CA, USA; [18]Dermatologists of the Central States, Probity Medical Research, and Ohio University, Bexley, OH, USA; [19]Arcutis Biotherapeutics, Inc., Westlake Village, CA, USA

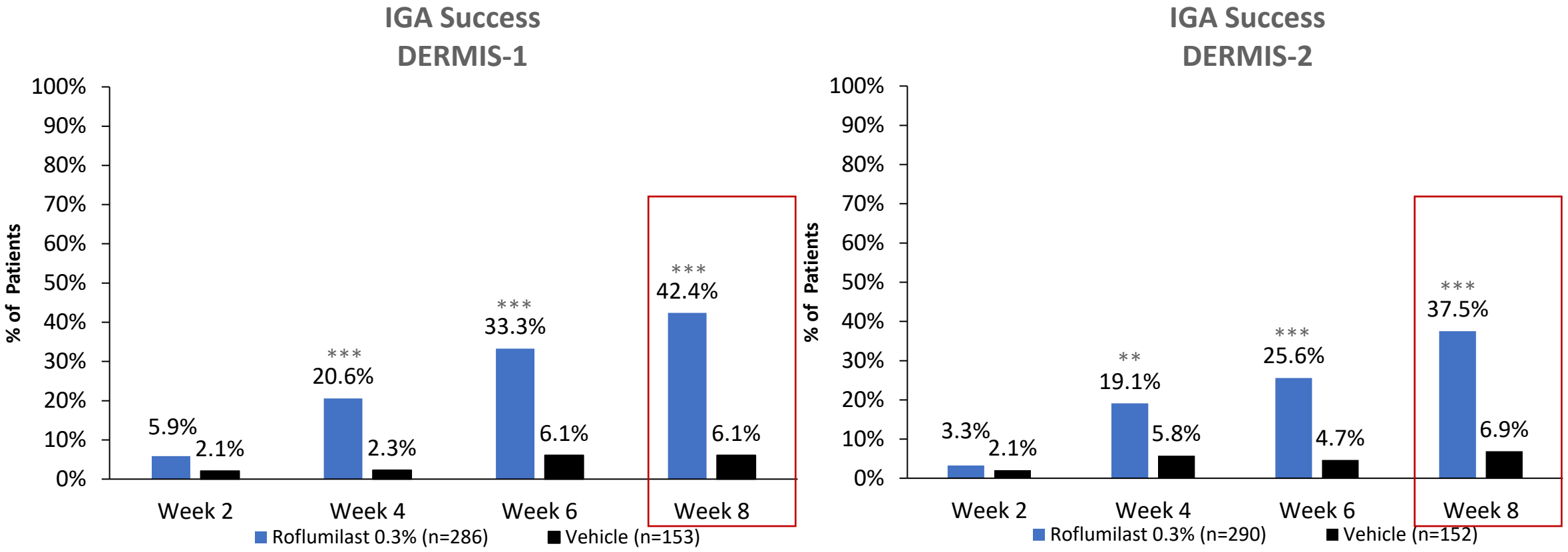
- Disclosures: Mark Lebwohl, Leon H. Kircik, Angela Moore, Linda Stein Gold, Zoe D. Draelos, Melinda J. Gooderham, Kim A. Papp, Jerry Bagel, Neal Bhatia, James Del Rosso, Laura K. Ferris, Lawrence J. Green, Adelaide A. Hebert, Terry Jones, Steven E. Kempers, David M. Pariser, Paul S. Yamauchi, and Matthew Zirwas are investigators and/or consultants for Arcutis Biotherapeutics, Inc. and received grants/research funding and/or honoraria; Robert C. Higham, Lynn Navale, and David R. Berk are employees of Arcutis Biotherapeutics, Inc. Additional disclosures provided on request.

- This work was supported by Arcutis Biotherapeutics, Inc.

- Writing support was provided by Christina McManus, PhD, Alligent Biopharm Consulting LLC, and funded by Arcutis Biotherapeutics, Inc.

Robust Efficacy on IGA Success in Both Phase 3 Studies

IGA Success = Clear or Almost Clear with at least a 2-grade improvement from baseline



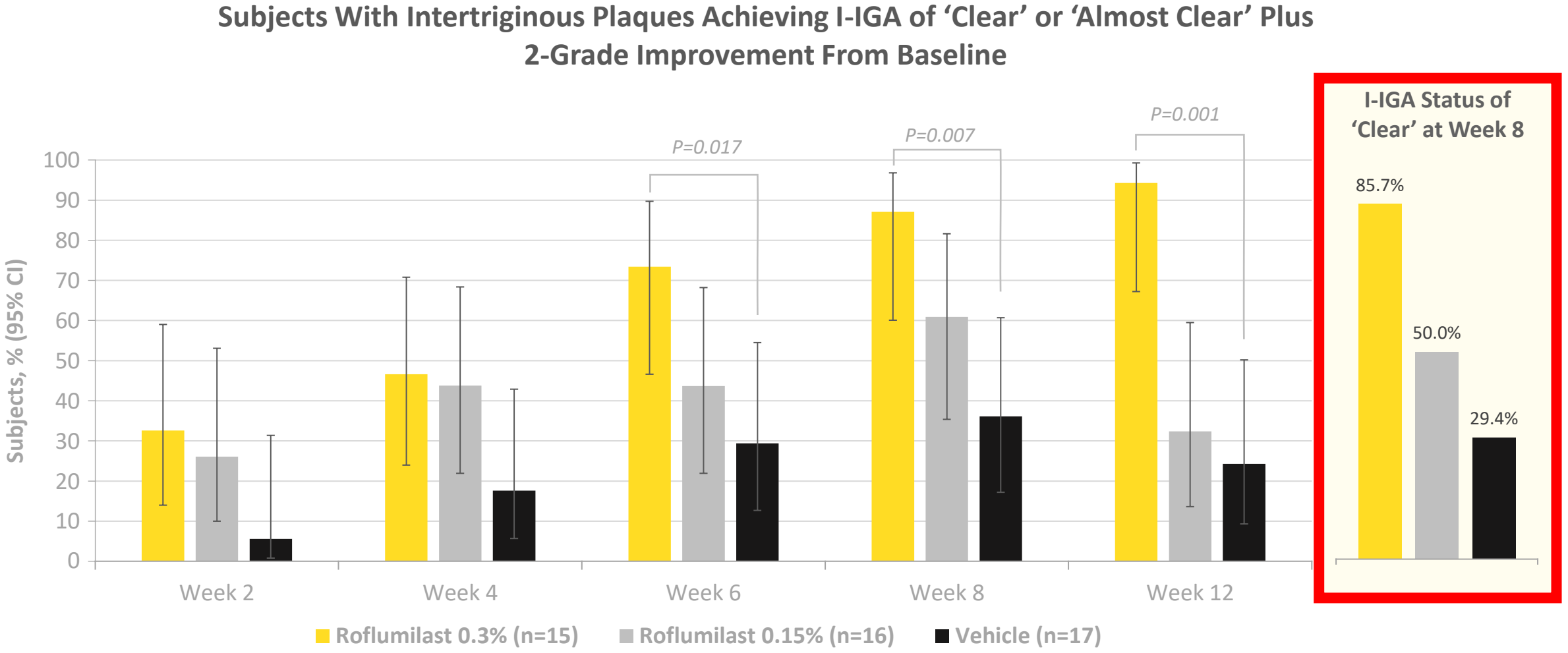
* p<0.05; ** p<0.01; *** p<0.001

The primary endpoint was achieved in both DERMIS-1 and DERMIS-2

Intent-to-treat population; missing scores imputed using multiple imputations

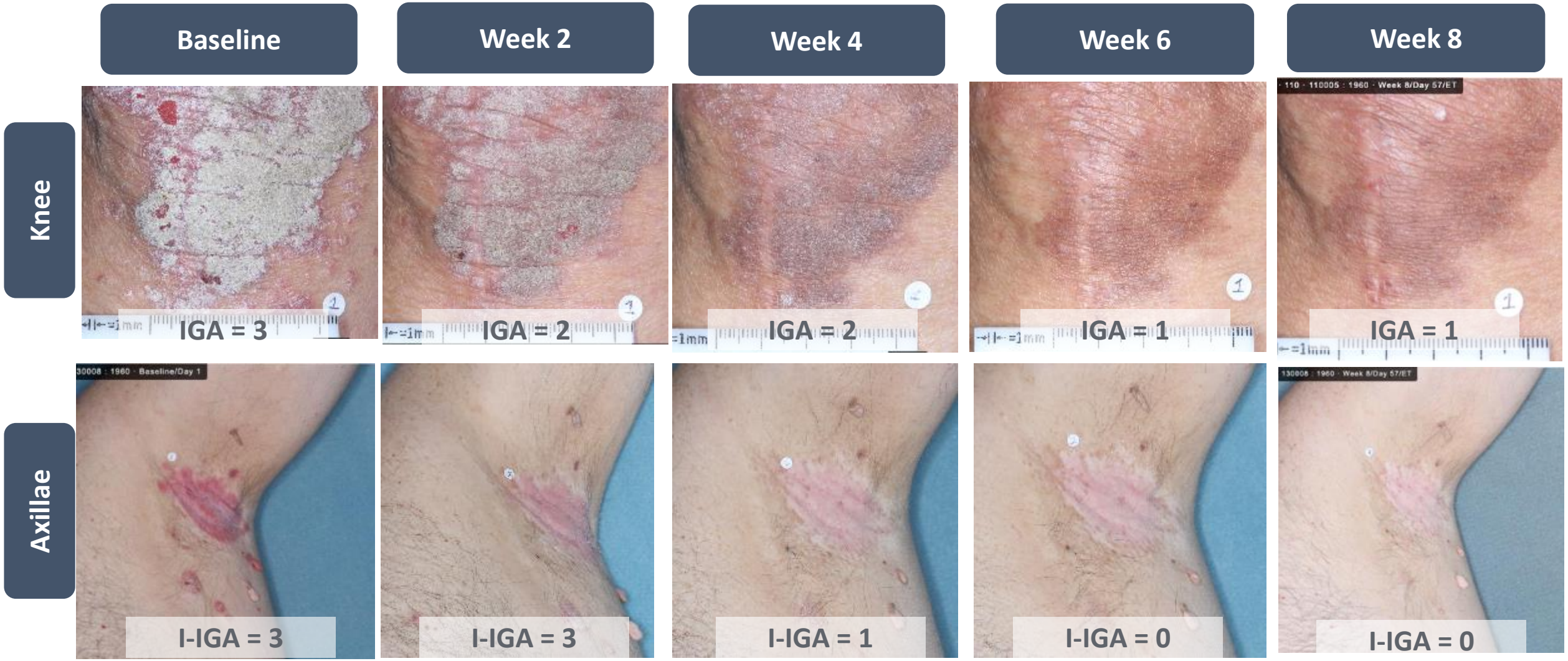
IGA: Investigator's Global Assessment

Most Subjects With Intertriginous Plaques Treated With Roflumilast Cream Achieved I-IGA Success by Week 6 With Continued Improvement Through Week 12



Data are presented for intent-to-treat population. CI: confidence interval; I-IGA: Intertriginous Investigator Global Assessment.

Patient Examples Illustrating Efficacy of Roflumilast Cream 0.3% From DERMIS-1 & DERMIS-2



IGA: Investigator's Global Assessment; I-IGA: intertriginous-IGA

Once-daily Roflumilast Foam 0.3% for Scalp and Body Psoriasis: A Randomized, Double-blind, Vehicle-controlled Phase 2b Study

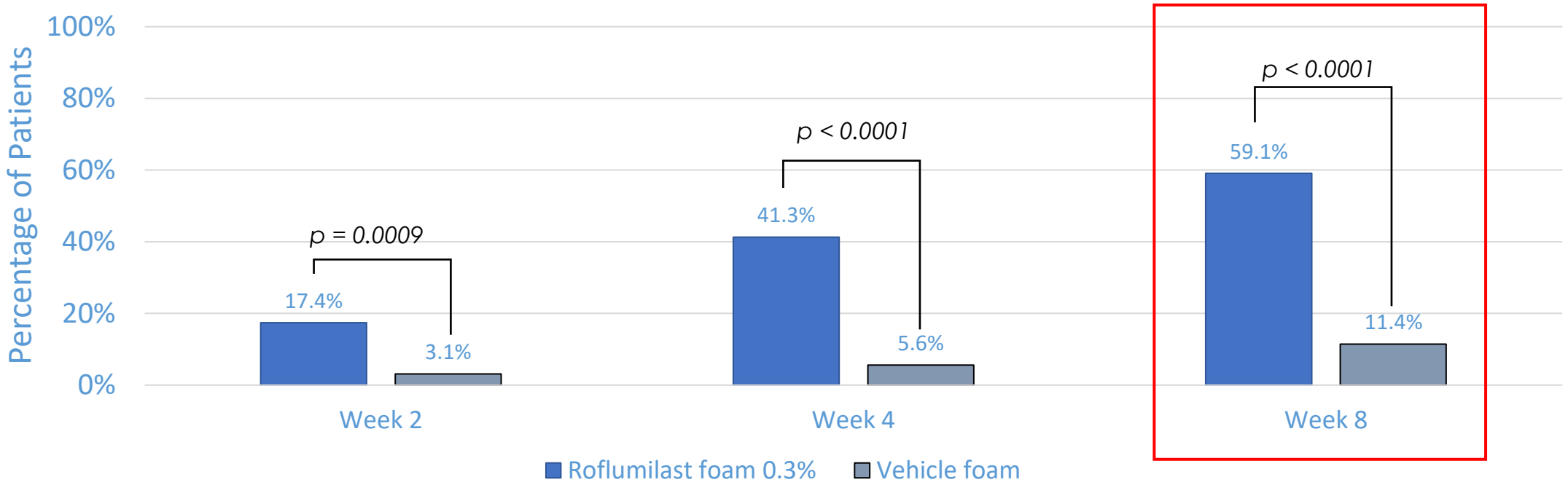
- Leon H Kircil[1], Angela Moore[2], Neal Bhatia[3], Alim R Devani[4], Zoe D Draelos[5], Janet DuBois[6], Melinda J Gooderham[7], Steven E Kempers[8], Edward Lain[9], Mark Lee[10], Dedee F Murrell[11], Kim A Papp[12], David M Pariser[13], Rodney Sinclair[14], Matthew Zirwas[15], Patrick Burnett[16], Robert C Higham[16], Lynn Navale[16], David R Berk[16]

- [1]Icahn School of Medicine at Mount Sinai, NY, Indiana Medical Center, Indianapolis, IN, Physicians Skin Care, PLLC, Louisville, KY, and Skin Sciences, PLLC, Louisville, KY, USA; [2]Arlington Research Center, Arlington, TX, USA, and Baylor University Medical Center, Dallas, TX; [3]Therapeutics Clinical Research, San Diego, CA, USA; [4]Dermatology Research Institute, Skin Health & Wellness Centre and Probity Medical Research, Calgary, AB, Canada; [5]Dermatology Consulting Services, High Point, NC, USA; [6]DermResearch, Inc., Austin, TX, USA; [7]SkiN Centre for Dermatology, Probity Medical Research and Queen's University, Peterborough, ON, Canada; [8]Minnesota Clinical Study Center, Fridley, MN, USA; [9]Sanova Dermatology, Austin, TX, USA; [10]Progressive Clinical Research, San Antonio, TX, USA; [11]UNSW, Sydney, Australia; [12]Probity Medical Research and K Papp Clinical Research, Waterloo, ON, Canada; [13]Eastern Virginia Medical School and Virginia Clinical Research, Inc., Norfolk, VA, USA; [14]Sinclair Dermatology, East Melbourne, Australia; [15]Dermatologists of the Central States, Probity Medical Research, and Ohio University, Bexley, OH, USA; [16]Arcutis Biotherapeutics, Inc., Westlake Village, CA, USA

This study was supported by Arcutis Biotherapeutics

Roflumilast Foam Significantly Increased the Percentage of Patients with S-IGA Success at Week 8 (Primary Endpoint)

Approx 60% of Patients Achieved S-IGA Success at Week 8
Significant Efficacy was Demonstrated as Early as Week 2



34.3% of patients on roflumilast achieved S-IGA = 0 (clear) versus 3.4% on vehicle

IGA Success = Clear or Almost Clear with at least a 2-grade improvement from baseline

Intent-to-treat population

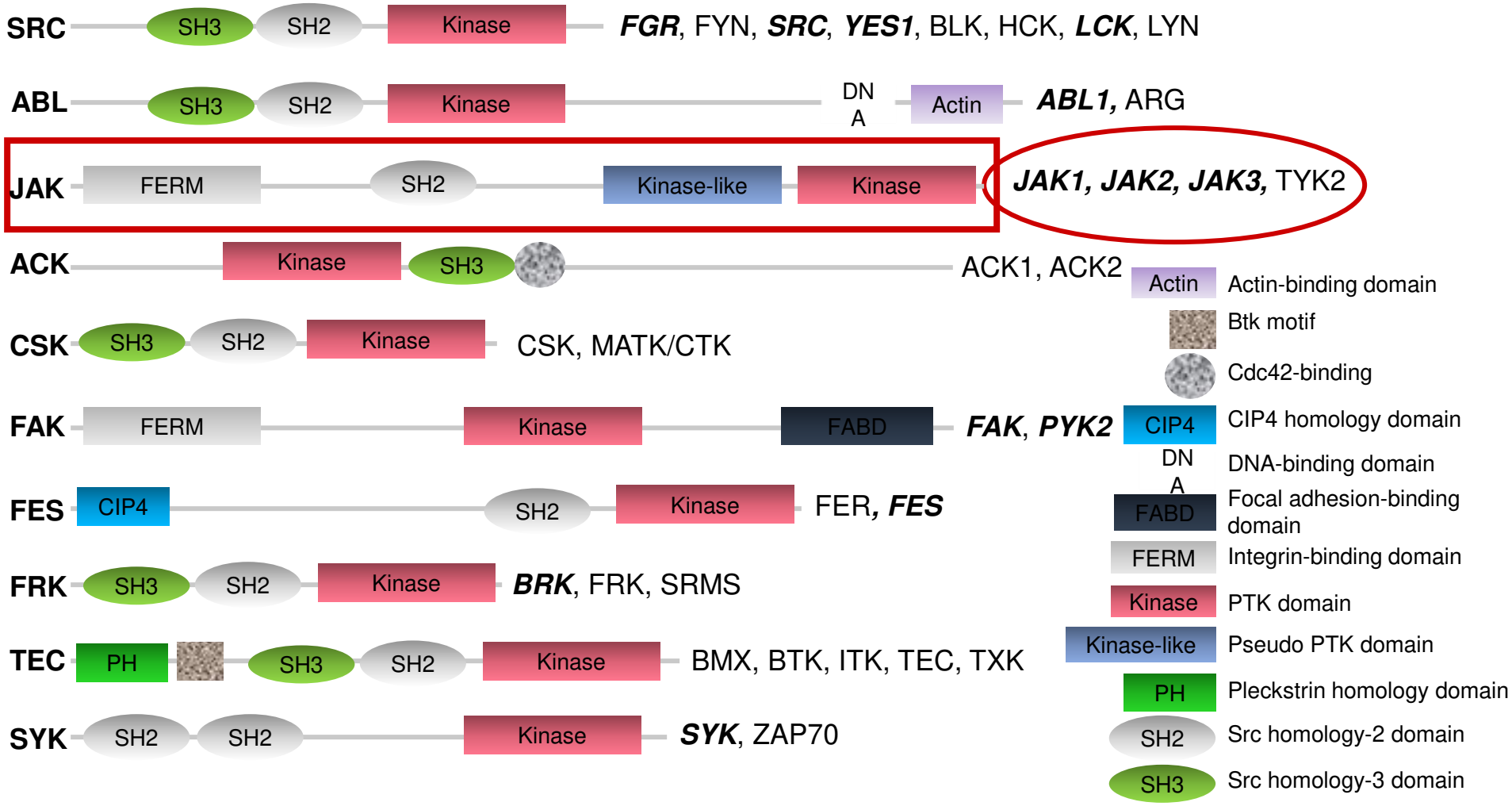
S-IGA: Scalp-Investigator's Global Assessment

GAME CHANGER IN PSORIASIS
AND
ATOPIC DERMATITIS TREATMENT

NEW CHEMICAL ENTITIES

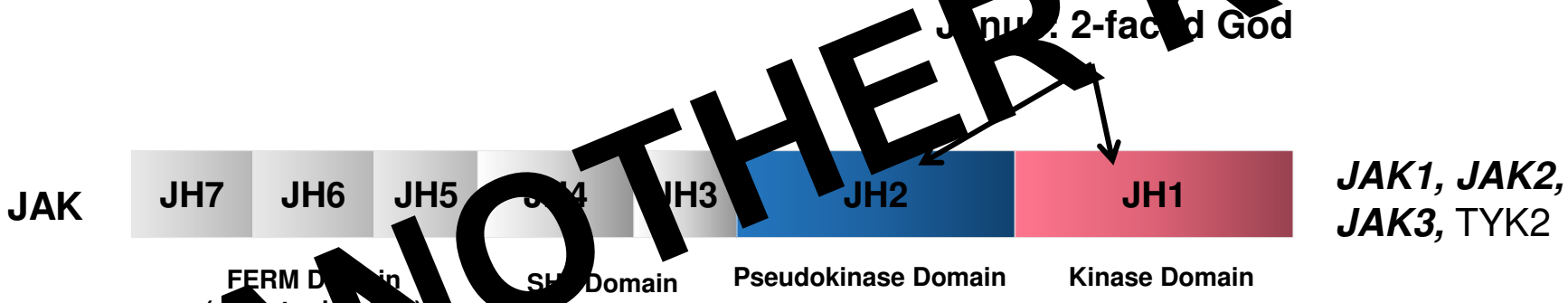
JAK/STAT Signaling Pathways

Janus Kinases (JAKs): Members of Nonreceptor Tyrosine Kinases



Blume-Jensen and Hunter. *Nature* 2001;411(6835):355-65.

JAK Structure[1,2]

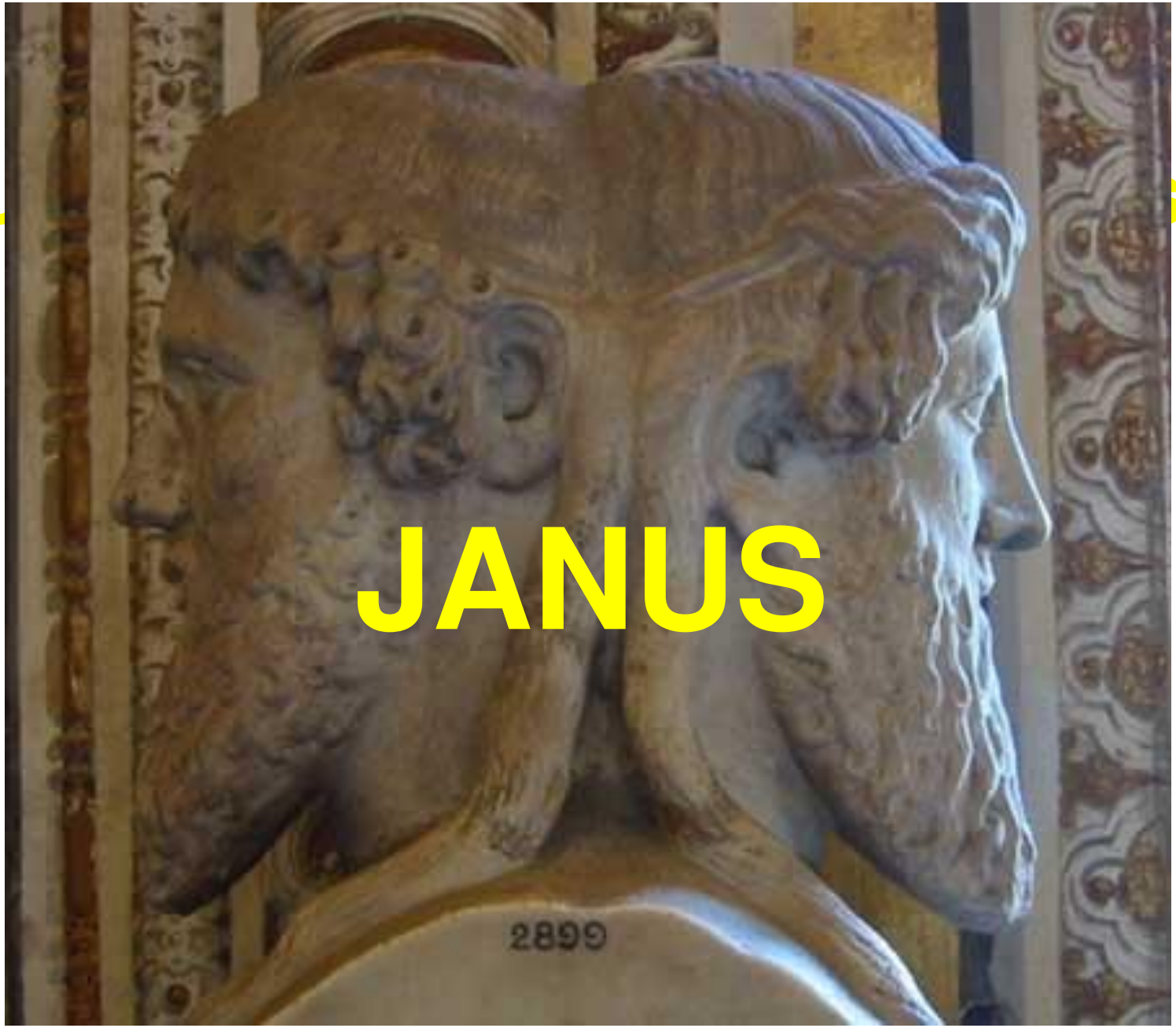


Of the 518 kinases identified in the human genome, only 5 have a pseudokinase and kinase domain present in the same protein, namely, the 4 members of the JAK family and GCN2, a serine threonine kinase

JUST ANOTHER KINASE

1.Pesu et al. *Immunol Rev* 2008;223:132-42.

2.Haan et al. In: Jak-Stat Signaling: From Basics to Disease, 2012.

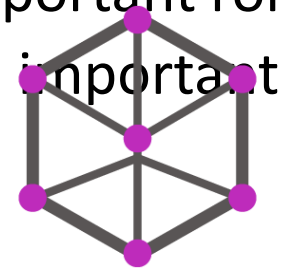

JANUS

How are the TYK2 and JAK 1/2/3 kinases different from each other?

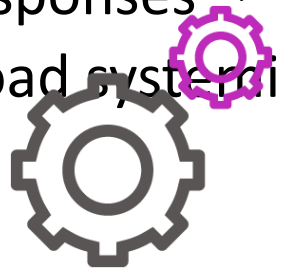

THIS IS MY BROTHER DARRYL,
AND MY OTHER BROTHER DARRYL
NEWHART

TYK2 and JAK1/2/3 kinases are each *structurally* and *functionally* different[25,26,28,29]

- TYK2 and JAK1/2/3 proteins belong to the same kinase family and are structurally distinct from each other[26,29]
- TYK2 and JAK1/2/3 proteins form different dimers to mediate different sets of cytokine signals that can influence immune and/or systemic responses[25,26,28,29]
  - TYK2 plays an important role in immune-specific responses[25,28]
  - JAK1/2/3 play an important role in immune and broad systemic responses[28]



Structural differences



Functional differences

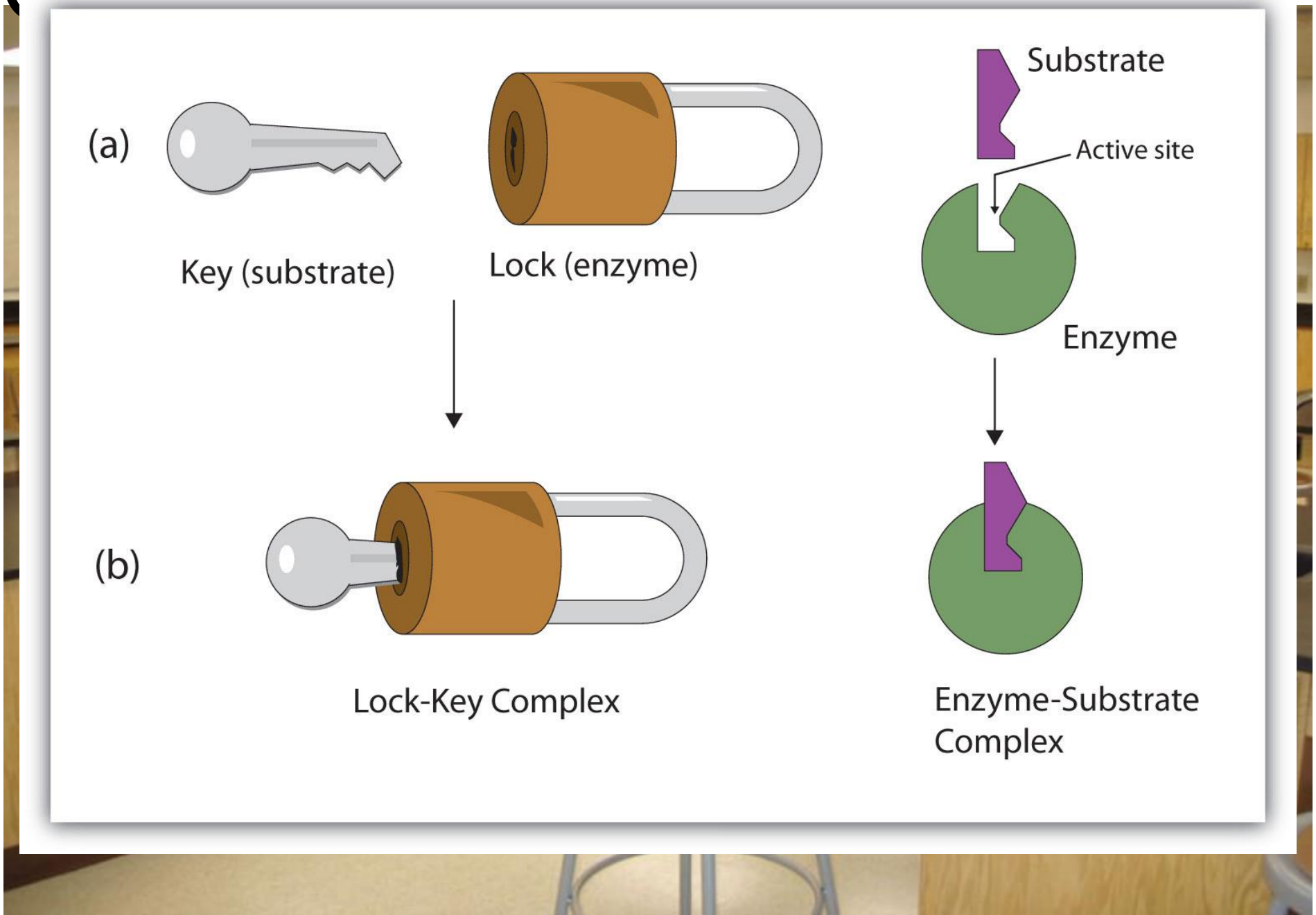
JAK=Janus kinase; TYK2=tyrosine kinase 2.

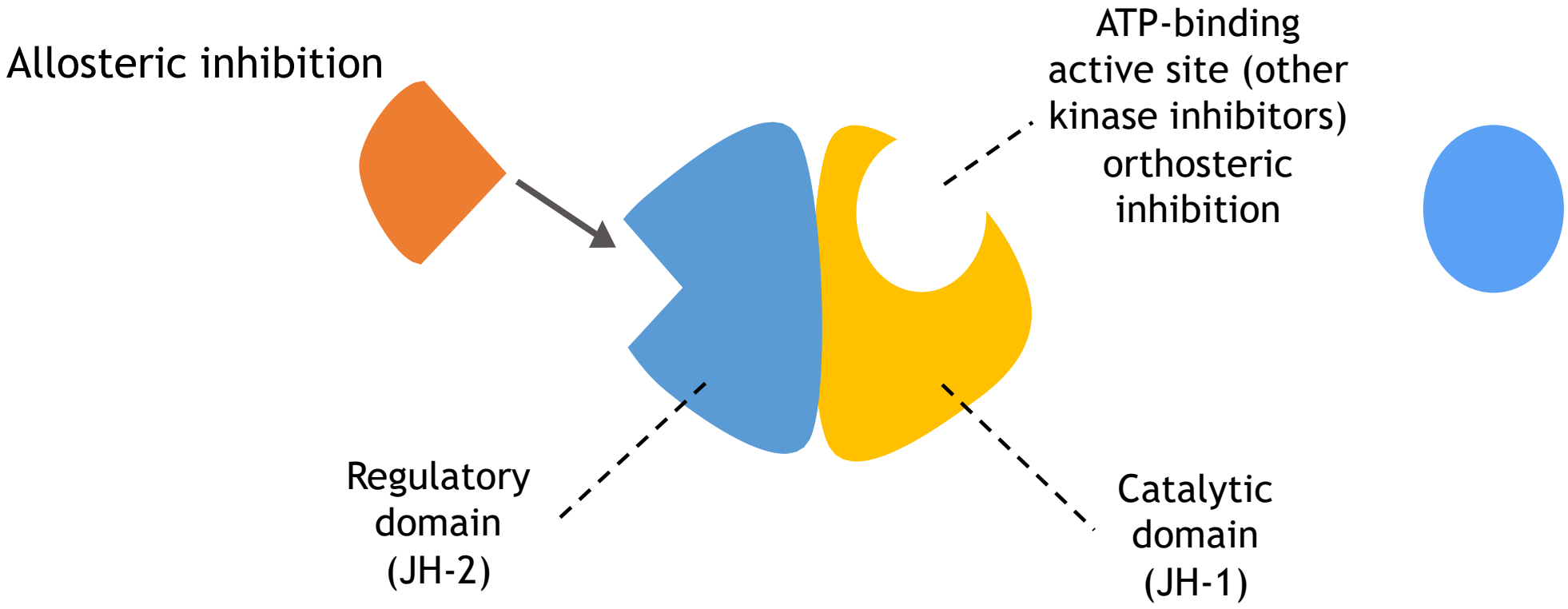
Systems affected by different cytokines mediated through TYK2 and JAK1/2/3

Systems affected by select pairings	JAK1	JAK2	JAK3	TYK2
 **Immune system**[25,28,33]	✓	✓	✓	✓
 **Blood cell development**[28,33]		✓		
 **Metabolic activity**[32,33,44]	✓	✓	✓	
 **Bone development and lipid metabolism**[33,35,38]	✓	✓		

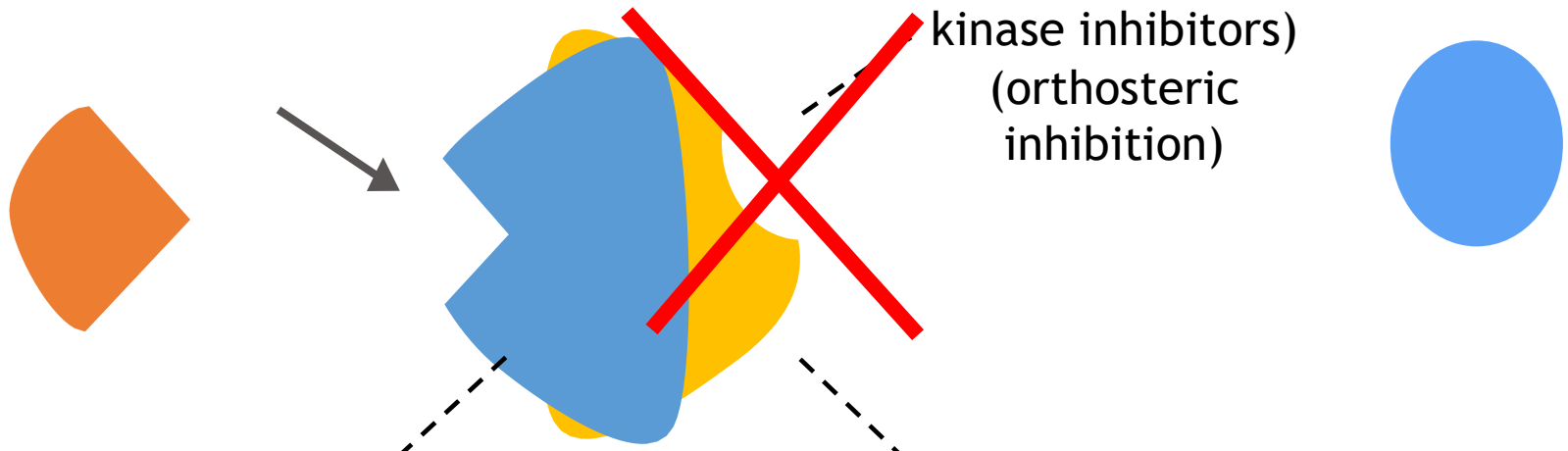
Please note that this list of systems affected by the different TYK2 and JAK1/2/3 pairings is not exhaustive.
JAK=Janus kinase; TYK2=tyrosine kinase 2.

HIGH SCHOOL CHEMISTRY



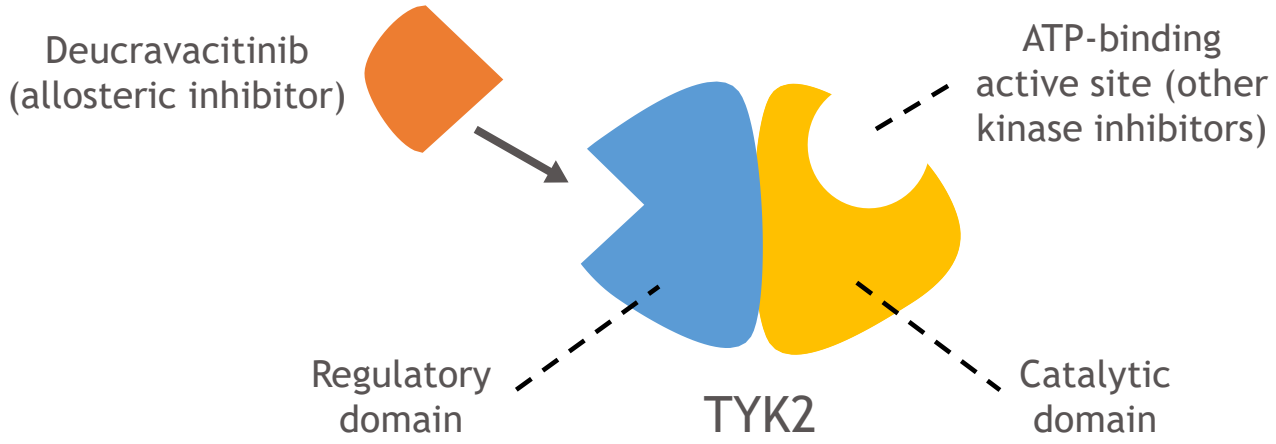

Allosteric inhibition
ATP-binding active site (other kinase inhibitors) orthosteric inhibition
Regulatory domain (JH-2)
Catalytic domain (JH-1)

Allosteric inhibition



Introduction

- Deucravacitinib
  - Novel, oral, selective tyrosine kinase 2 (TYK2) inhibitor with a unique mechanism of action distinct from Janus kinase (JAK) 1, 2, 3 inhibitors[1]
    - Binds to the TYK2 regulatory domain with high selectivity and inhibits TYK2 via an allosteric mechanism[1]
      - ≥100-fold greater selectivity for TYK2 vs JAK1/3 and ≥2000-fold greater selectivity for TYK2 vs JAK2[1,2]
    - Inhibits TYK2-mediated signaling by cytokines involved in psoriasis pathogenesis (eg, IL-23, IL-12, and Type 1 interferon)[1]
  - Previously demonstrated efficacy and tolerability in Phase 2 trials in moderate to severe plaque psoriasis[3] and active psoriatic arthritis[4]



1. Burke JR et al. *Sci Transl Med*. 2019;11:1-16; **2**. Wrobleski ST et al. *J Med Chem*. 2019;62:8973-8995; **3**. Papp K et al. *N Engl J Med*. 2018;379:1313-21; **4**. Mease PJ et al. Presented at Annual Scientific Meeting of the American College of Rheumatology; Nov 5-9, 2020.

Laboratory abnormalities observed with individual JAK family inhibitors[1,2]*

No head-to-head trials were conducted

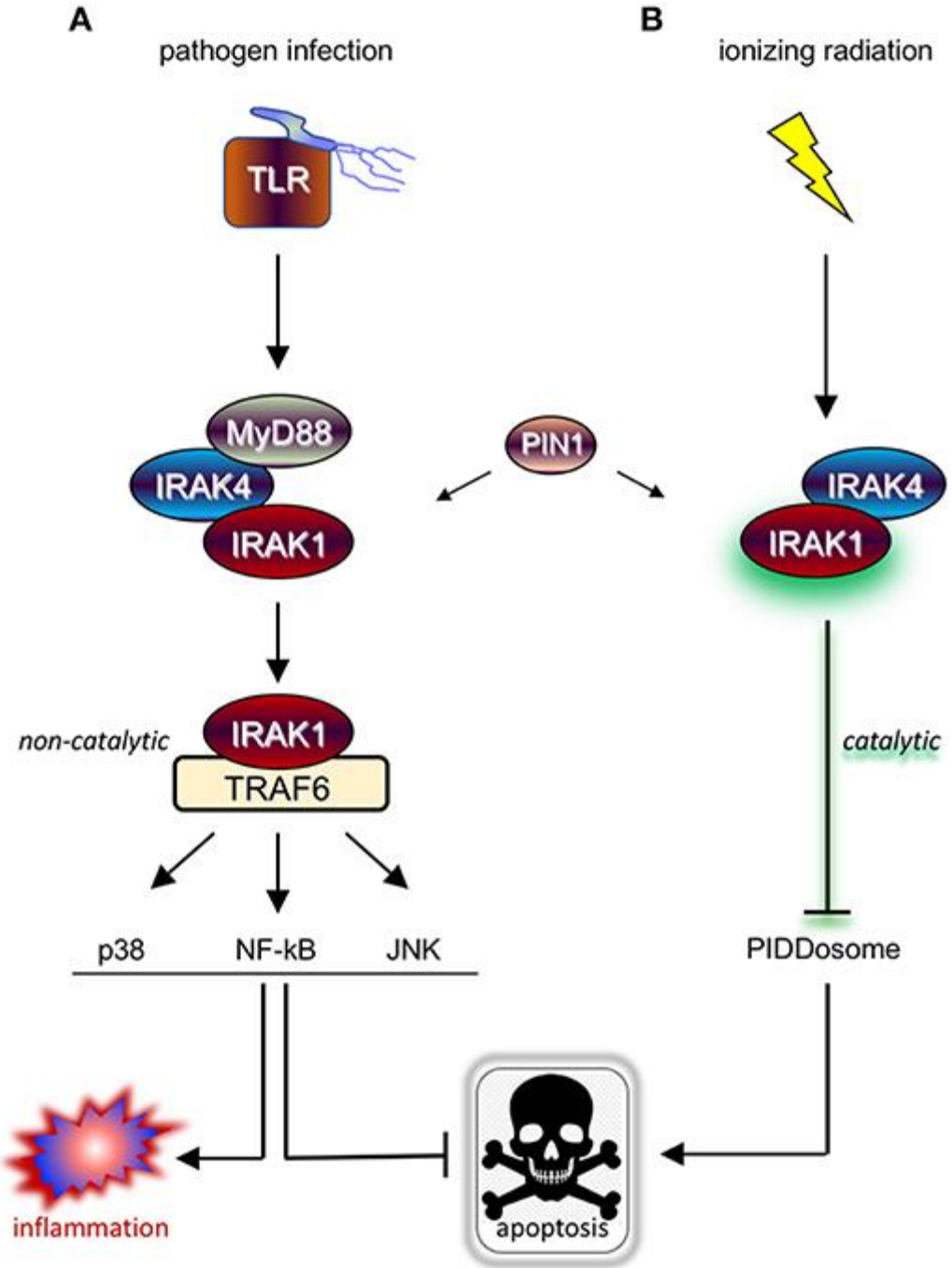
	Tofacitinib[2]	Baricitinib[2]	Filgotinib[2]	Upadacitinib[2]	BMS-986165[1]
Selectivity	JAK1, JAK3	JAK1, JAK2	JAK1	JAK1	TYK2
Lymphocyte number	↓*	No change**	No change**	↓†	No change
NK cell number	↓*	↓‡	No change	↓§	No change
Neutrophil number	↓*	↓*	↓	↓†	No change
Hemoglobin level	↑¶	↓*	↑	↓†	No change
Platelet count	↓‡	No change‖	↓	NA	No change
Liver transaminase level	↑*	↑*	No change	↑†	No change
Creatine phosphokinase level	↑*	↑*	NA	↑†	↔‡
HDL level	↑*	↑*	↑	↑†	No change
LDL level	↑*	↑*	No change	↑†	No change
Creatinine level	↑*	↑*	↑	↑§	No change

*Shown are the general trends reported in the development programs of each compound across different indications and doses.[1] †Initial rise followed by a decrease. ‡Cas[illegible] kinase elevations from baseline (most of which had a Common Terminology Criteria for Adverse Events grade of 1 or 2) were observed in 12/44 (27%) patients who received placebo, and 57/221 (26%) patients treated with BMS-986165, with no clear dose dependence; these were associated with increased physical activity, resolved spontaneously, and did not result in trial drug discontinuation. *Noted in both USPI and SmPC. **Decreases noted in both USPI and SmPC. †Noted in USPI (SmPC not available). ‡Not noted in either USPI or SmPC. §Not noted in USPI (SmPC not available). ¶According to the SmPC, hemoglobin levels are decreased with tofacitinib. While the USPI does not specifically state that tofacitinib use is associated with changes in hemoglobin, it does provide guidance for drops in hemoglobin. ‖Increases noted in both USPI and SmPC.

HDL=high-density lipoprotein; JAK=Janus kinase; LDL=low-density lipoprotein; NA=not applicable; NK=natural killer; TYK=tyrosine kinase.

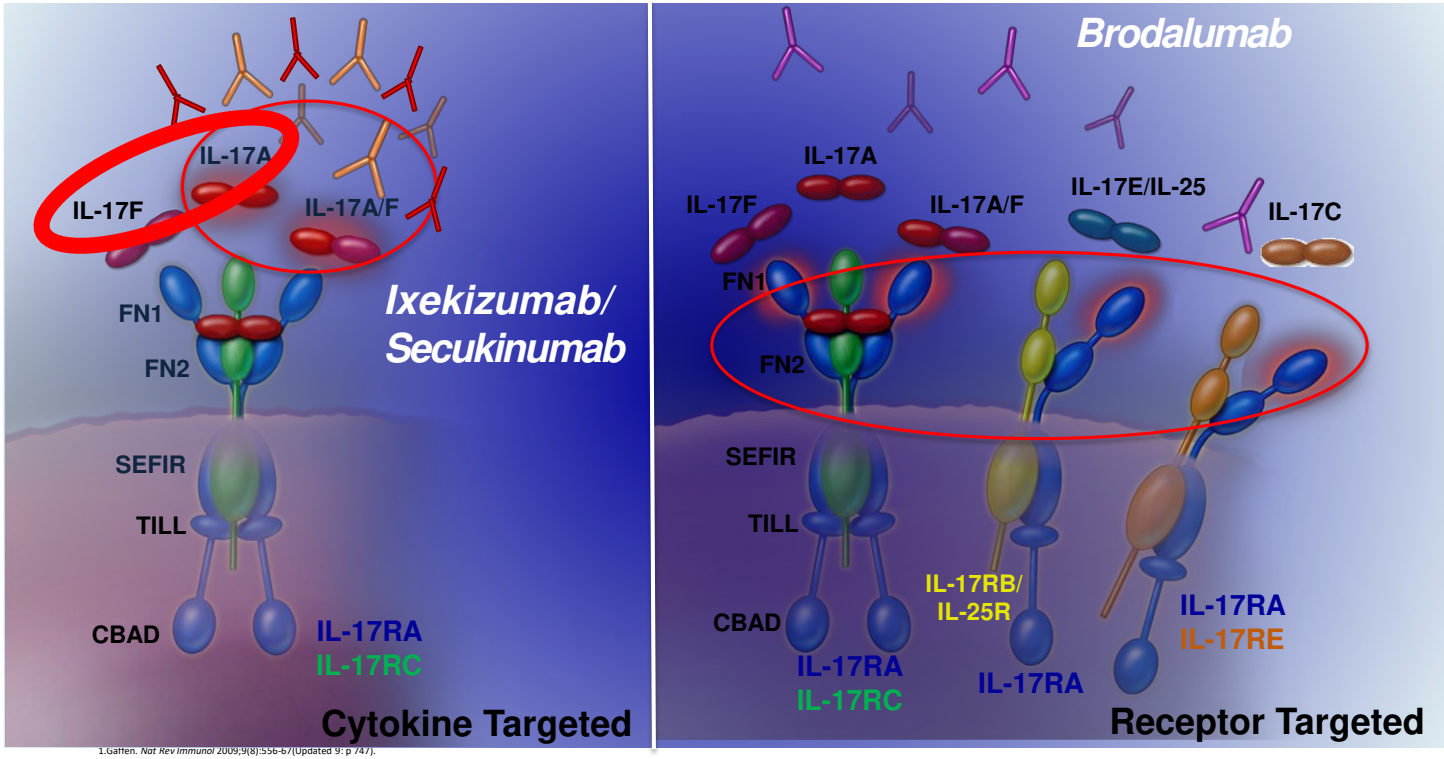
1. Papp K et al. *N Engl J Med.* 2018;379:1313-1321. 2. Winthrop KL. *Nat Rev Rheumatol.* 2017;13:234-243.


COUSINS
MY NAME IS NOT JAK
JAK
TYK


A
pathogen infection
B
ionizing radiation
TLR
MyD88
IRAK4
IRAK1
PIN1
IRAK4
IRAK1
non-catalytic
IRAK1
TRAF6
catalytic
p38
NF-kB
JNK
PIDDosome
inflammation
apoptosis

- IRAK4 is the most proximal kinase in the Toll-like receptor (TLR)/IL-1R signaling cascade. Activation of the cascade triggers assembly of the myddosome complex and the downstream production of proinflammatory cytokines. Human and rodent genetics support the role of IRAK4 in the immune response.
- Over a dozen pharmaceutical companies have reported the discovery of IRAK4 inhibitors. Many of the reported compounds are potent enzyme inhibitors. IRAK4 inhibitors have been found to be active in a broad range of cellular and *in vivo* models.
- The work disclosed in patent applications over the last several years has led to multiple IRAK4 inhibitors being advanced to the clinic. Pfizer has enrolled patients in a phase II trial for RA.
- Emerging data suggests IRAK4 inhibition may offer a therapeutic benefit in the treatment of cancer. Aurigene and Curis have reported the start of a clinical trial evaluating IRAK4 inhibition for non-Hodgkin lymphoma.

BIMEKIZUMAB



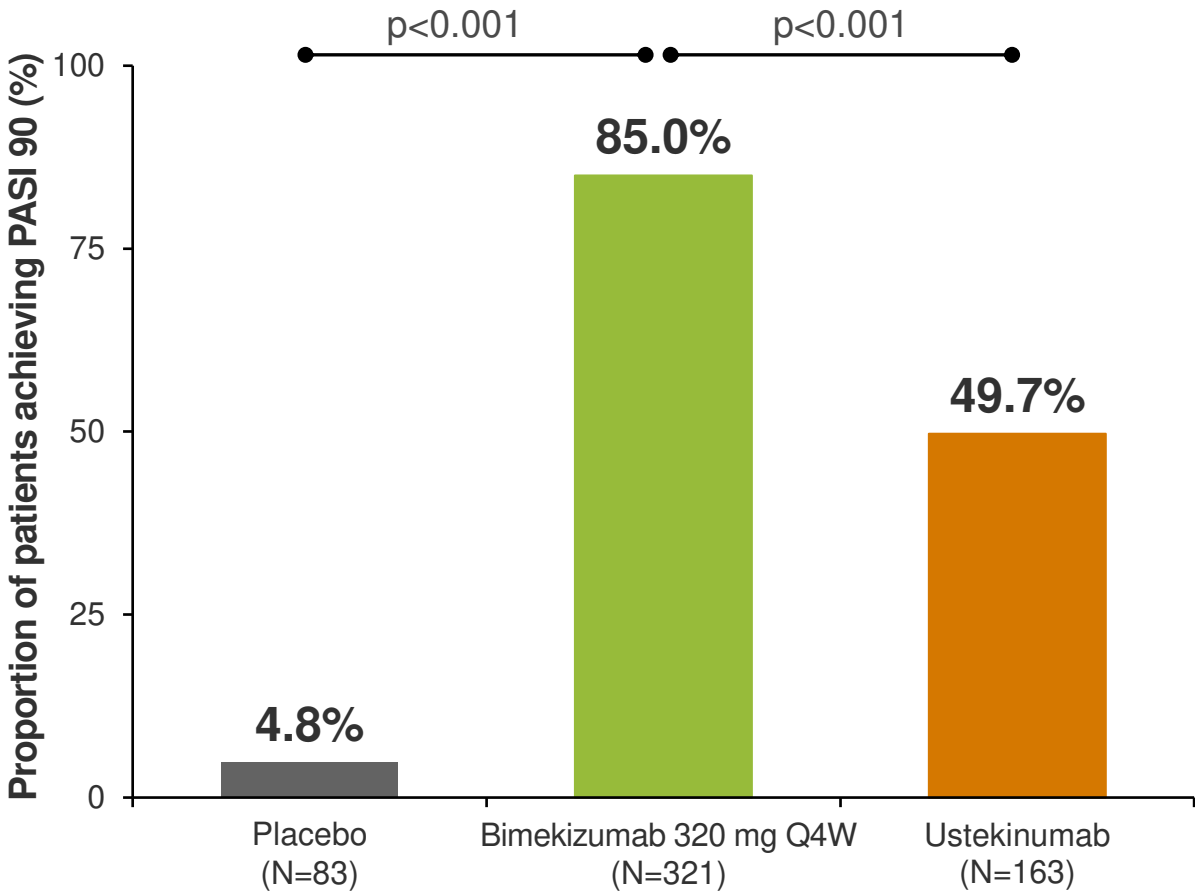
1.Gaffen. *Nat Rev Immunol* 2009;9(8):556-67(Updated 9: p 747).

2.Chang et al. *Immunity* 2011;35(4):611-21.

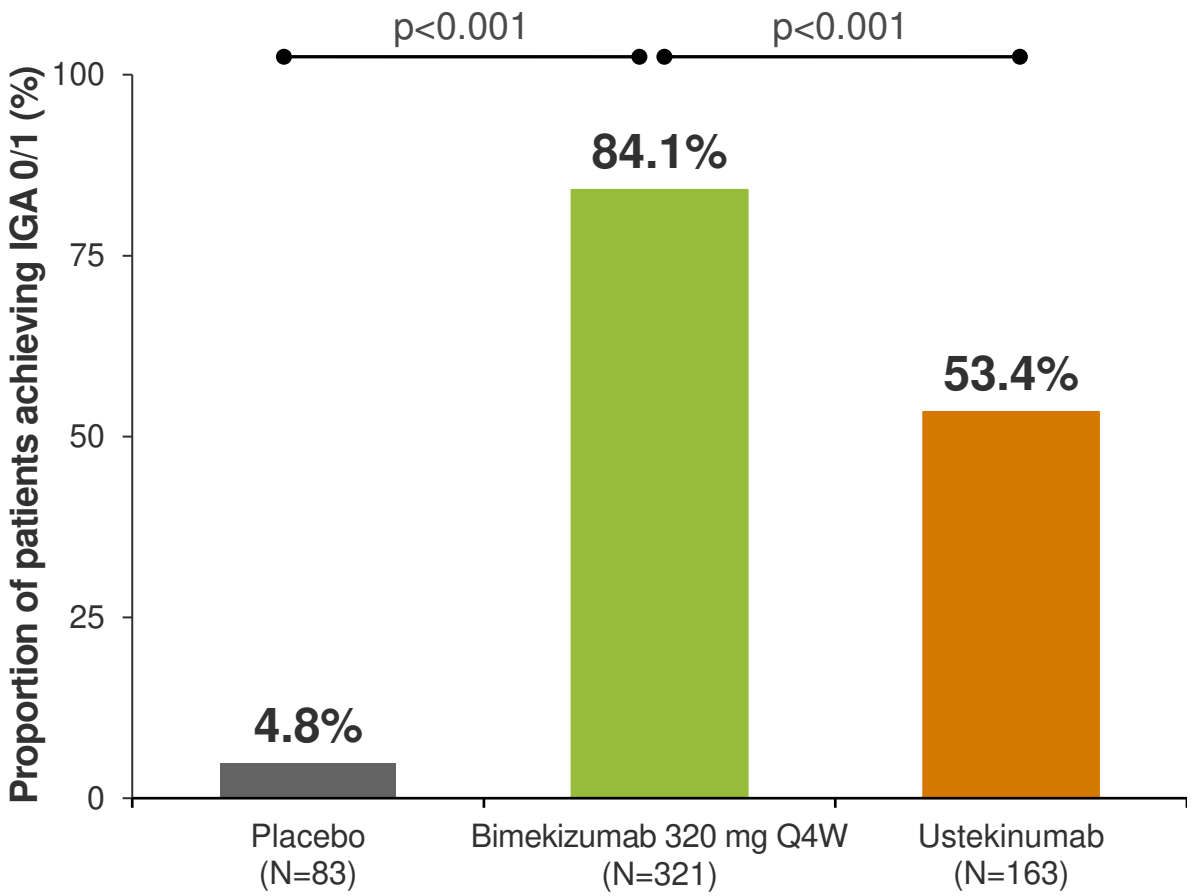
BE VIVID PASI 90 and IGA 0/1 at Week 16 (ITT, NRI)

Co-primary endpoints: superiority with bimekizumab versus placebo
Key secondary endpoints: superiority with bimekizumab versus ustekinumab

Week 16 PASI 90



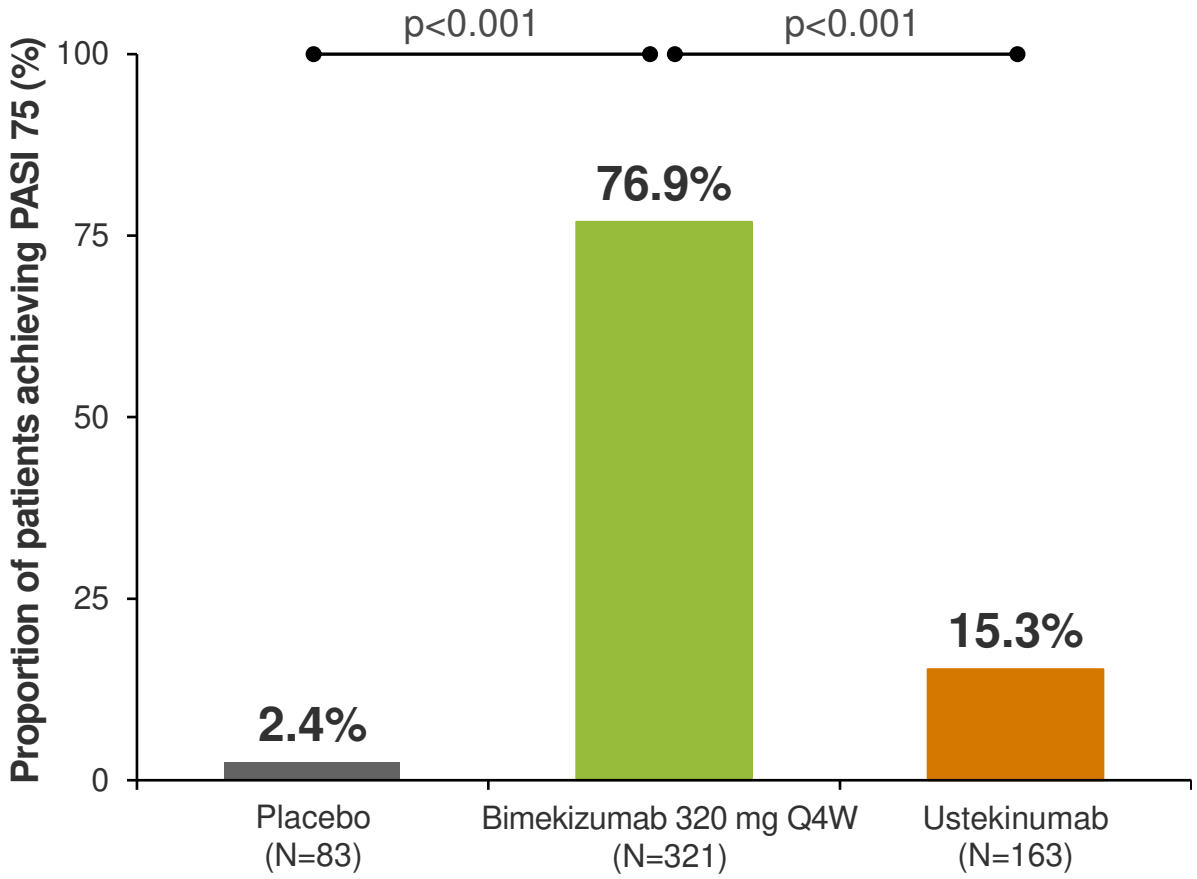
Week 16 IGA 0/1



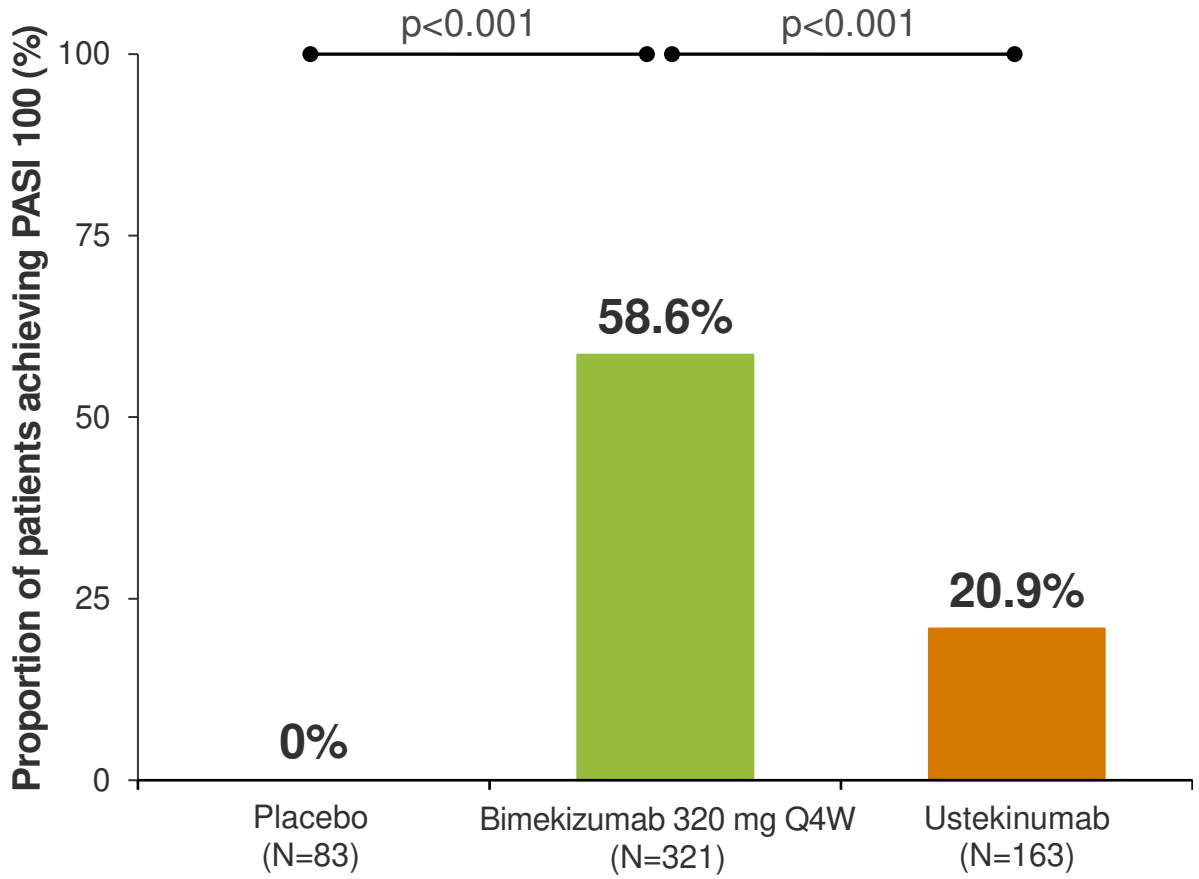
p values for the comparison of treatment groups were based on the Cochran–Mantel–Haenszel test from the general association. Proportions were calculated using non-responder imputation (NRI). IGA 0/1: score of 0 (clear) or 1 (almost clear) with ≥2-category improvement relative to Baseline in Investigator's Global Assessment, scored on a 5-point scale; ITT: intent-to-treat; PASI 90: ≥90% improvement from Baseline in Psoriasis Area and Severity Index; Q4W: every four weeks.

BE VIVID PASI 75 at Week 4 and PASI 100 at Week 16 (ITT, NRI)

Week 4 PASI 75

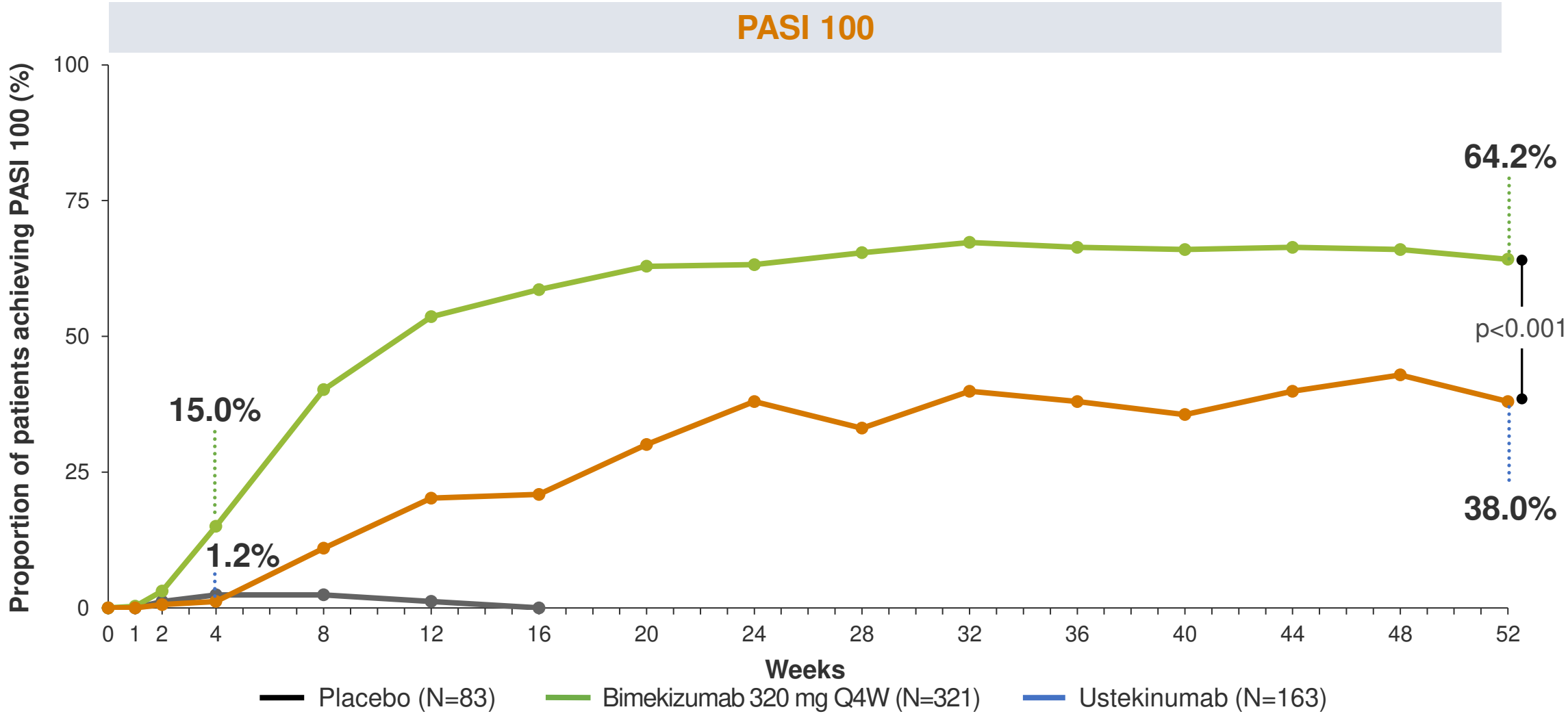


Week 16 PASI 100



For PASI 75 at Week 4, the p value for the comparison of treatment groups was based on the Cochran–Mantel–Haenszel test from the general association; for PASI 100 at Week 16, the p value for a general association was based on a stratified Cochran–Mantel–Haenszel test where region and prior biologic exposure were used as stratification variables, is considered nominal, and was not controlled for multiplicity. Proportions were calculated using non-responder imputation (NRI). ITT: intent-to-treat; PASI 75/100: ≥75/100% improvement from Baseline in Psoriasis Area and Severity Index; Q4W: every four weeks.

BE VIVID PASI 100 over 52 weeks (ITT, NRI)



The p value for a general association was based on a stratified Cochran–Mantel–Haenszel test where region and prior biologic exposure were used as stratification variables, is considered nominal, and was not controlled for multiplicity. Proportions were calculated using non-responder imputation (NRI). At Week 16, patients receiving placebo were switched to bimekizumab 320 mg Q4W.

ITT: intent-to-treat; PASI 100: 100% improvement in psoriasis area severity index (PASI) score; Q4W: every four weeks.

TEAEs of Interest (Short- and Longer-Term)

	Initial treatment period (Week 0–16) n (%)				Short-term (Week 0–16) EAIR per 100 PY (95% CI)	Longer-term EAIR per 100 PY (95% CI)
	BKZ 320 mg Q4W[a] N=989	ADA[b] N=159	UST[c] N=163	PBO[d] N=169	BKZ 320 mg Q4W[a] N=989	All BKZ[e] N=1789
Exposure	306.0 PY	48.8 PY	[illegible]	51.6 PY	306.0 PY	1830.4 PY
Serious infections	3 (0.3)	0	(1.2)	0	1.0 (0.2, 2.9)	1.4 (0.9, 2.0)
Inflammatory bowel disease	1 (0.1)	0	0	0	0.3 (0.0, 1.8)	0.1 (0.0, 0.3)
Candida infections	(9.1)	0	0	0	30.6 (24.6, 37.6)	18.7 (16.7, 21.0)
Oral candidiasis	75 (7.6)	[illegible]	0	0	25.3 (19.9, 31.8)	16.4 (14.5, 18.5)
Adjudicated MACE	1 (0.1)	0	0	0	0.3 (0.0, 1.8)	0.7 (0.3, 1.1)
Malignancies (inc. NMSC)	4 (0.4)	1 (0.6)	0	1 (0.6)	1.3 (0.4, 3.4)	0.8 (0.5, 1.4)
Adjudicated SIB[f]	0	0	0	0	0	0.1 (0.0, 0.3)
Serious hypersensitivity reactions[g]	0	0	0	0	0	0.2 (0.0, 0.5)
Injection site reactions	27 (2.7)	3 (1.9)	2 (1.2)	2 (1.2)	9.0 (5.9, 13.1)	3.1 (2.4, 4.1)
Hepatic events	19 (1.9)	9 (5.7)	0	2 (1.2)	6.3 (3.8, 9.8)	5.6 (4.6, 6.8)

FLUCONAZOLE
KETOCONAZOLE

- EAIRs are patient incidence of new cases per 100 PY. [a]BKZ initial treatment period data are included from three pivotal phase 3 studies; [b]ADA initial treatment period data are from BE SURE; [c]UST initial treatment period data are from BE VIVID; [d]PBO initial treatment period data are from BE VIVID and BE READY; [e]BKZ longer-term data are pooled from four phase 3 trials and four phase 2 trials; [f]Includes one event adjudicated by the external Neuropsychiatric Committee (active suicidal ideation with some intent to act) in a patient with pre-existing psychiatric conditions; [g]Includes one fatal event of circulatory failure (adjudicated MACE), one event of atopic dermatitis-like disseminated eczema and one case of anaphylactic shock due to insect sting, all considered unrelated to study treatment. ADA: adalimumab; BKZ: bimekizumab: CI: confidence interval; EAIR: exposure-adjusted incidence rate; MACE: major adverse cardiovascular event; NMSC: non-melanomic skin cancers; PBO: placebo; PY: patient-years; Q4W: every 4 weeks; SIB: suicidal ideation and behaviour; UST: ustekinumab.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