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I have received funding either as an investigator, consultant, or a speaker from the following pharmaceutical companies:

- Abbott
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- Allergan
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- Amgen
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- Xenoport
- ZAGE

On the horizon…

EMERGING THERAPIES

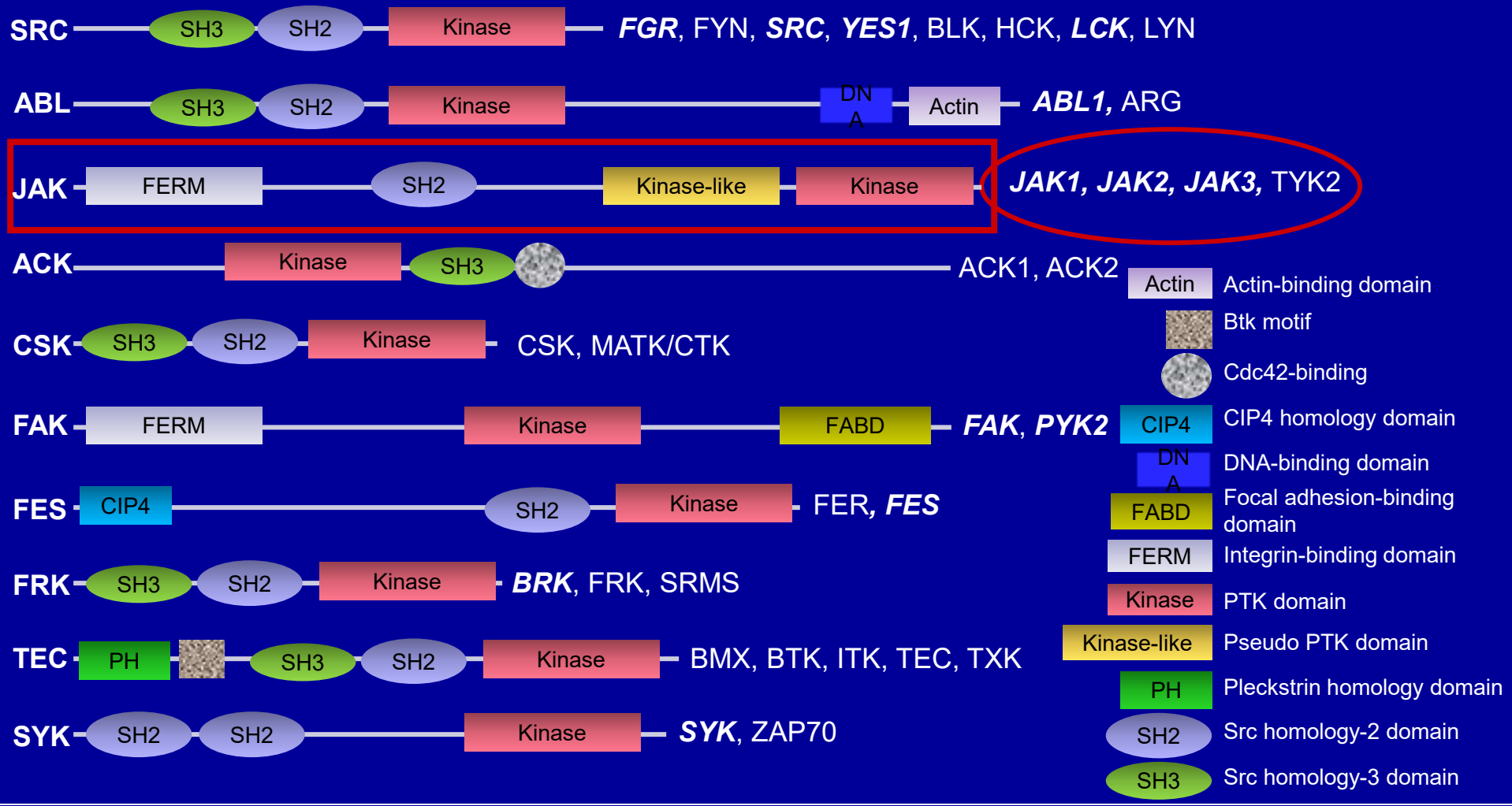
IS MEDICAL PRACTICE CATCHING UP WITH MEDICAL INNOVATION?

MY PERSPECTIVE FROM KENTUCKY

NEW CHEMICAL ENTITIES

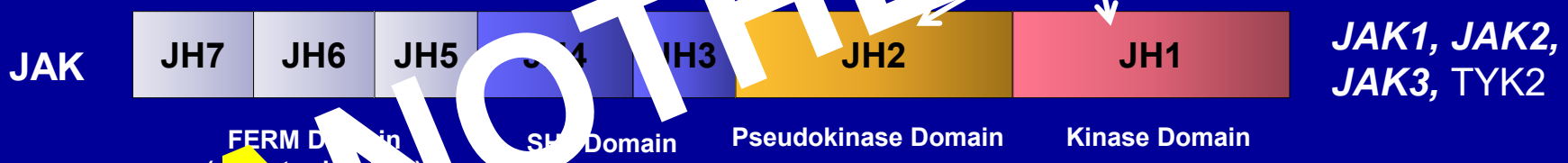
JAK/STAT Signaling Pathways

Janus Kinases (JAKs): Members of Nonreceptor Tyrosine Kinases



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

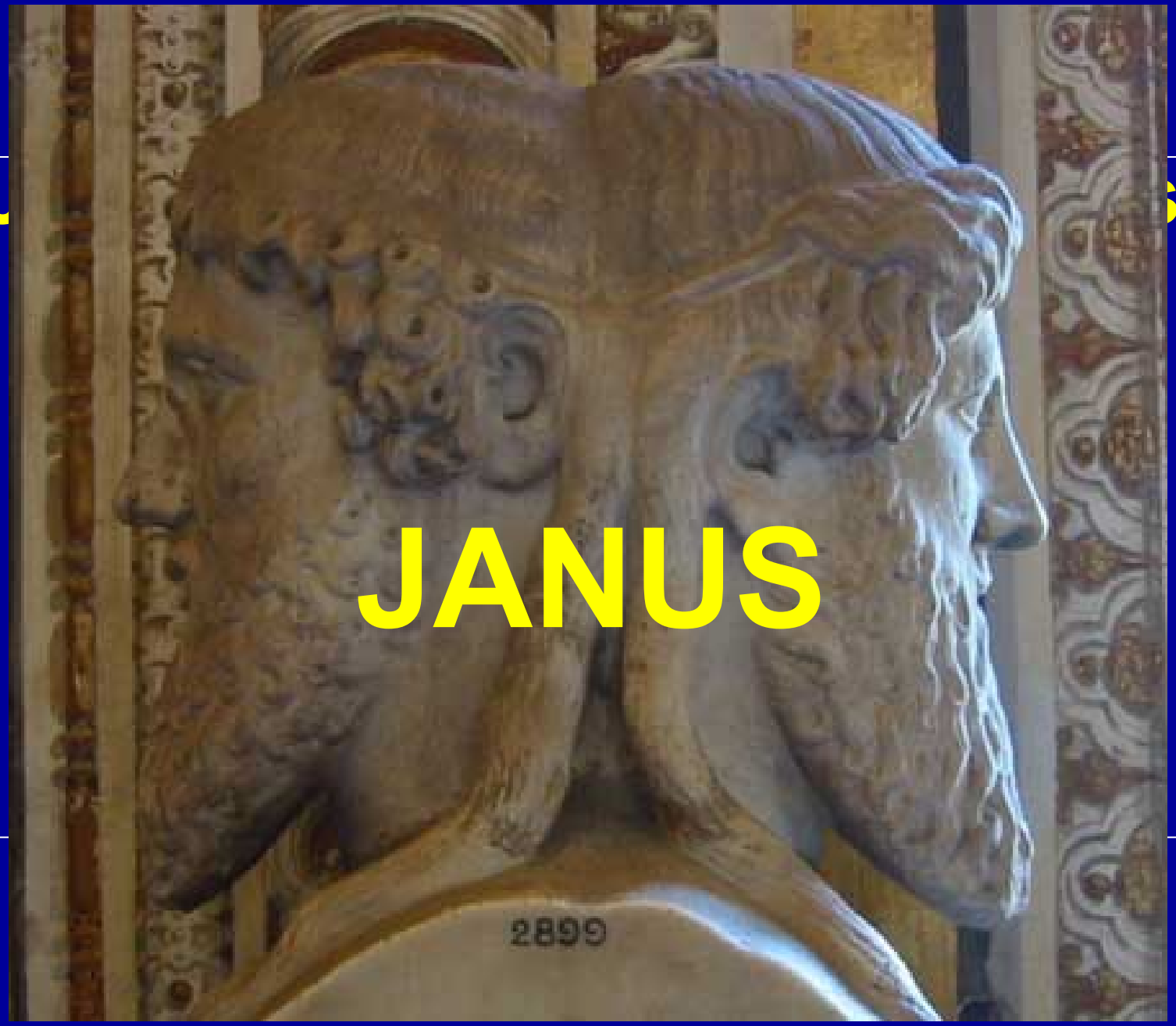
JAK Structure[1,2]



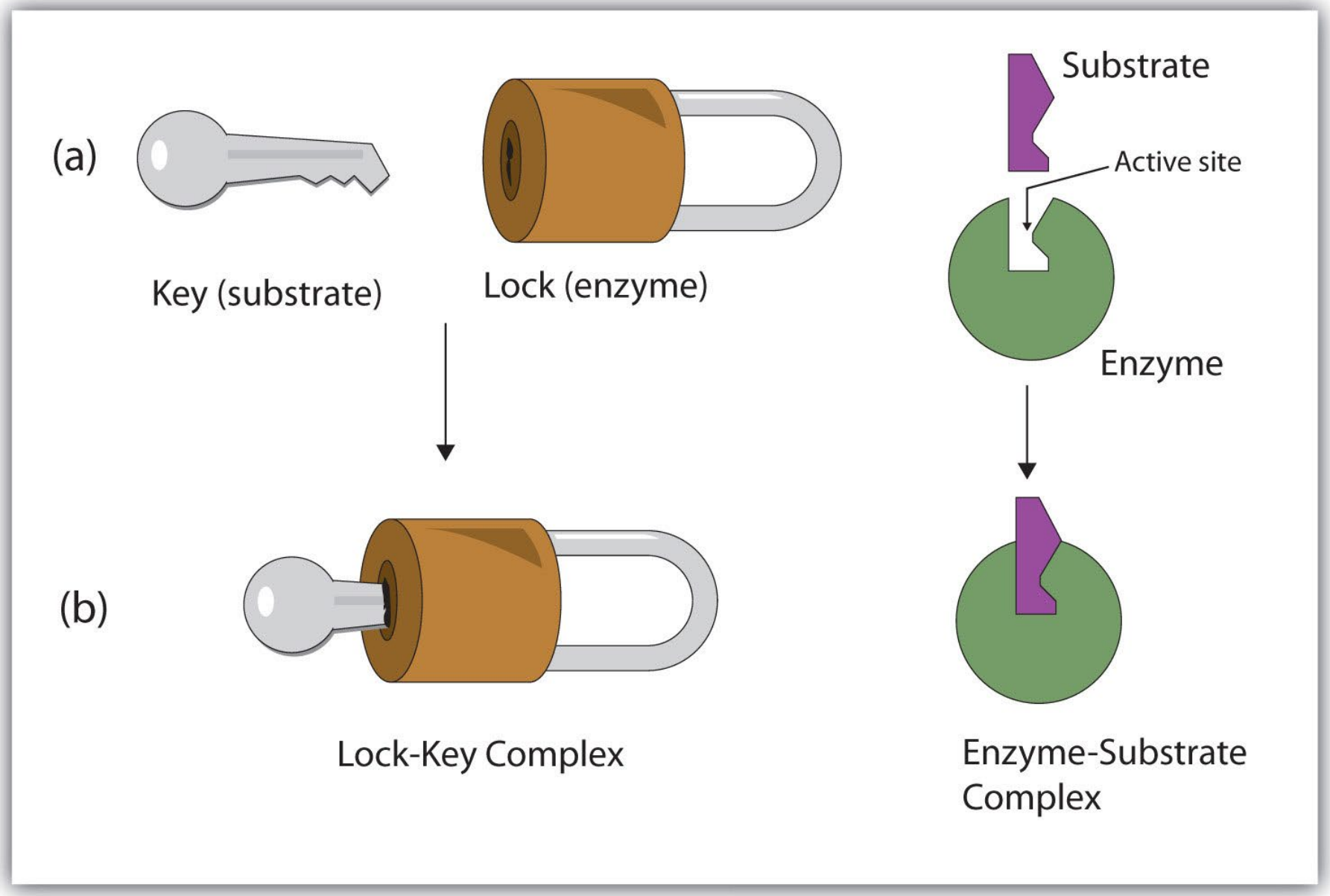
JUST ANOTHER KINASE

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

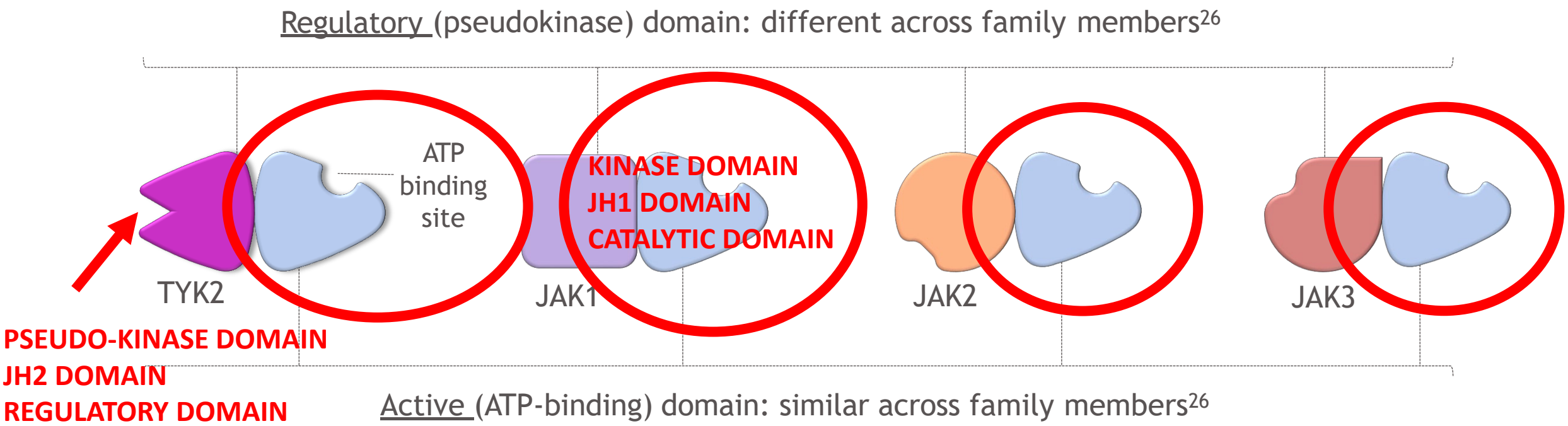
1. Pesu et al. *Immunol Rev* 2008;223:132-42.
2. Haan et al. In: Jak-Stat Signaling: From Basics to Disease, 2012.


JANUS

HIGH SCHOOL CHEMISTRY



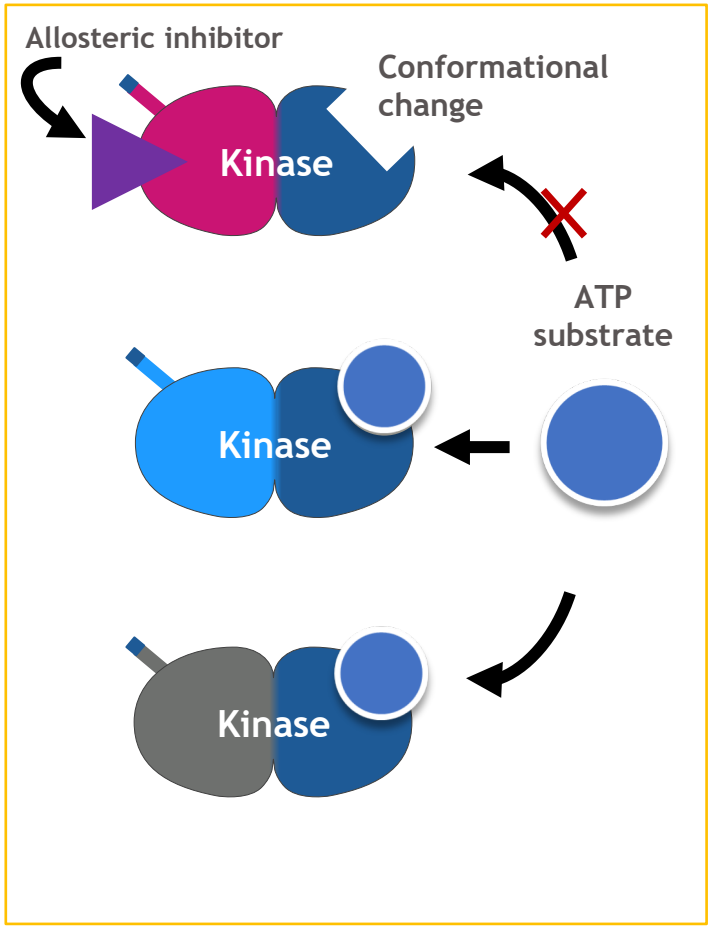
TYK2 and JAK1/2/3 kinases are *structurally* different from each other[26]



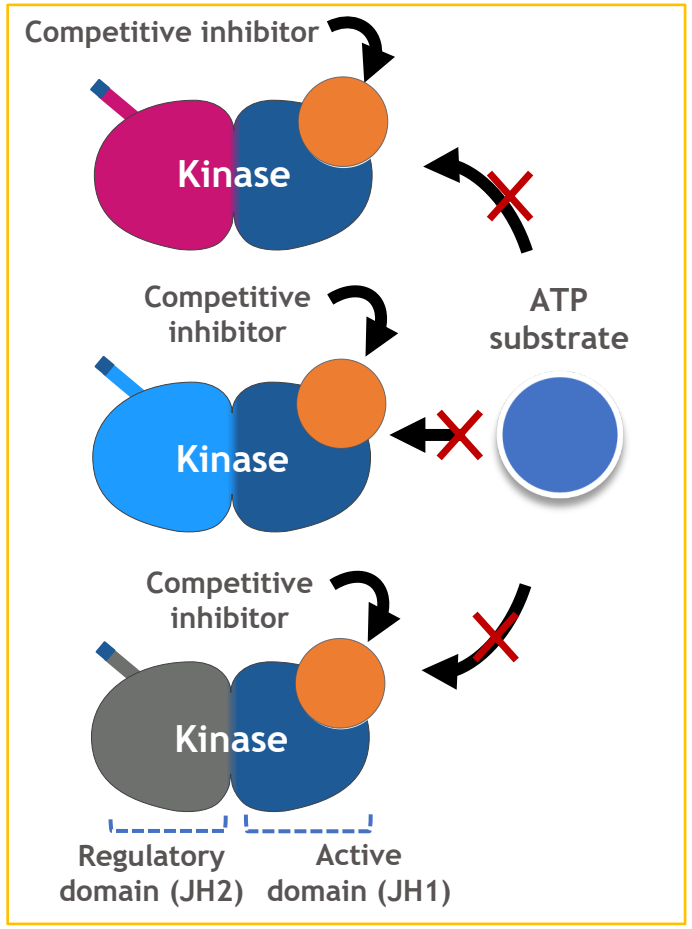
ATP=adenosine triphosphate; JAK=Janus kinase; TYK2=tyrosine kinase2.

Allosteric kinase inhibition by small molecules[1,2]

Allosteric inhibitors bind to a site other than the active site[1,3-5]



Competitive inhibitors bind to the conserved active site[1,3-5]



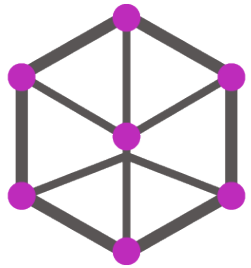
- Allosteric inhibition can prevent ATP from binding to the active domain in several ways[1,4]:
  - Inducing a conformational change to the active site construction
  - Blocking access to the active site
- Allosteric inhibitors tend to target less conserved sites versus competitive inhibitors and therefore can have a higher degree of specificity for a particular enzyme[1]

ATP=adenosine triphosphate.

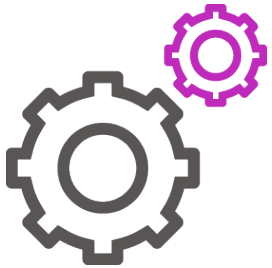
1. Nussinov R, Tsai C-J. *Cell*. 2013;153:293-305. 2. Imai K, Takaoka A. *Nat Rev Cancer*. 2006;6:714-727. 3. Berg JM et al. *Biochemistry*. 5th ed. 2002. 4. Strelow J et al, In: Markossian S et al, eds. *Assay Guidance Manual*. 2012. 5. Lu X et al. *Angew Chem Int Ed Engl*. 2020;59:1-13. doi.org/10.1002/anie.201914525.

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

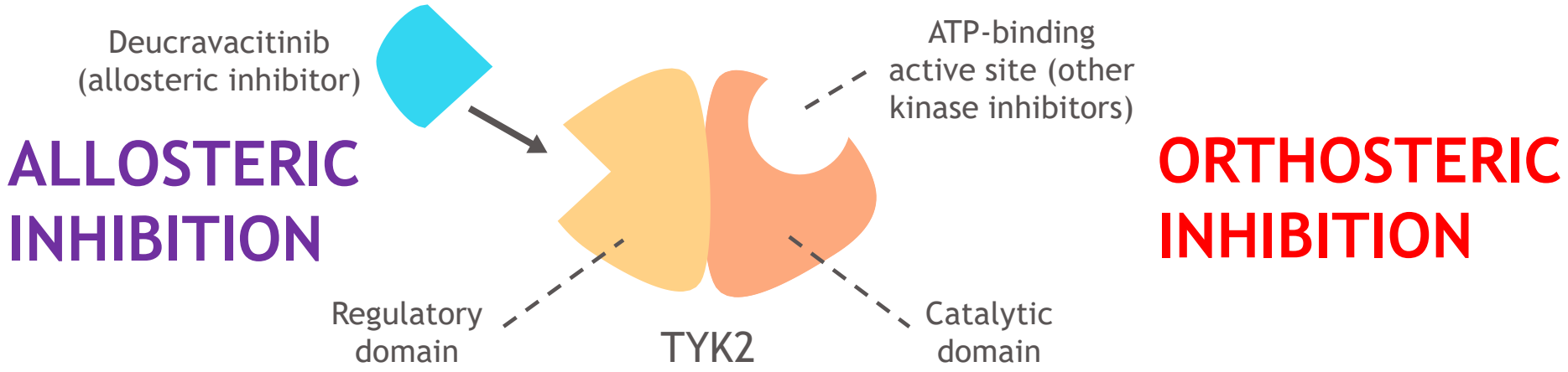


Functional differences

JAK=Janus kinase; TYK2=tyrosine kinase 2.

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.

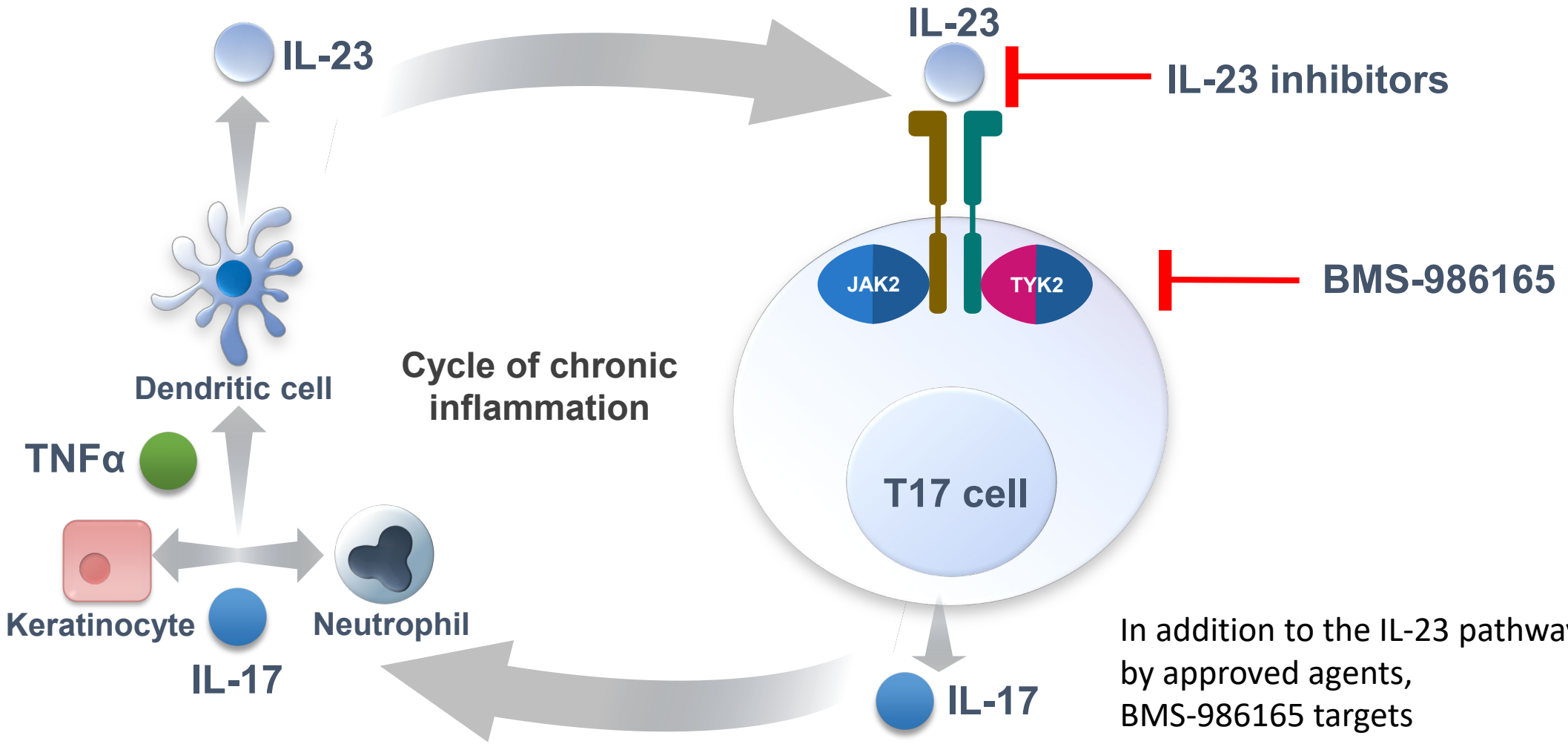
Bristol Myers Squibb Announces Deucravacitinib (BMS-986165) Demonstrated Superiority to Placebo and Otezla® (apremilast) in Pivotal Phase 3 Psoriasis Study

Deucravacitinib (BMS-986165) is the first and only novel, oral, selective tyrosine kinase 2 (TYK2) inhibitor in clinical studies across multiple immune-mediated diseases.

Deucravacitinib's selectivity is driven by a unique mechanism of action that is distinct from other kinase inhibitors.

TYK2 is an intracellular signaling kinase that mediates signaling of IL-23, IL-12 and Type I IFN, which are naturally occurring cytokines involved in inflammatory and immune responses.

BMS-986165 inhibits pathways in the IL-23/Th17 axis that are central to psoriasis pathogenesis[1-7]

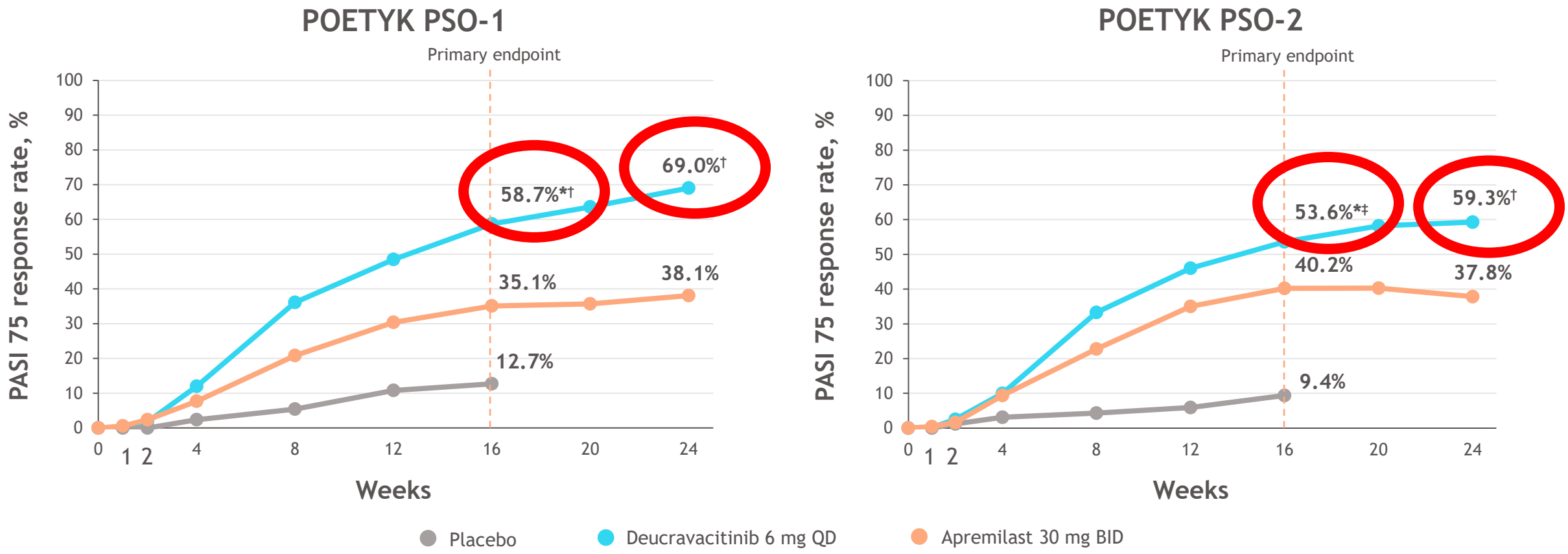


IFN=interferon; IL=interleukin; TNF=tumor necrosis factor.

1. Blauvelt A. *Expert Opin Biol Ther*. 2016;16(2):255-263. 2. Greb JE et al. *Nat Rev Dis Primers.* 2016;2:16082. 3. Alwan W, Nestle FO. *Clin Exp Rheumatol.* 2015;33(suppl 93):S2-S6. 4. Nestle FO et al. *N Engl J Med.* 2009;361:496-509. 5. Mahil SK et al. *Semin Immunopathol.* 2016;38:11-27. 6. Hodge JA et al. *Clin Exp Rheumatol.* 2016;34:318-328. 7. O'Shea JJ et al. In: Rich RR et al, eds. *Clinical Immunology: Principles and Practices.*5th ed. Cambridge, MA: Elsevier Inc; 2019. 8. Burke JR et al. *Sci Transl Med.* 2019;11(502):eaaw1736.

In addition to the IL-23 pathway inhibited by approved agents, BMS-986165 targets TYK2–mediated activity in T17, dendritic, and other immune cells[7,8]

PASI 75 response at Week 16 (coprimary endpoint) and through Week 24 (NRI)



- Significantly greater proportions of patients in the deucravacitinib compared with placebo and apremilast arms achieved PASI 75 response at Week 16 in both trials
  - Deucravacitinib was also superior to apremilast at Week 24
- 82.5% (PSO-1) and 81.4% (PSO-2) of deucravacitinib patients who achieved PASI 75 at Week 24 and continued treatment maintained PASI 75 response at Week 52

*P<0.0001 vs placebo. †P<0.0001 vs apremilast. ‡P=0.0003 vs apremilast.
NRI, nonresponder imputation.

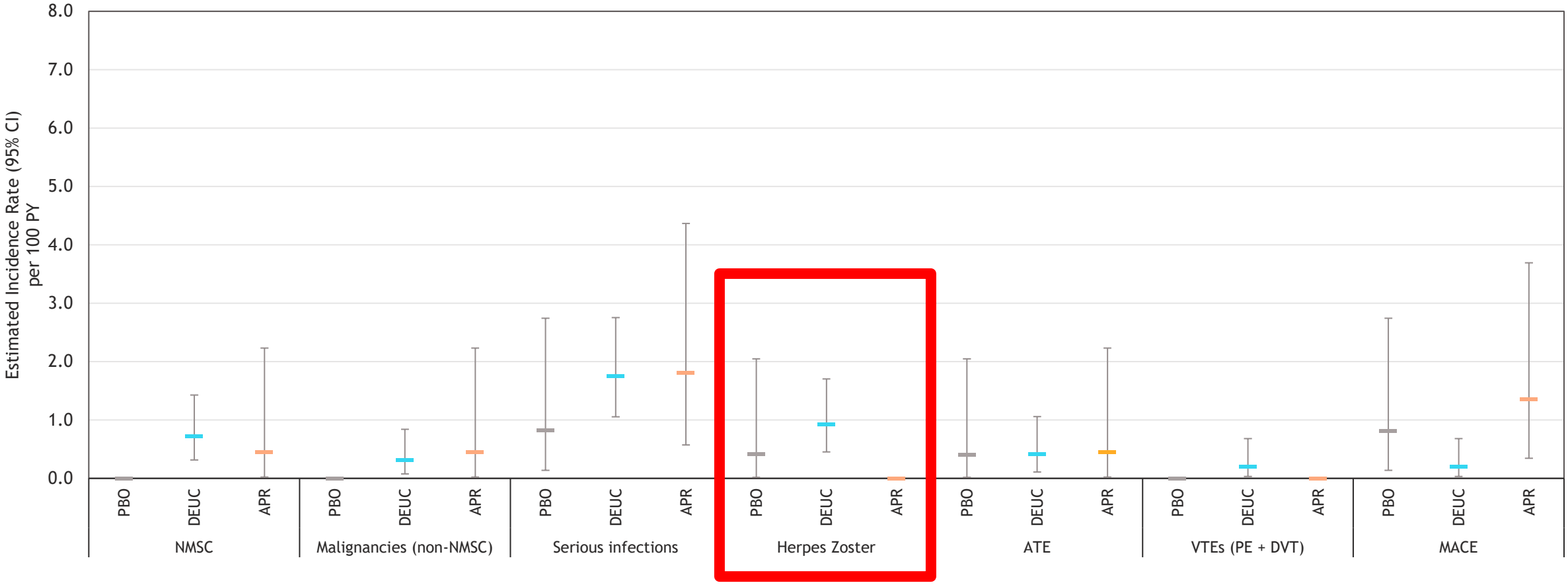
Safety summary, Weeks 0–52

AE category, n*, exposure-adjusted incidence rate (EAIR) events per 100 patient-years (PY)	POETYK integrated safety (PSO-1 and PSO-2)		
	Placebo n=666 (total PY; 240.9)	Deucravacitinib n=1364 (total PY, 969.0)	Apremilast n=422 (total PY, 221.1)
Any AEs	347, **217.9**	995, **229.2**	299, **281.1**
Serious AEs	14, **5.7**	55, **5.7**	9, **4.0**
AEs leading to discontinuation	23, **9.4**	43, **4.4**	26, **11.6**
Deaths	1	2†	1
Most common AEs (≥5%) in any active treatment group, n, EAIR			
Nasopharyngitis	54, **22.9**	229, **26.1**	54, **25.9**
Upper respiratory tract infection	33, **13.6**	124, **13.4**	27, **12.4**
Headache	21, **8.6**	80, **8.5**	53, **26.0**
Diarrhea	28, **11.6**	69, **7.3**	54, **26.5**
Nausea	10, **4.1**	20, **2.1**	47, **22.9**

- Per each study design, patients receiving placebo switched to deucravacitinib at Week 16 and patients receiving apremilast failing to meet study-specific efficacy thresholds (PASI 50 in PSO-1, PASI 75 in PSO-2) switched to deucravacitinib at Week 24
- Skin events of interest: folliculitis and acne
  - Folliculitis, 2.0% (EAIR, **2.8**) and acne, 2.1% (EAIR, **2.9**) with deucravacitinib
  - All cases were mild to moderate; 1 patient with folliculitis discontinued deucravacitinib treatment
- No new safety signals observed during Weeks 16–52

*Includes AEs between first dose and 30 days following last dose or rollover to long-term extension.
†1 additional death between Week 16—52 due to hepatocellular carcinoma in a patient with a history of HCV infection and liver cirrhosis.

AEs of interest (integrated), Weeks 0–52



- None of the serious infections with deucravacitinib led to discontinuation
- No cases of herpes zoster with deucravacitinib were serious, systemic, or led to discontinuation
- No tuberculosis events and no opportunistic systemic infections were reported with deucravacitinib
- 1 SAE adjudicated as a VTE occurred in a patient receiving deucravacitinib who had an aortic dissection complicated by a PE

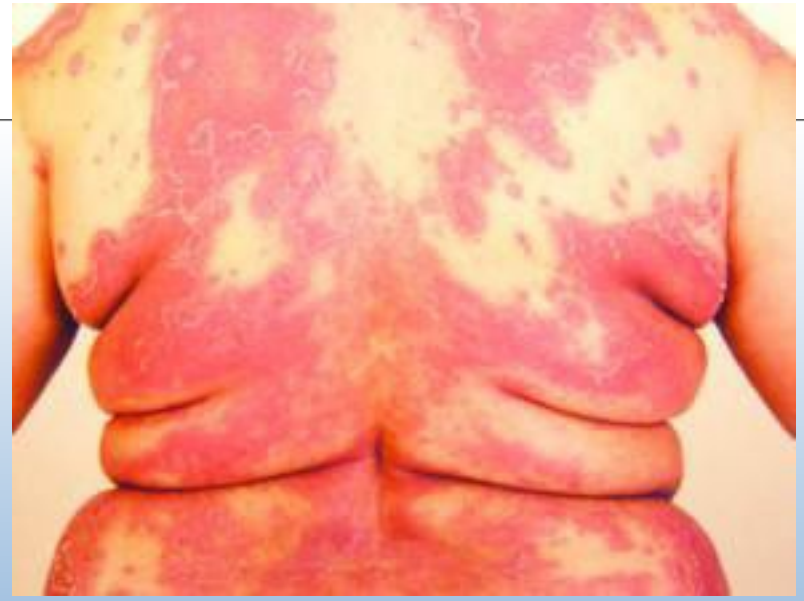
Total exposure: deucravacitinib, 969.0 patient-years; placebo, 240.9 patient-years; apremilast, 221.1 patient-years. Most placebo-related data were obtained during Weeks 0–16.
ATE, arterial thromboembolic events; MACE, major adverse cardiovascular events; NMSC, nonmelanoma skin cancers; PE, pulmonary embolism; VTE, venous thromboembolism.


COUSINS
MY NAME IS NOT JAK
JAK
TYK

Why to Target IL-36 in Inflammatory Disease

GPP (acute pustular psoriasis Von Zumbusch type) Is a Multisystemic Disease With a Relapsing/Remitting Clinical Course

- Acute GPP is characterized by **rapid onset of sterile pustules** occurring on **nonacral skin** and **not within psoriasis plaques**[1-2]
  - Pustules can coalesce to form "lakes of pus"
  - Repeated flares
- Can occur **with or without psoriasis vulgaris** or systemic inflammation[2]
- Associated symptoms can include fever, malaise, pain, and neutrophilic cholangitis[1]
- Systemic signs can include increased inflammatory marker levels and liver enzyme abnormalities[1]



Images reproduced with permission from DermNet NZ.

GPP, generalized pustular psoriasis.

1. Gooderham MJ et al. *Expert Rev Clin Immunol.* 2019; 15:907-919. 2. Navarini AA, et al. *J Eur Acad Dermatol Venereol.* 2017;31:1792; 4. Viguier M, et al. *Hepatology*. 2004;40(2):452-458.

GPP Is a Heterogeneous Disease That Lacks Consistent Classification[1]

- GPP was first described in 1910, yet clinical diagnosis criteria are still not consistent between expert centers and vary internationally[1]
- The rarity of pustular psoriasis and lack of consensus on diagnostic criteria are a challenge[2]
- Descriptions of GPP are inconsistent in dermatology textbooks due to the fact that it may occur in existence with plaque psoriasis[2]

Textbook Definitions of GPP[2]

GPP	Braun-Falco 6th edition 2005	Rook 9th edition 2016	Fitzpatrick 8th edition 2012	Saurat 3rd edition 2016	Baker/ Ryan 1968
Fever	+	+	+	+	+
Generalized pustules	+	+	+	+	+
Sterile pustules	+	+	+	+	+
Arthritis	–	(+)	–	(+)	(+)
Localization trunk	+	+	+	+	+
Localization intertriginous	+	+	–	?	(+)
Subtypes	2	4	4	5	4

GPP, generalized pustular psoriasis.

1. Boehner A, et al. *Exp Dermatol.* 2018;27:1067; 2. Navarini AA, et al. *J Eur Acad Dermatol Venereol.* 2017;31:1792.

In 2015, the JDA[a] Published Diagnostic Guidance on GPP

公益社団法人
日本皮膚科学会
Japanese Dermatological Association

GPP	
Primary parameters	• Systemic symptoms such as fever and fatigue • Systemic or extensive flush accompanied by multiple sterile pustules that sometimes merge to form lakes of pus • Neutrophilic subcorneal pustules histopathologically characterized by spongiform pustules of Kogoj • The above clinical and histological features recur repeatedly

A definitive diagnosis of GPP can be made in patients with all 4 features above, and GPP would be suspected in those with features 2 and 3

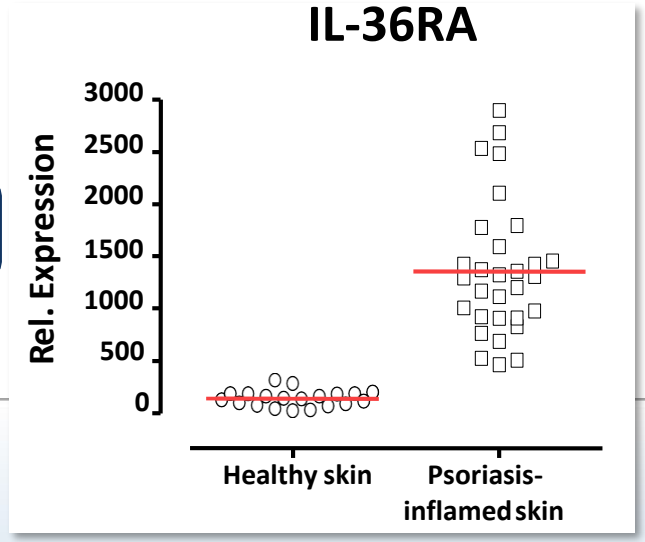
GPP, generalized pustular psoriasis; JDA, Japanese Dermatological Association.

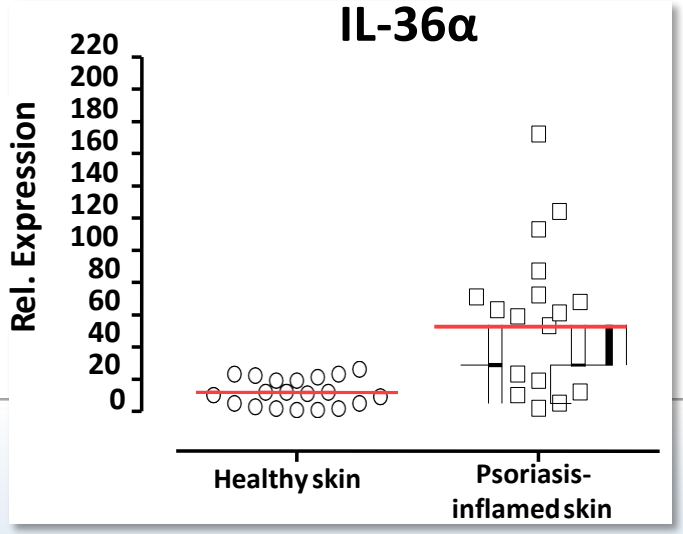
[a] The guidelines were founded as a collaborative project between the JDA and the Study Group for Rare Intractable Skin Diseases under the Ministry of Health, Labour, and Welfare Research Project on Overcoming Intractable Diseases and were first published in Japan in 2015.

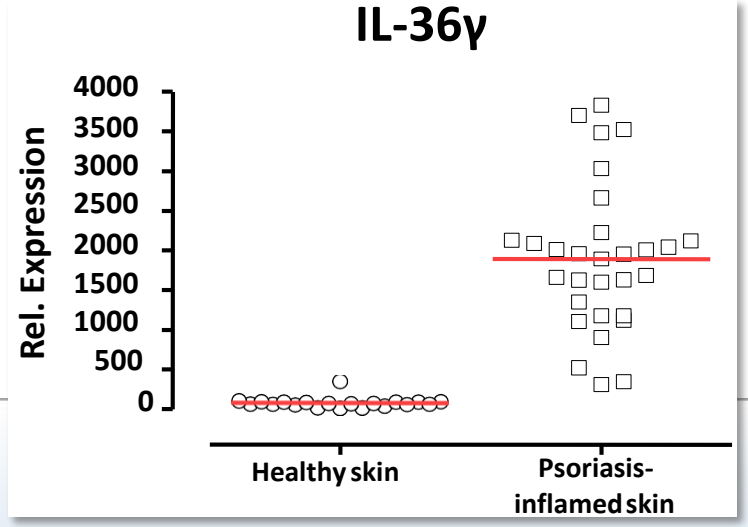
Fujita H, et al. *J Dermatol.* 2018;45:1235.

IL-36R Ligands Are Upregulated in Skin and Peripheral Blood of Patients With Psoriasis

Skin[1]

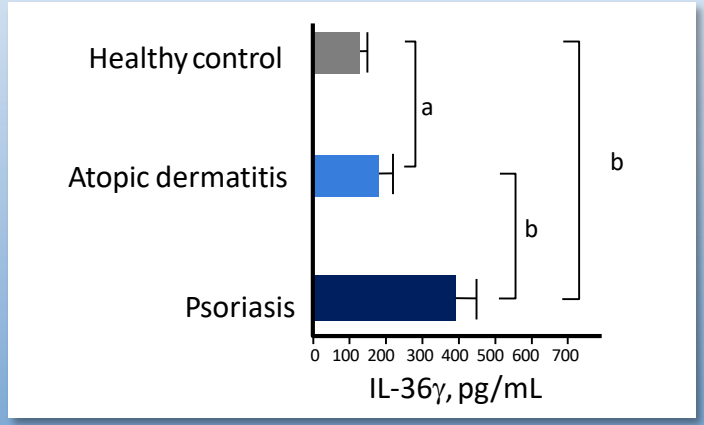


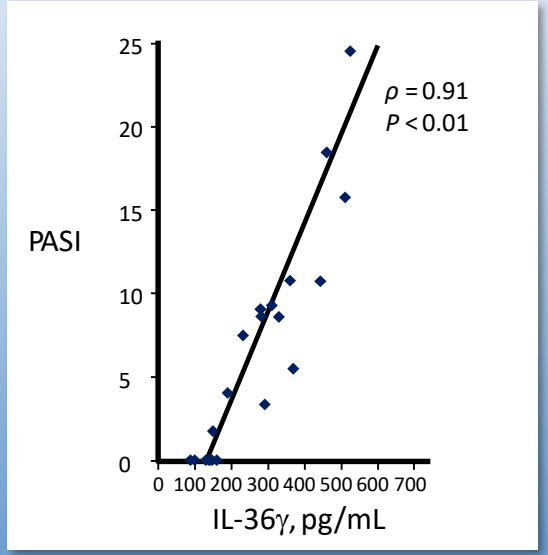




Blood[2]

IL-36γ upregulation in peripheral blood correlates with psoriasis severity





[a] $P < .05$; [b] $P < .01$

IL-36, interleukin 36; IL-36RA, IL-36 receptor antagonist; PASI, Psoriasis Area Severity Index.

1. Boehringer Ingelheim. Data on file (In-house gene chip analyses). 2. D'Erme AM, et al. *J Invest Dermatol*. 2015;135:1025.

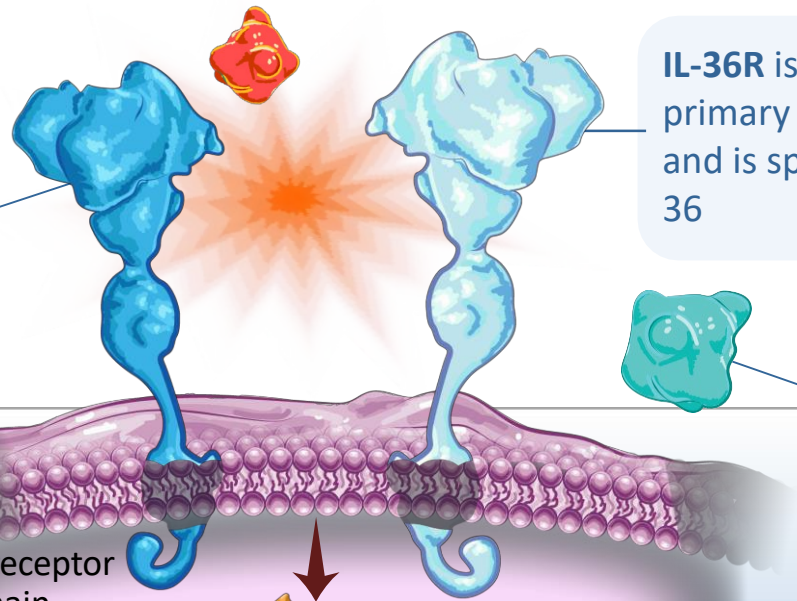
Normal IL-36 Pathway Mediates Appropriate Inflammatory Response[1-4]

There are 3 **IL-36** isoforms (α, β, γ)

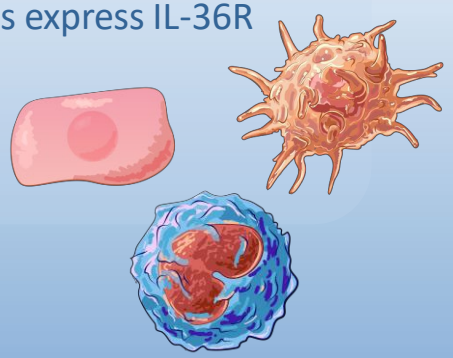
IL-1RAcP is a receptor accessory protein that is the co-receptor for IL-36R

IL-36R is the primary receptor and is specific to IL-36

IL-36RA is a receptor antagonist that blocks IL-36 from binding to IL-36R



Keratinocytes, monocytes, and myeloid DCs express IL-36R



Under normal conditions, IL-36 and IL-36RA take turns in regulating the pathway

Normal inflammatory response[1,2]

DC, dendritic cell; ERK, extracellular signal–regulated kinase; IL, interleukin; IL-1RAcP, IL-1 receptor accessory protein; IL-36R, IL-36 receptor; IL-36RA, interleukin 36 receptor antagonist; MAPK, mitogen-activated protein kinase; NF-κB, nuclear factor κ light-chain enhancer of activated B cells; TNF, tumor necrosis factor.

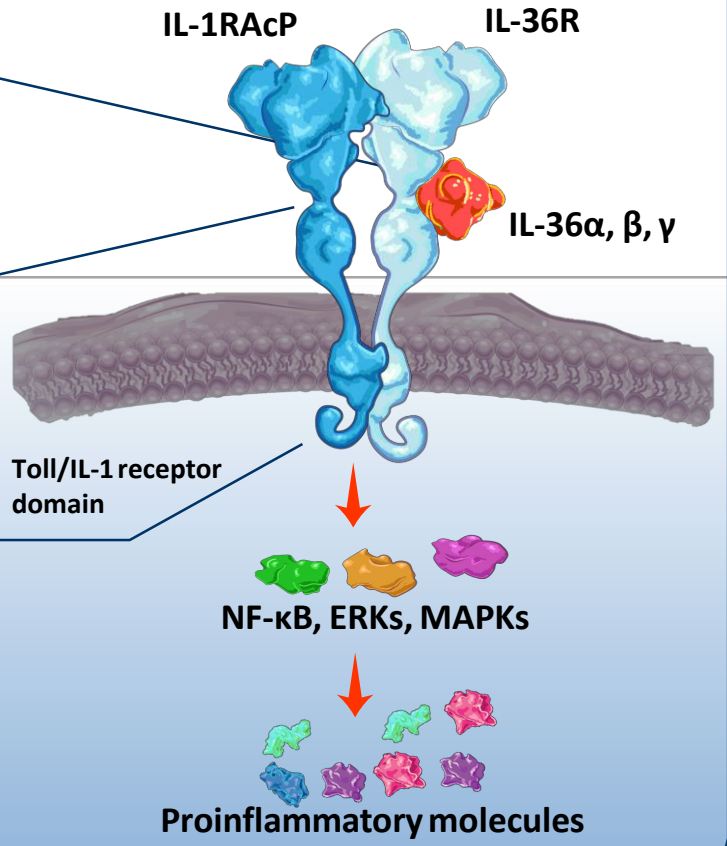
1. Marrakchi S, et al. *N Engl J Med*. 2011;365(7):620-628. 2. Bassoy EY, et al. *Immunol Rev*. 2018;281(1):169-178. 3. Gabay C, et al. *J Leukoc Biol*. 2015;97(4):645-652. 4. Furue K, et al. *Acta Derm Venereol*. 2018;98(1):5-13.

Under Normal Conditions, IL-36R Signaling Leads to a Balanced and Regulated Inflammatory Response[1-4]

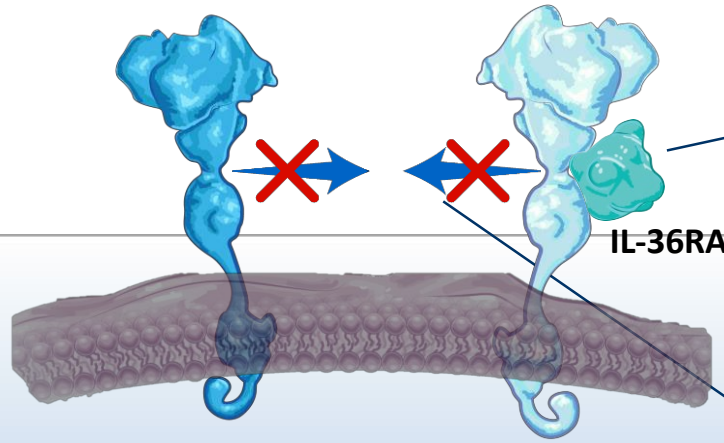
Agonist (IL-36) binding

1. **IL-36α, β, or γ** binds to IL-36R
2. IL-1RAcP (accessory protein) is recruited, forming a **heterodimeric signaling complex**
3. **TIR domains** are brought **together**, enabling signaling

Proinflammatory molecules include IL-1, IL-6, IL-12, IL-23



Antagonist (IL-36RA) binding



1. **IL-36RA** binds to IL-36R and prevents binding of IL-36α, β, or γ
2. **Heterodimeric signaling complex** is unable to form
3. **TIR domains** remain **separate** and unable to signal

No signaling

IL-36–regulated inflammatory signaling[1-4]

ERK, extracellular signal–regulated kinase; IL, interleukin; IL-1RAcP, IL-1 receptor accessory protein; IL-36R, IL-36 receptor; IL-36RA, IL-36 receptor antagonist; MAPK, mitogen-activated protein kinase; NF-κB, nuclear factor κ light chai -enhancer of activated B cells; TIR, Toll/IL-1 receptor.

1. Ganesan R, et al. *MAbs*. 2017;9:1143. 2. Bassoy EY, et al. *Immunol Rev*. 2018;281:169. 3. Gabay C, et al. *J Leukoc Biol.* 2015;97:645. 4. Marrakchi S, et al. *N Engl J Med*. 2011;365:620.

Imsidolimab, an Anti-IL-36 Receptor Monoclonal Antibody, in the Treatment of Generalized Pustular Psoriasis: Results from a Phase 2 Trial

Johann E. Gudjonsson[1], Adam Reich[2], Jonathan Barker[3], Andrew Pink[3], Nick J. Reynolds[4], Christopher E. M. Griffiths[5], Irina Khanskaya[6], Rupal Kalapanda[6], Jihao Zhou[6], Paul Lizzul[6], and Richard B. Warren[5]

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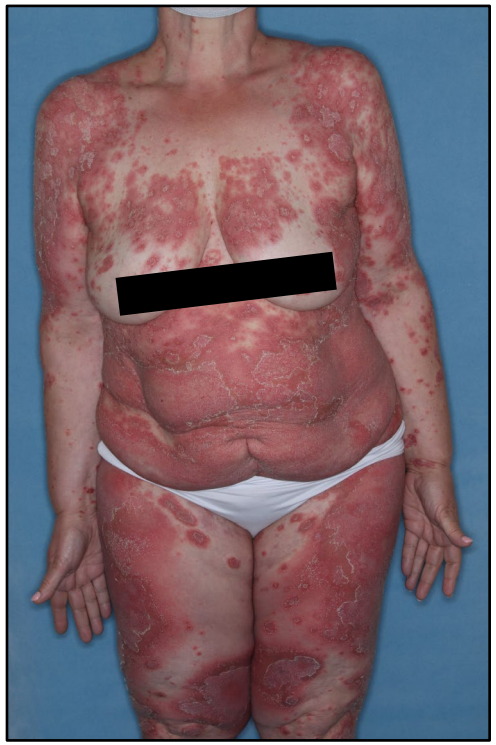
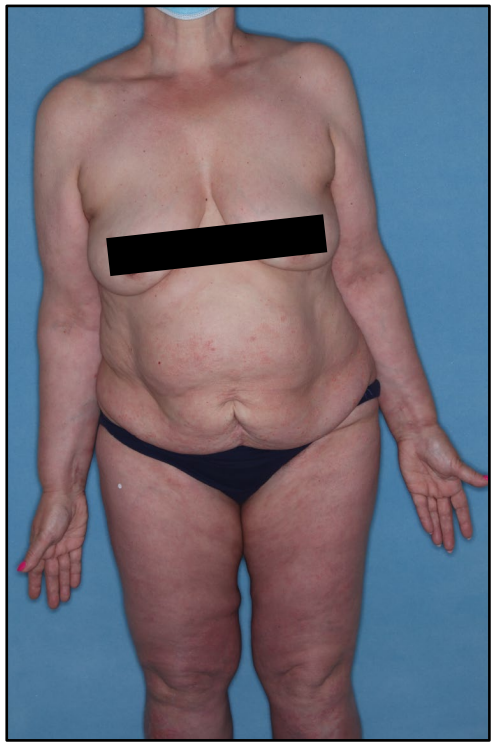
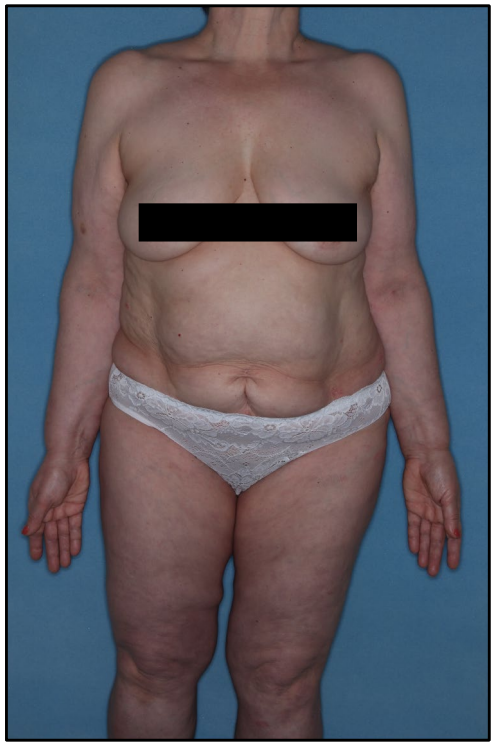
Fifty and Seventy-Five Percent of Subjects were GPPPGA *Clear* or *Almost Clear* at Weeks 4 and 16, Respectively

GPPPGA Responder Status[a]	Week 4	Week 16
Responder, n (%)		
0 (Clear)	0 (0.0)	1 (25.0)
1 (Almost Clear)	2 (50.0)	2 (50.0)
Total, n (%) (95% CI)	**2 (50.0) (6.76, 93.24)**	**3 (75.0) (19.41, 99.37)**
Non-Responder, n (%)		
2 (Mild)	2 (50.0)	1 (25.0)
3 (Moderate)	0 (0.0)	0 (0.0)
4 (Severe)	0 (0.0)	0 (0.0)
Total, n (%) (95% CI)	2 (50.0) (6.76, 93.24)	1 (25.0) (0.63, 80.59)
Total, n (%)	4 (100.0)	4 (100.0)

[a]Clinical response based on the GPP Physician Global Assessment (GPPPGA) scale; CI, confidence interval.

- The GPPPGA was implemented by protocol amendment after study start and only 4 subjects had assessments at Baseline, Week 4, and Week 16

Subject Photographic Evidence Consistent with Investigator Assessments of GPP Disease Severity

	Baseline	Week 4	Week 16
			
CGI	N/A	*Very Much Improved*	*Very Much Improved*
Area E/P	30	0	0
GPPPGA	4	1	1

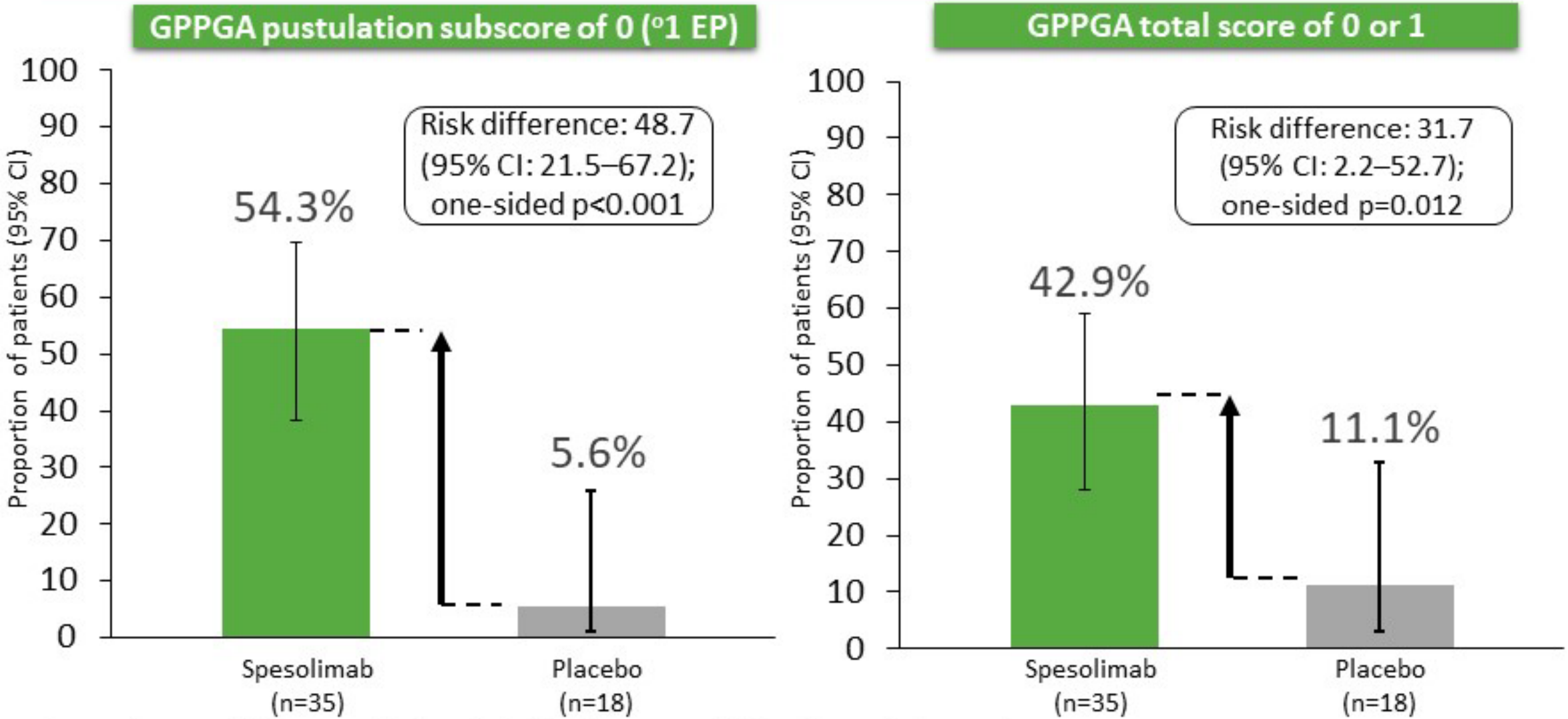
Area E/P, percent body surface area of erythema with pustules; CGI, Clinician Global Impression scale; GPPPGA, GPP Physician Global Assessment scale.

The NEW ENGLAND JOURNAL of MEDICINE

Trial of Spesolimab for Generalized Pustular Psoriasis

Hervé Bachelez, M.D., Ph.D., Siew-Eng Choon, F.R.C.P., Slaheddine Marrakchi, M.D., A. David Burden, M.D., Tsen-Fang Tsai, M.D., Akimichi Morita, M.D., Alexander A. Navarini, M.D., Ph.D., Min Zheng, M.D., Ph.D., Jinhua Xu, M.D., Ph.D., Hamida Turki, M.D., Milan J. Anadkat, M.D., Sushmita Rajeswari, M.Sc., et al., for the Effisayil 1 Trial Investigators*

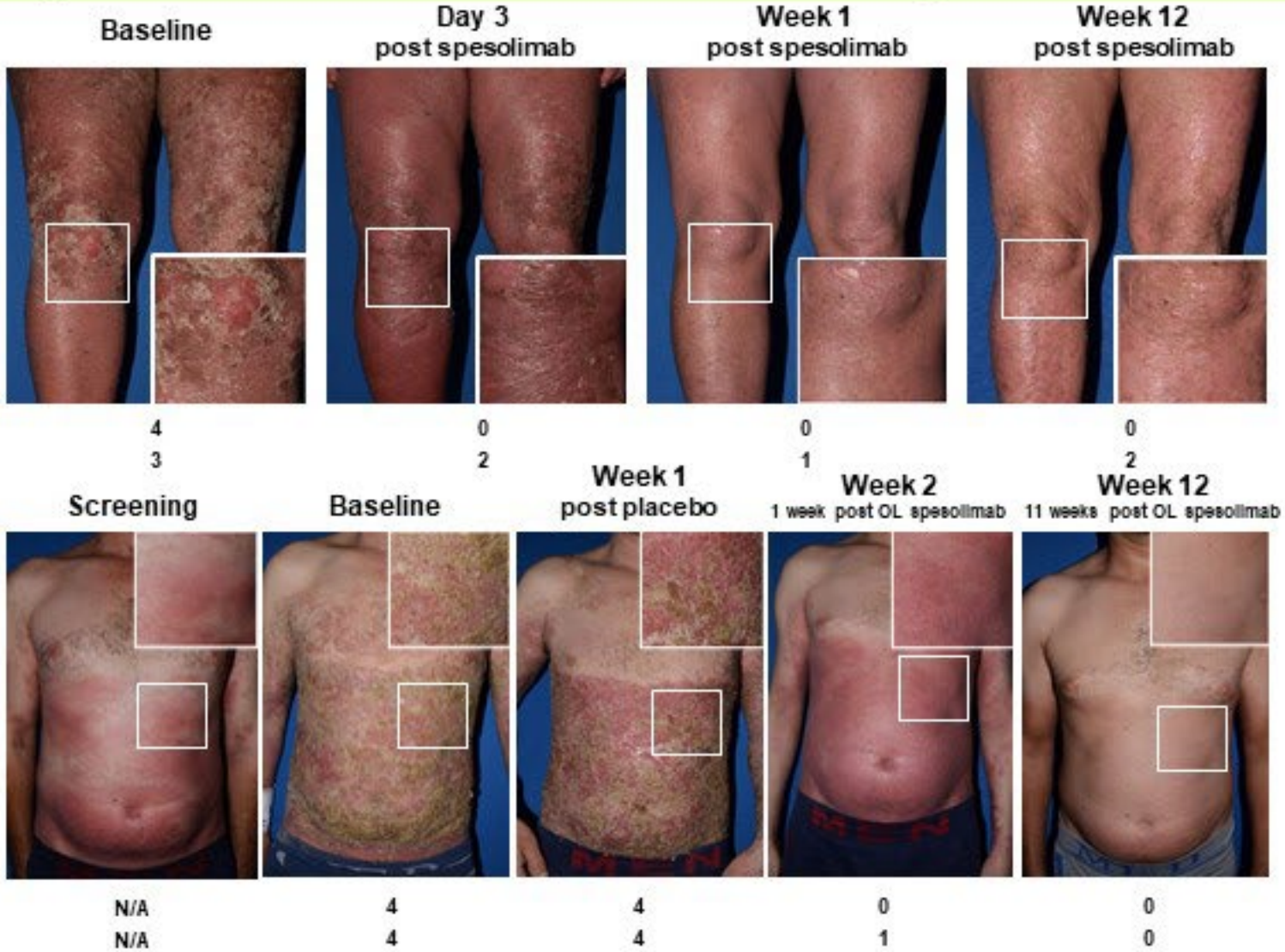
54% and 43% of patients with GPP receiving spesolimab had no visible pustules and clear/almost clear skin at Week 1, respectively



Any use of escape medication or open-label spesolimab at Day 8, or rescue medication with spesolimab represents non-response.

o1 EP, primary endpoint; CI, confidence interval; GPP, generalized pustular psoriasis; GPPGA, Generalized Pustular Psoriasis Physician Global Assessment.

Improvements in skin were observed within 1 week of receiving spesolimab and sustained through 12 weeks

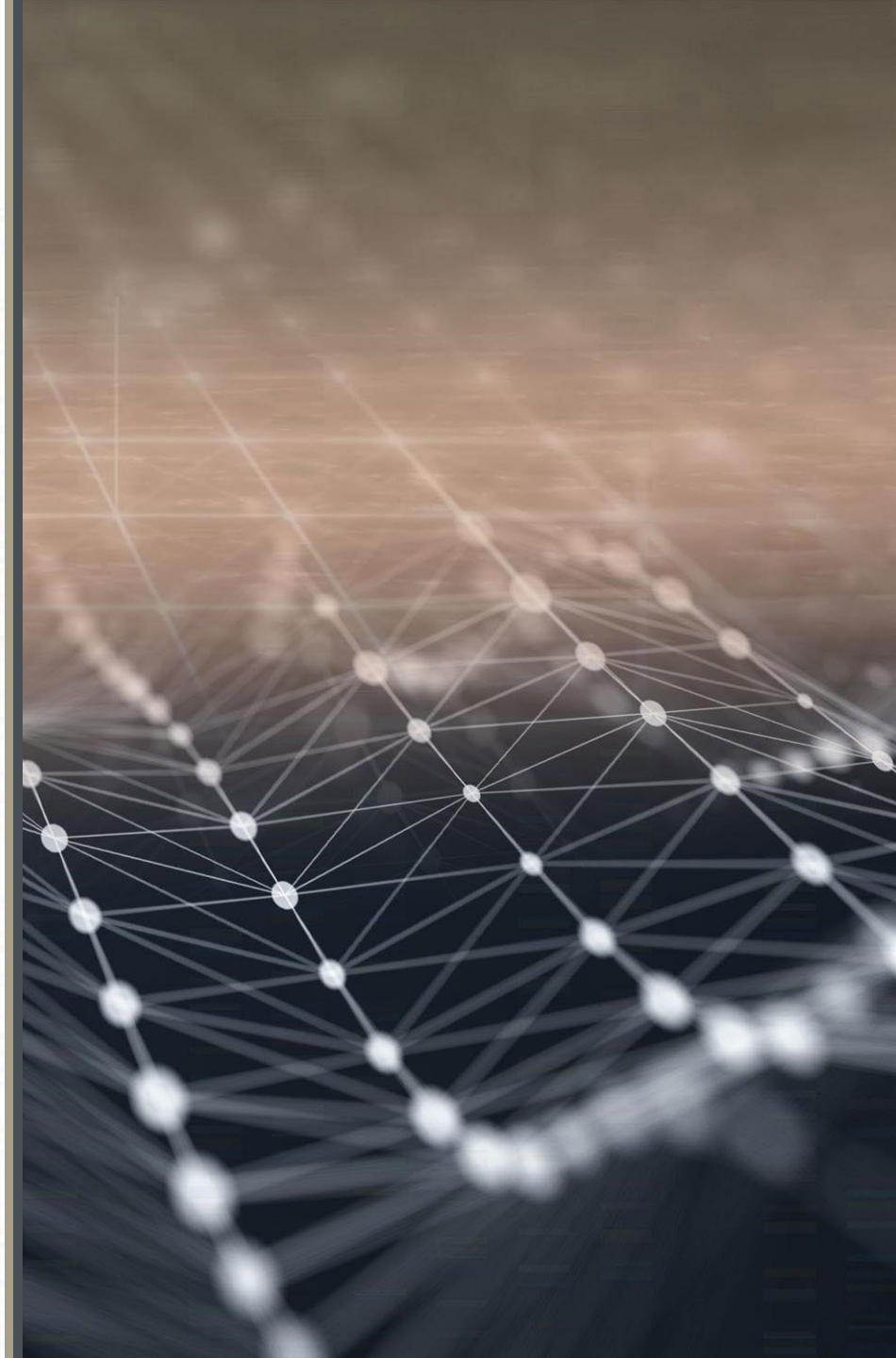


GPPGA, Generalized Pustular Psoriasis Physician Global Assessment; N/A, not applicable; OL, open-label.

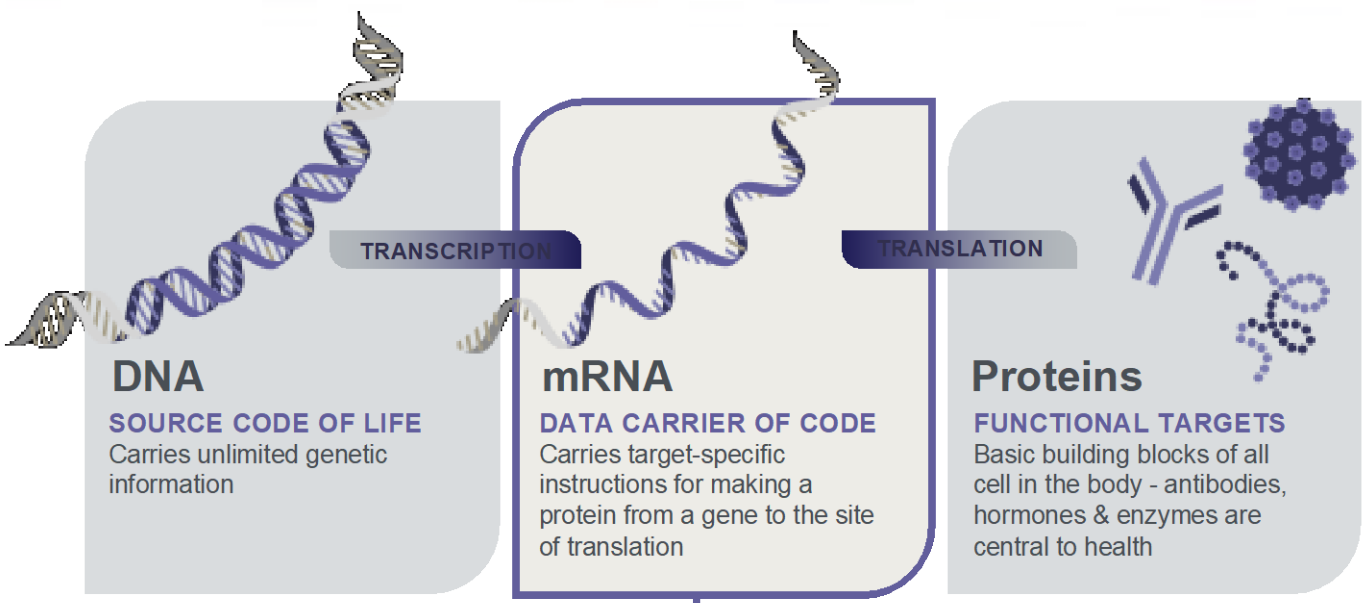
Precision Medicine…Realized

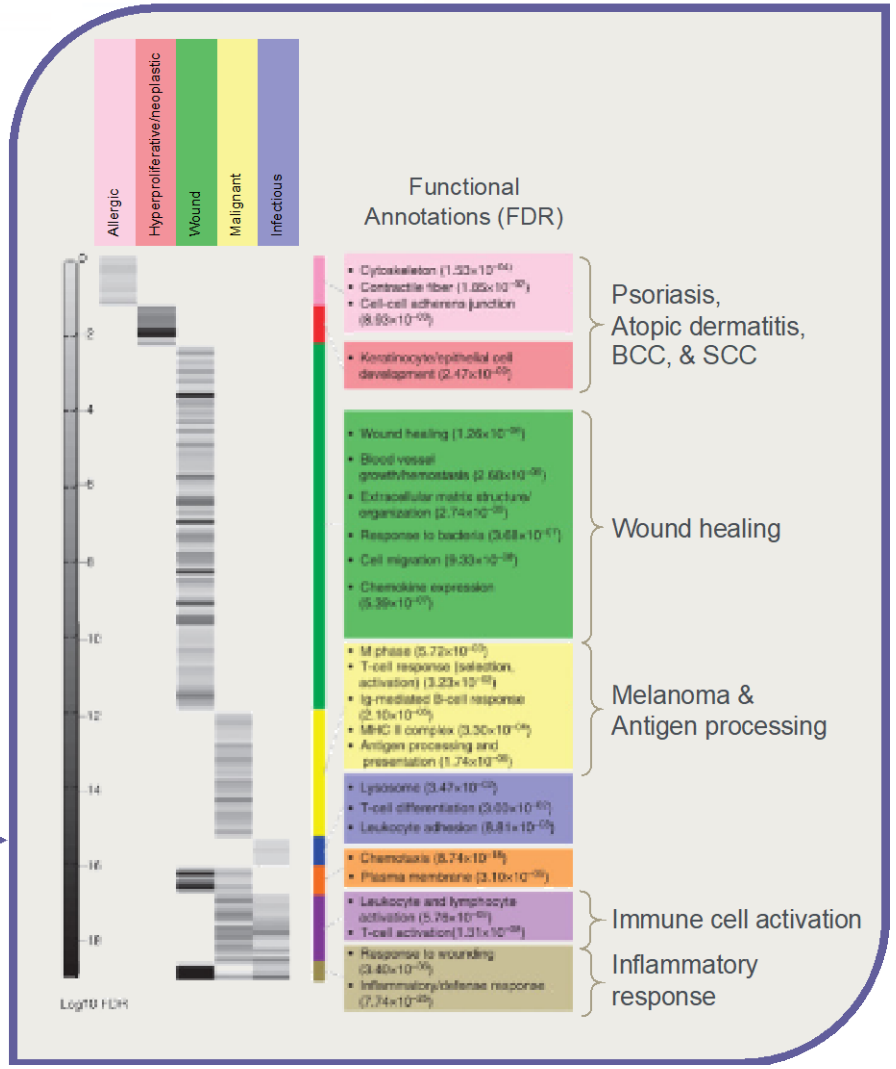
Predicts biologic drug response to confidently select the ideal therapy

December 2021



Dermal biomarkers enable precision medicine

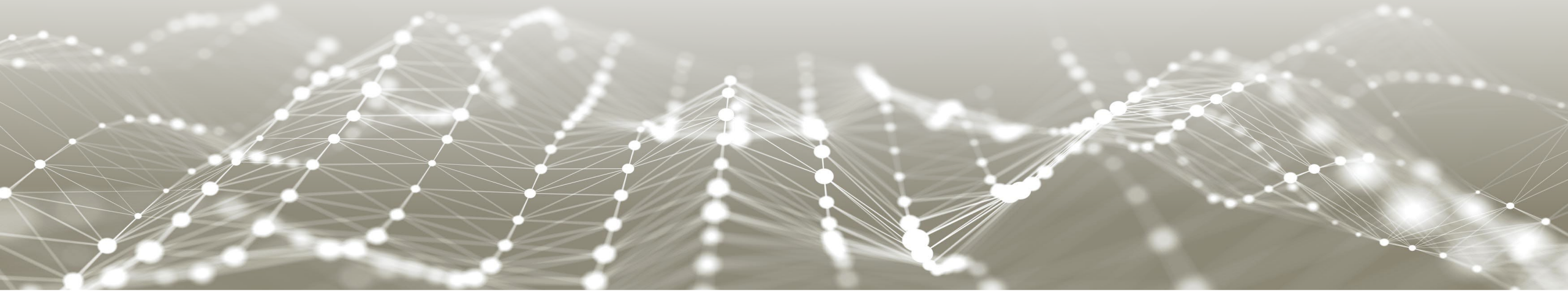




Source: Inkeles, MS, *et al. J Invest Dermatol* 2015, *135*, 151-159

AAD guidelines state, “There is… an important need to identify biomarkers that can potentially predict the appropriate biologic agent for individual patients.”

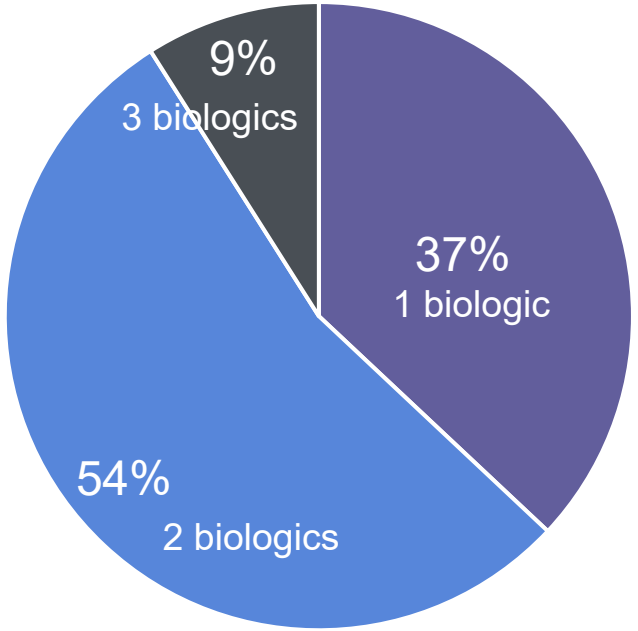
Menter A, Strober BE, Kaplan DH, et al. Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with biologics. JAAD. VOLUME 80, ISSUE 4, P1029-1072, APRIL 01, 2019.



Trial & Error Leads to Frequent Biologic Switching[1]

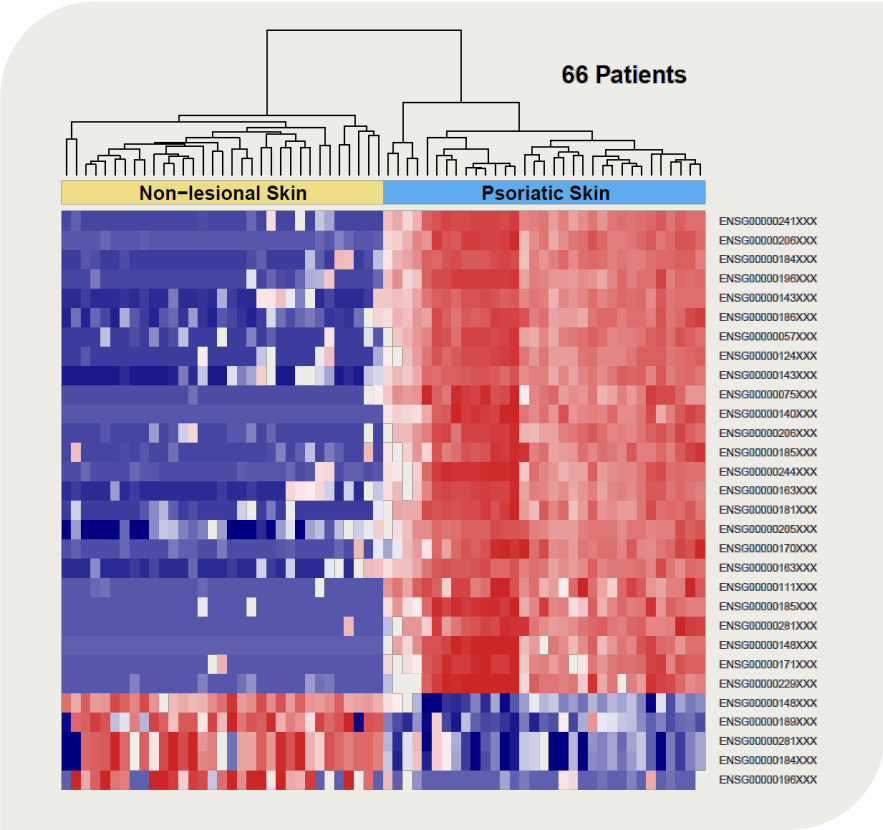
- 82% of dermatologists switch 10-30% of their patients in the first year
- 98% switch intra-class for at least 50% of non-responding patients
- Insurance formulary was compatible with first-line choice at least 75% of the time for only 14% of respondents

How many different biologics are typically needed to find the right biologic for the patient to achieve an adequate response?



[1]Strober, B., Pariser, D., Deren-Lewis, A. *et al.* A Survey of Community Dermatologists Reveals the Unnecessary Impact of Trial-and-Error Behavior on the Psoriasis Biologic Treatment Paradigm. *Dermatol Ther (Heidelb)* (2021).

Dermal Biomarker Patch Has Been Clinically Validated in Psoriasis Patients

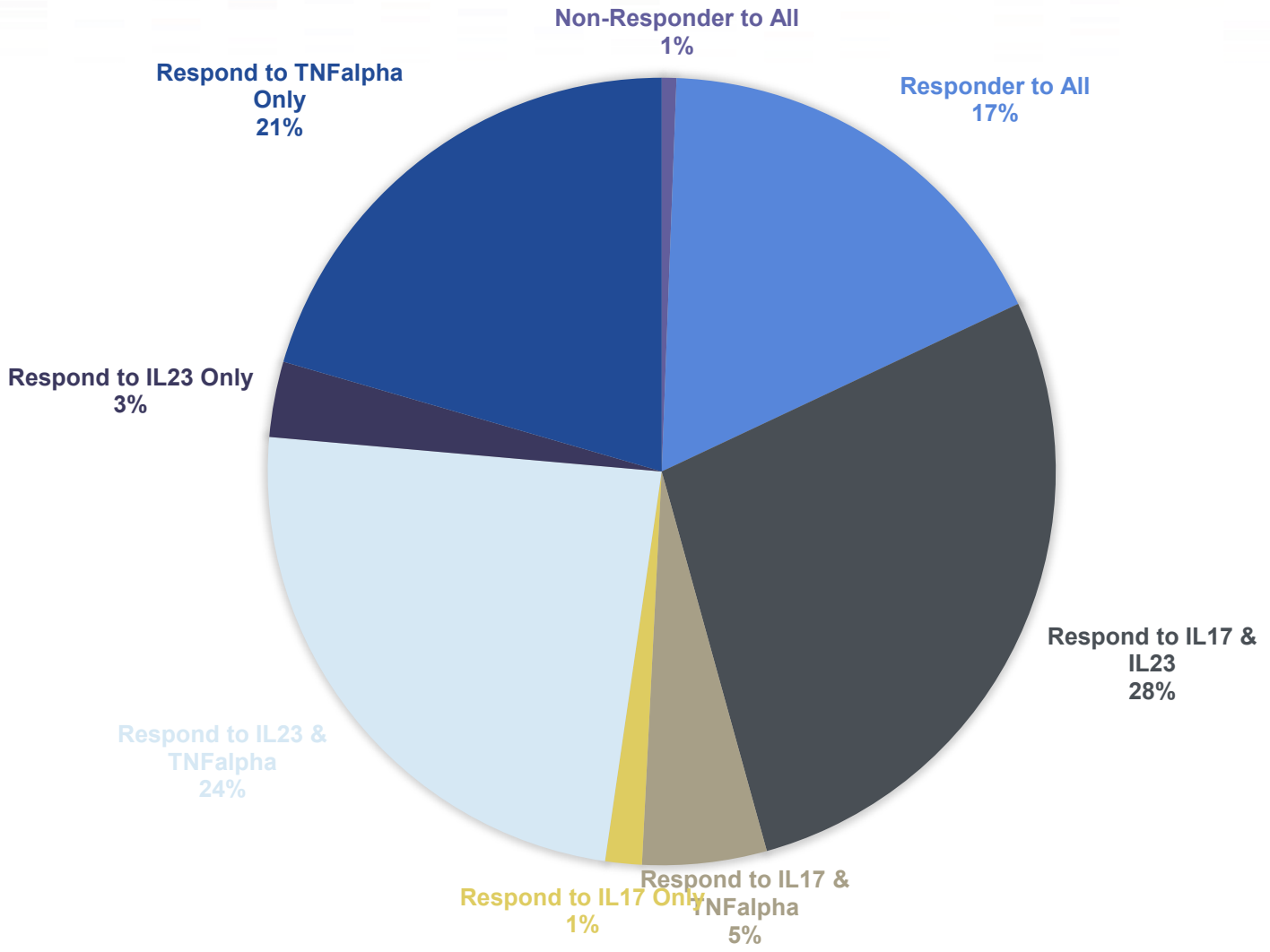


- Lesional vs. non-lesional skin sampled using dermal biomarker patches in **66 patients** and compared to punch biopsy
- Dermal biomarker patches and punch biopsy yielded **equivalent biomarker data**
- 30 highest variance genes selected & unsupervised clustering performed
- Excellent discrimination between **lesional** and **non-lesional** skin in same patient
- **Signature recapitulates known transcriptomic differences in psoriasis**

Patient Distribution

- Supplemental report is automatically triggered for triple negative patients (1 patient out of total cohort, N=242)[1]

- Supplemental report recommends a single biologic class to give a dermatologist guidance to the most likely drug class to show an effect



1. Bagel J, Wang Y, Montgomery III, P, et al. A Machine Learning-Based Test for Predicting Response to Psoriasis Biologics. *SKIN The Journal of Cutaneous Medicine*, 2021;5(6):621-638.

We are lucky to have new vehicles and new molecules in Dermatology.

HOWEVER…

IS MEDICAL PRACTICE CATCHING UP WITH MEDICAL INNOVATION?

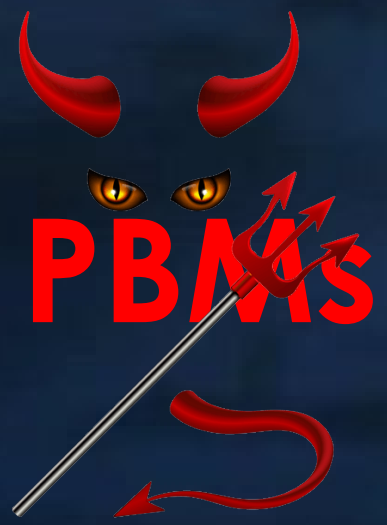
GENERICS

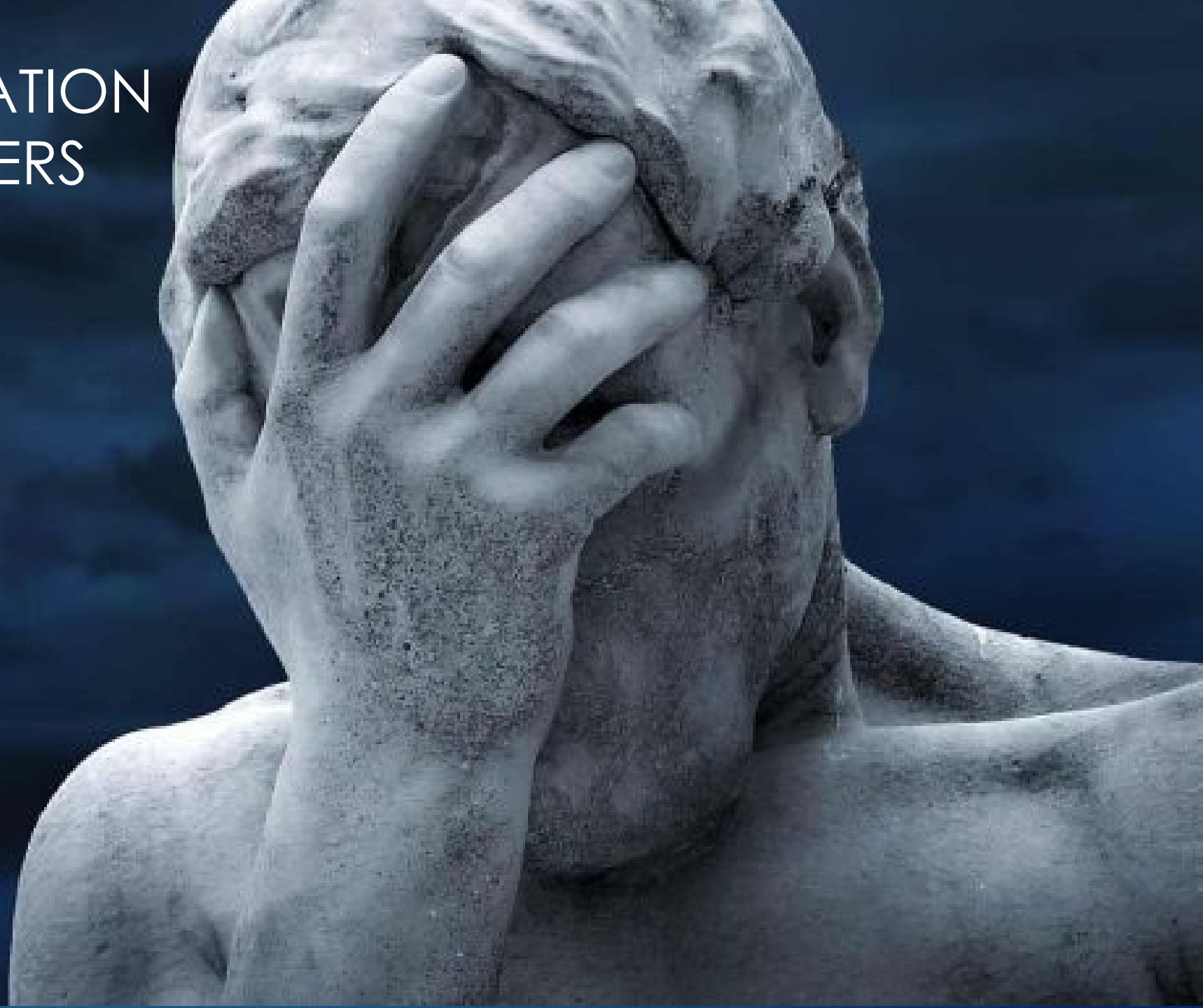
SUBSTITUTIONS

GENERICS

SUBSTITUTIONS

1.PRIOR AUTHORIZATION
2.THIRD PARTY PAYERS
3.DEDUCTIBLES
4.COPAYS





JAMA Dermatology | Original Investigation

Administrative Burden and Costs of Prior Authorizations in a Dermatology Department

Ryan P. Carlisle, BS; Nicholas D. Flint, BS; Zacha[illegible]H. Hopkins, M[illegible]Mark J[illegible]liason, MD; Kristina C. Duffin, MD, MS; Aaron M. Secrest, MD, PhD

IMP[illegible]CE [illegible]surance [illegible]omp[illegible]ies use prior authorizations (PAs) to address inappropriate pr[illegible]cribing or u[illegible]ary variations in care, most often for expensive medications. Prior aut[illegible]rizati[illegible]s negatively affect patient care and add costs and administrative burden to dermatology offices.


ROAD
CLOSED

IS THIS IS THE END OF R&D FOR DERM IN USA?

NATIONAL HEALTH SERVICE


FUTURE OF
DERMATOLOGY?


Thank You For Your Attention!
PRIVATE
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NO TRESPASSING

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