BTLA Agonist (ANB032) R&D Event

May 25, 2023

1:15pm PT / 4:15pm ET

AnaptysBio

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This presentation and any accompanying oral presentation contains forward-looking statements within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995, including, but not limited to: the timing of initiation of the Company's clinical trials, including rosnilimab's clinical trials in rheumatoid arthritis and in a second indication; the timing of the release of data from the Company's clinical trials, including imsidolimab's Phase 3 clinical trial in GPP, rosnilimab's Phase 2b clinical trial in rheumatoid arthritis and ANB032's Phase 2b clinical trial in atopic dermatitis; the timing of ANB033's IND filing; statements regarding efficacy, safety and proof of mechanism from blinded data from rosnilimab's clinical trial in alopecia areata and the timing of release of additional data from this trial; the Company's ability to find a licensing partner for imsidolimab or etokimab and the timing of any such transaction; and the Company's projected cash runway. Statements including words such as "plan," "continue," "expect," or "ongoing" and statements in the future tense are forward-looking statements. These forward-looking statements involve risks and uncertainties, as well as assumptions, which, if they do not fully materialize or prove incorrect, could cause its results to differ materially from those expressed or implied by such forward-looking statements. Forward-looking statements are subject to risks and uncertainties that may cause the company's actual activities or results to differ significantly from those expressed in any forward-looking statement, including risks and uncertainties related to the company's ability to advance its product candidates, obtain regulatory approval of and ultimately commercialize its product candidates, the timing and results of preclinical and clinical trials, the company's ability to fund development activities and achieve development goals, the company's ability to protect intellectual property and other risks and uncertainties described under the heading "Risk Factors" in documents the company files from time to time with the Securities and Exchange Commission. These forward-looking statements speak only as of the date of this presentation, and the company undertakes no obligation to revise or update any forward-looking statements to reflect events or circumstances after the date hereof.

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Agenda for today

TOPIC	SPEAKER
AnaptysBio: Best-In-Class Immune Cell Modulators	Dan Faga Chief Executive Officer
Checkpoint Agonism and ANB032, a BTLA Agonist	Martin Dahl, Ph.D. Senior Vice President, Research
Unmet Needs in Atopic Dermatitis (AD) and Need for Additional Targets	Emma Guttman, M.D., Ph.D. Professor and Chair of Dermatology Icahn School of Medicine, Mount Sinai
Targeting AD with ANB032: Translational Data	Martin Dahl, Ph.D. Senior Vice President, Research
ANB032: Phase 1 Results and AD Phase 2b Trial	Paul Lizzul, M.D., Ph.D. Chief Medical Officer
Closing Remarks	Dan Faga Chief Executive Officer
Q&A	AnaptysBio

Best-in-class immune cell modulating antibodies restore immune balance across autoimmune & inflammatory diseases

Immune Cell Modulators

Rosnilimab (PD-1 agonist)

Phase 2b in Rheumatoid Arthritis

ANB032 (BTLA agonist)

Phase 2b in Atopic Dermatitis

ANB033 (CD122 antagonist)

IND-enabling

Cytokine Antagonists (legacy programs for out-licensing)

Imsidolimab
(IL-36R)
Phase 3 in GPP

Etokimab
(IL-33)
Phase 2b/3-ready in epithelial driven diseases

Research-driven

Preclinical pipeline of immunology targets

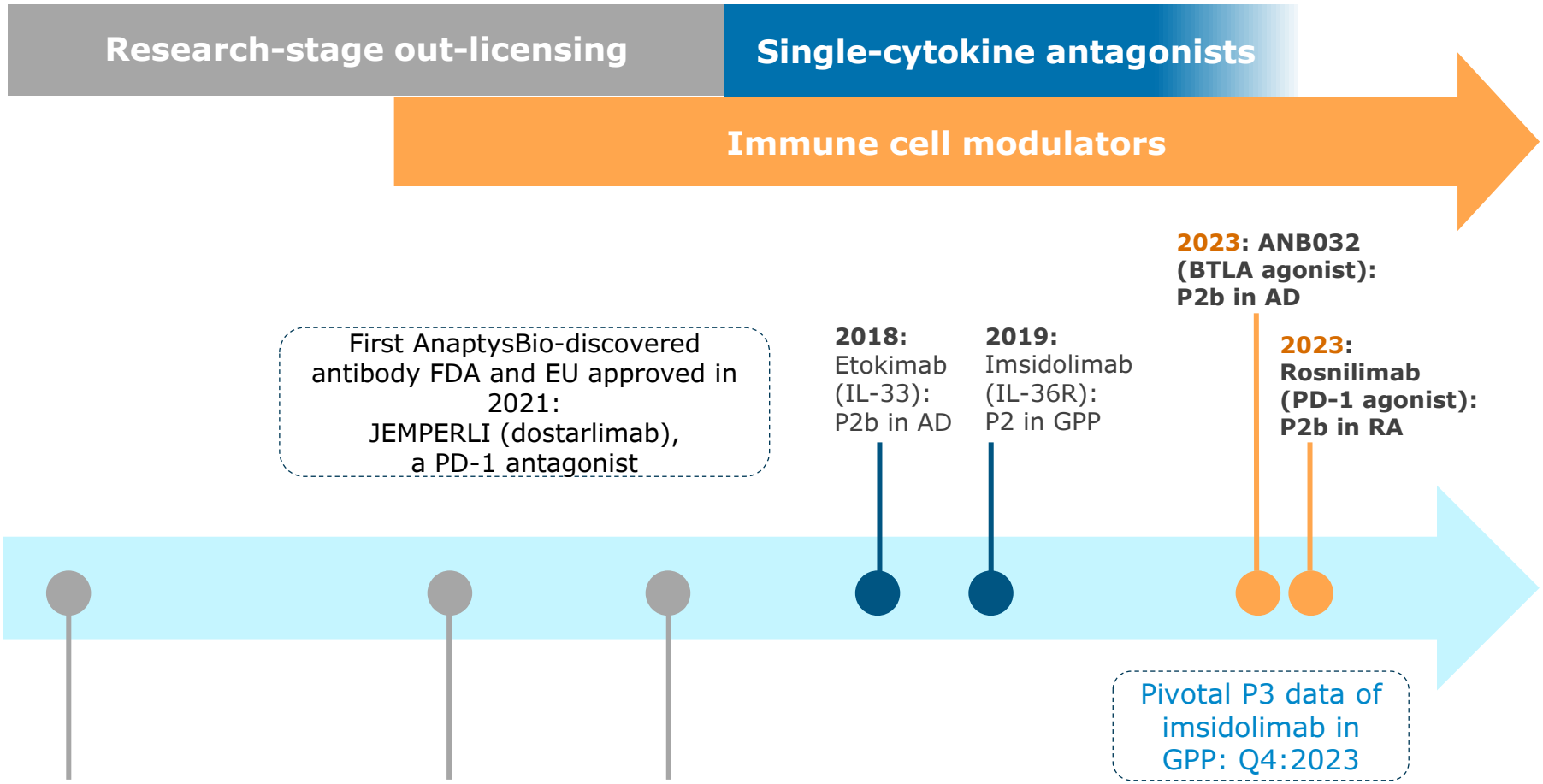
Strong capital position

Cash runway to YE 2026

GSK immuno-oncology financial collaboration

Significant royalty potential

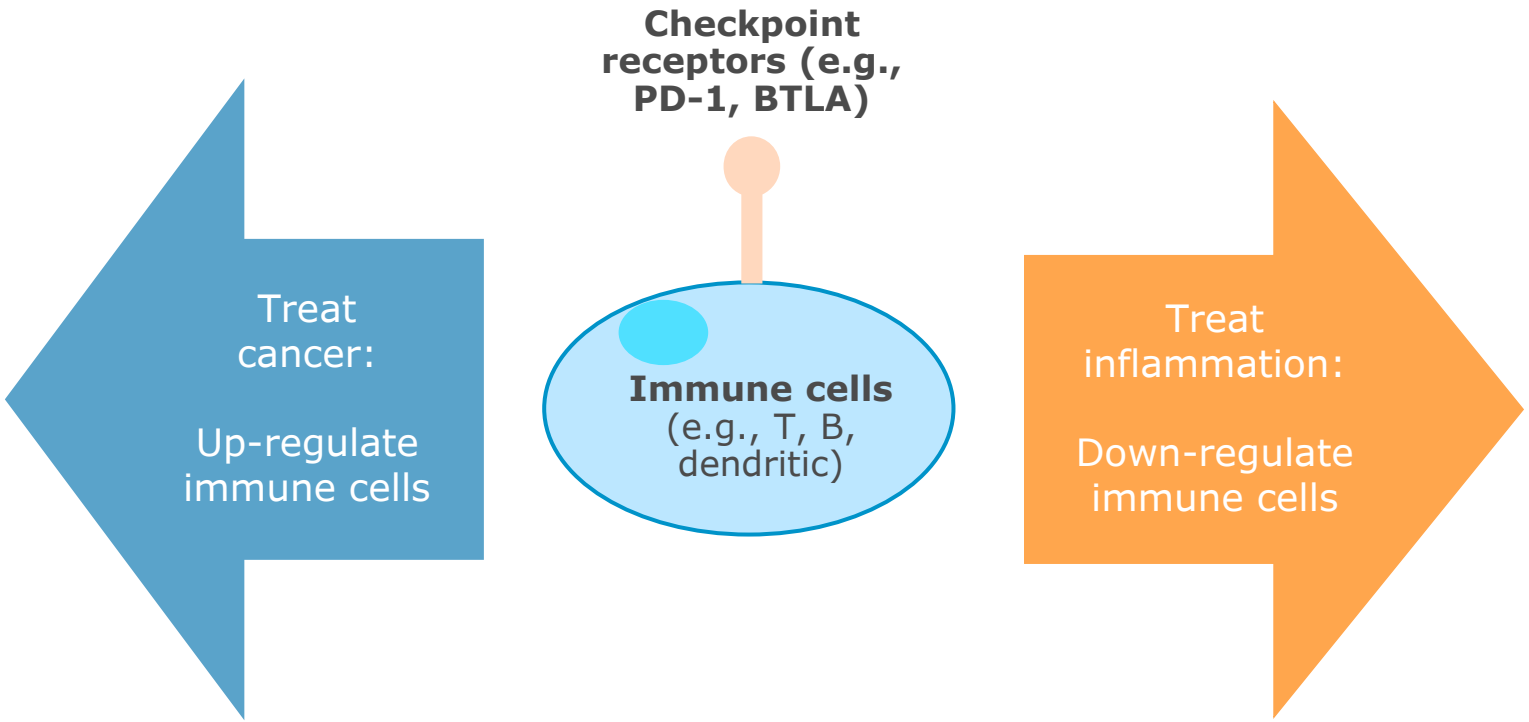
Two decades of leadership in antibody discovery



Checkpoint agonists restore immune balance

Checkpoint antagonists:
"release the brakes"

Checkpoint agonists:
"hit the brakes"



ANB032 has potential to treat wide range of systemic inflammatory diseases*

BTLA agonists modulate immune cells:
inhibit activated T cell proliferation, reduce inflammatory cytokine secretion and modulate DC function including inducing Tregs

Th1 cell | Th2 cell | Th17 cell | Th22 cell | Dendritic cell (DC) | Treg | B cell

Atopic dermatitis:

*>$16 billion global sales by 2030***

AD market to grow significantly given unmet patient need

Th1, Th2, Th17, Th22 and DCs drive atopic dermatitis pathogenesis

- SOC only directly targets Th2 pathway

Expect ANB032 to drive deeper responses across broader patient population

- Restore immune balance

* Therapeutic area classes include dermatology, rheumatology, gastroenterology, metabolic, neurology and respiratory. ** GlobalData, AD Global Drug Forecast and Analysis, 2030.

ANB032's mechanism of action matches atopic dermatitis disease pathogenesis

- Checkpoint agonism may deliver differentiated outcomes while restoring immune balance in inflammatory and autoimmune diseases
- BTLA agonism inhibits activated T cell proliferation, reduces inflammatory cytokine secretion and modulates DC function including induction of Tregs
- Th1, Th2, Th17, Th22 and dendritic cells drive AD pathogenesis
- ANB032, supported with translational preclinical and Phase 1 safety data, has potential for deep responses across a broad patient population in AD
- Global Phase 2b trial initiated with top-line data expected by year-end 2024

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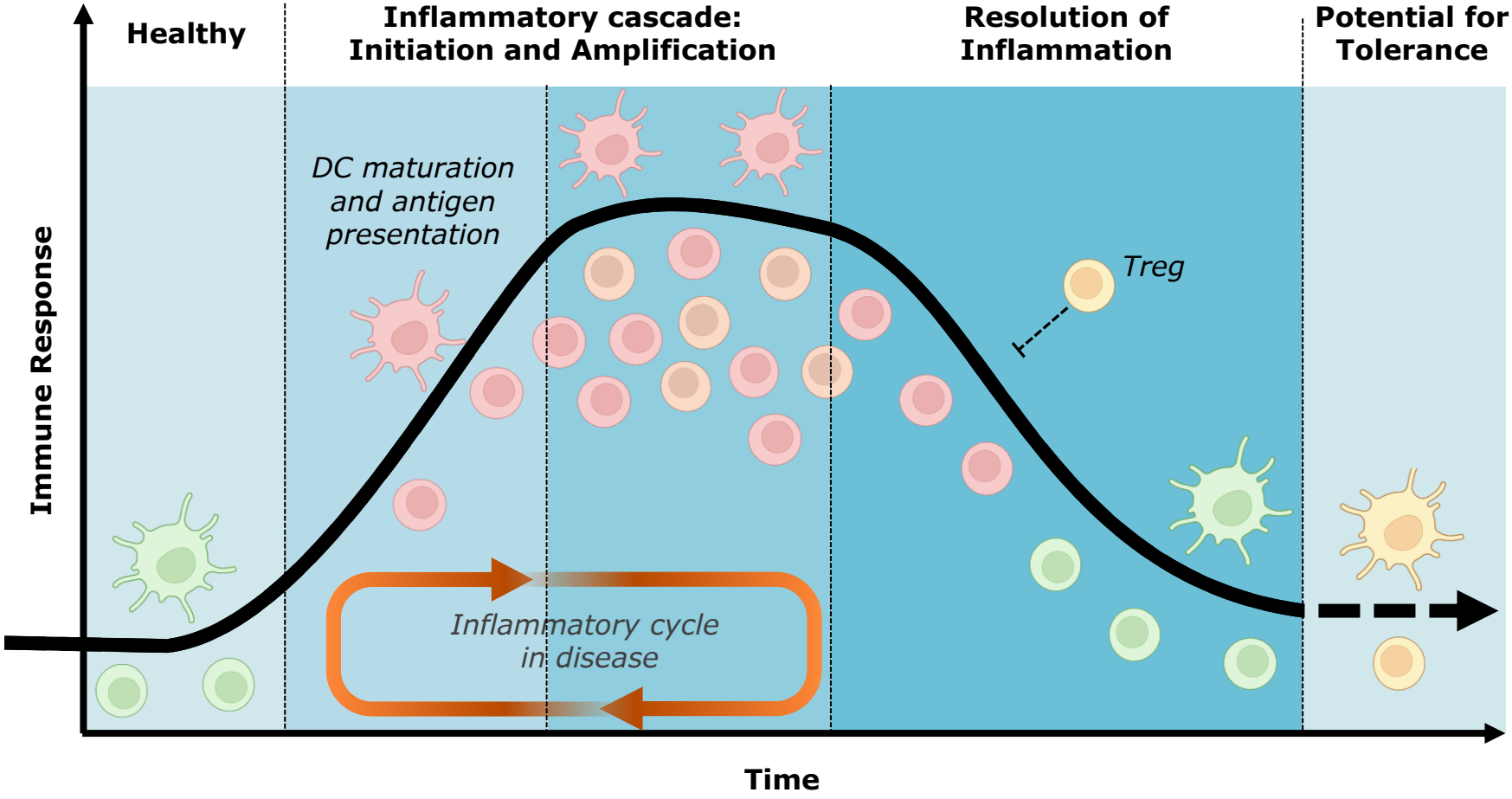
Modulating immune cells by targeting checkpoint receptors may restore immune balance



Checkpoint agonists can restore immune balance to deliver differentiated outcomes

Efficacy

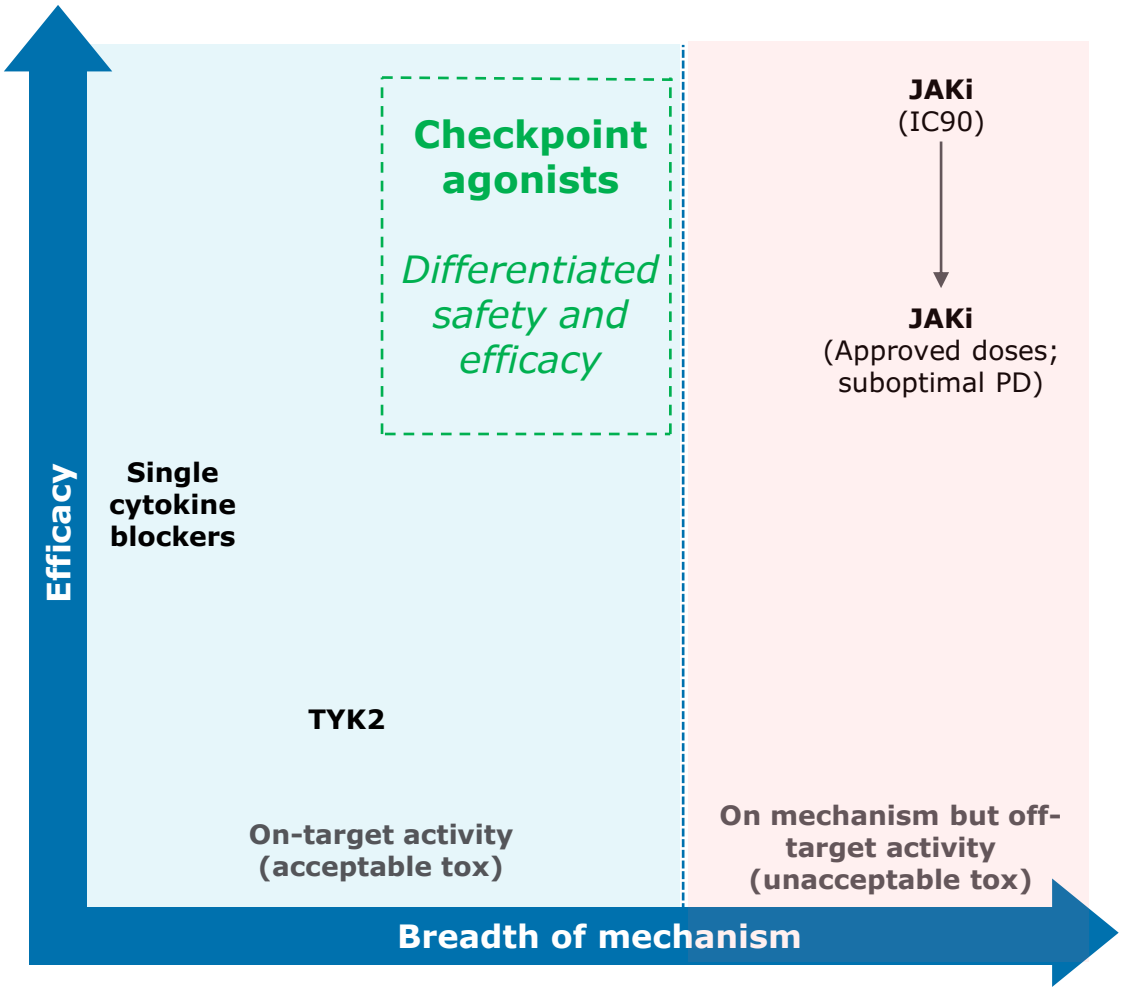
- Inhibits activated immune cells, acting directly on dysregulation
- Deep responses across broad patient population

Durability

- Restoring immune balance

Safety

- Agonist class well-tolerated to date
- No evidence of carcinogenicity with T cell modulators, such as abatacept, in decades of use



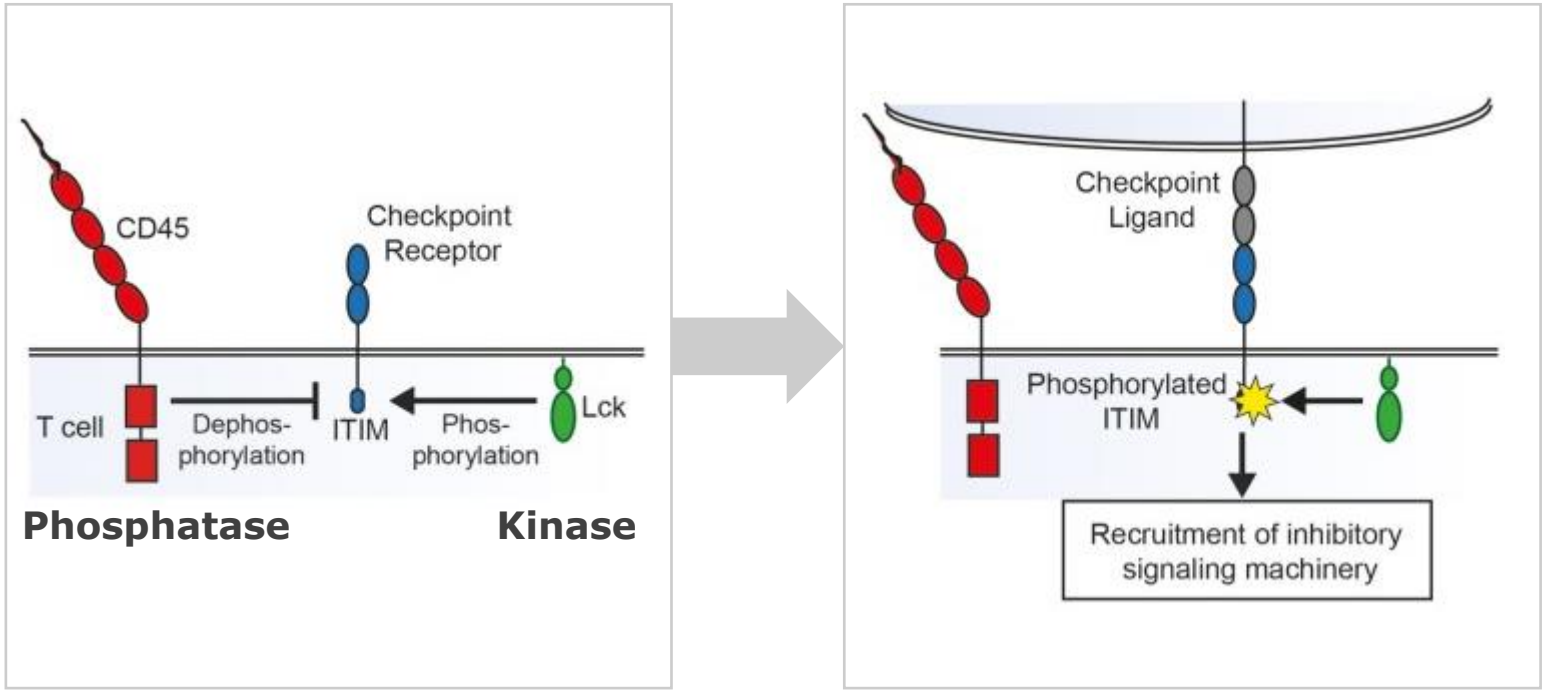
Ligands on opposing cells bind to receptors, forming tight synapses to initiate checkpoint agonism

Checkpoint receptors on immune cells:

- regulated by kinases (on) and phosphatases (off)

Checkpoint ligands on opposing cells:

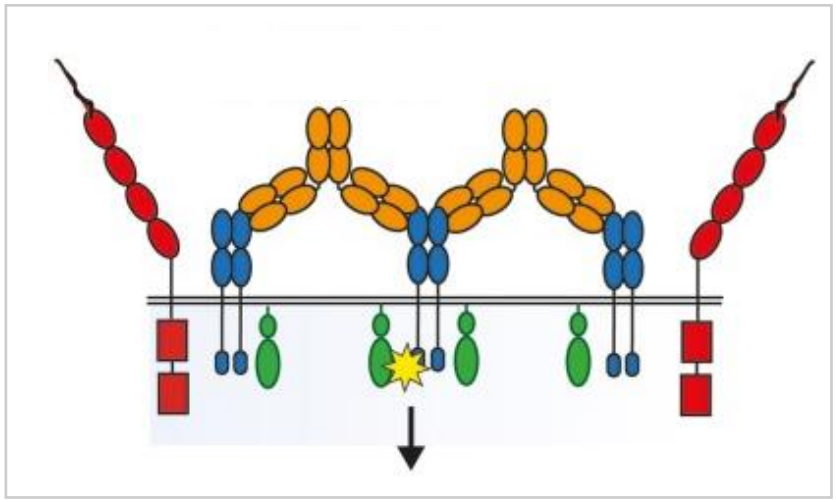
- engage initiating kinases
- exclude phosphatases



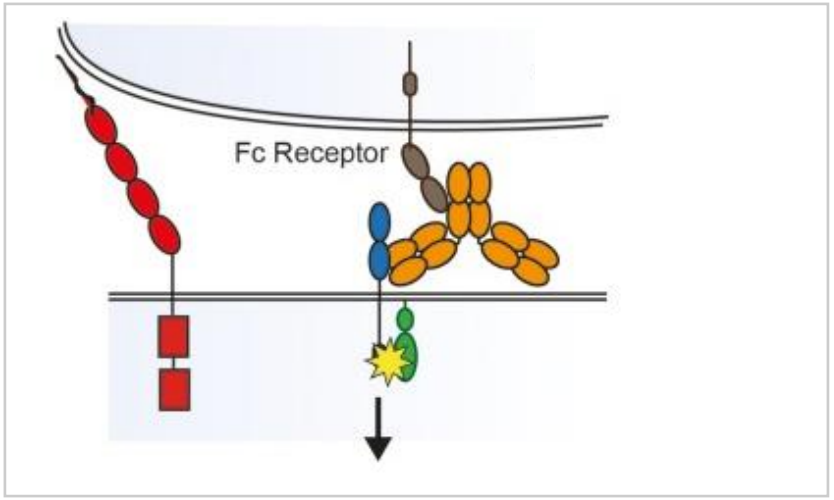
* Figure adapted from Frontiers in Immunology. 2018 Oct 8; 9: 2306; Immunoreceptor tyrosine-based inhibitory motif (ITIM)

AnaptysBio's checkpoint agonists optimize inhibitory signaling by enabling tight immune synapse formation

Fc independent
checkpoint receptor agonism

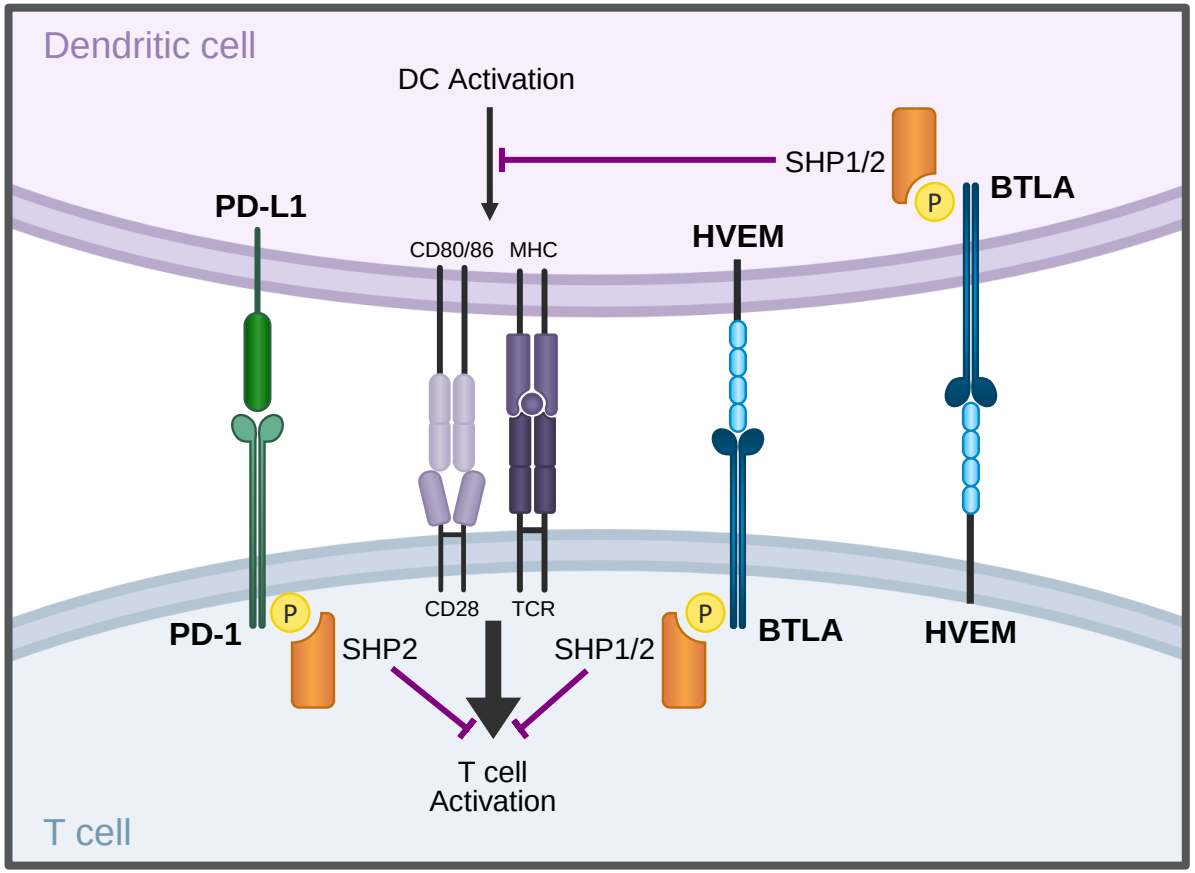


Fc receptor binding affinity AND
membrane proximal binding optimizes
agonism



* Figure adapted from Frontiers in Immunology. 2018 Oct 8; 9: 2306

BTLA is key node of immune regulation

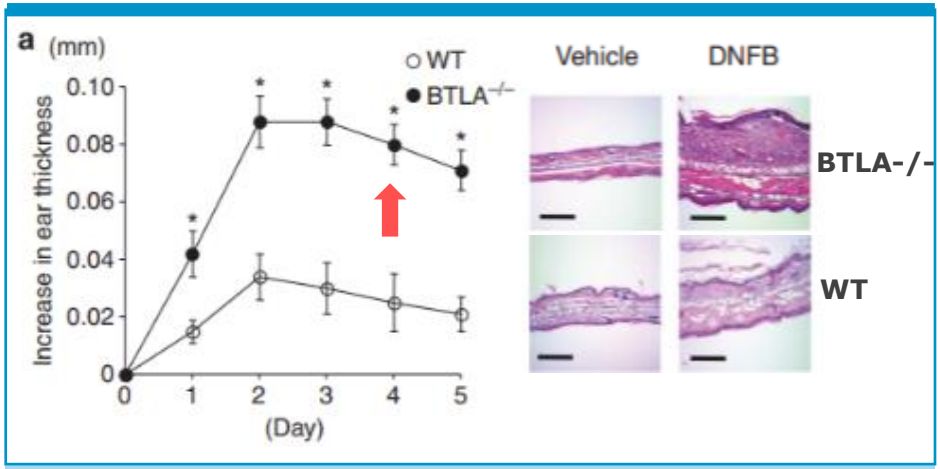


- B and T lymphocyte attenuator (BTLA) is a potent modulator of T cells, B cells and dendritic cells (DC)
- Expressed only on immune cells and preferentially on activated immune cells
- Dysregulation of BTLA pathway accelerates onset and exacerbates disease

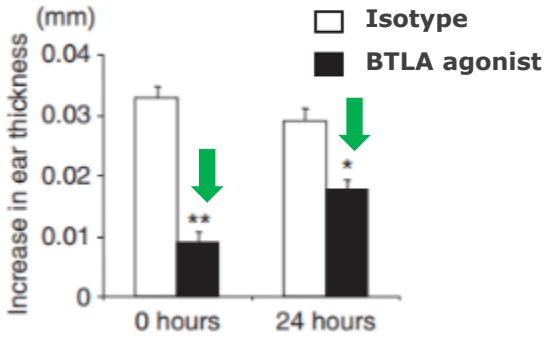
Adapted from Xu et al, J. Cell Biol. 2020 Vol. 219 No. 6

Proof of mechanism: Agonism of BTLA pathway in dermatitis model

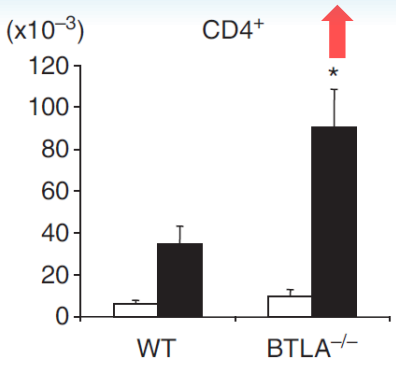
BTLA knock-out mice have exacerbated T cell-mediated skin disease



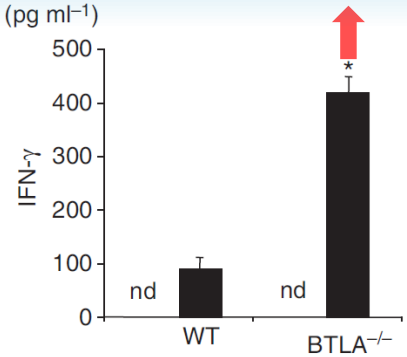
BTLA agonist-treated WT mice have reduced T cell-mediated skin disease



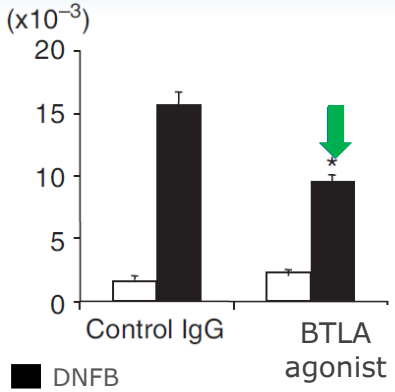
Infiltrating CD4 T cells (similar for CD8)



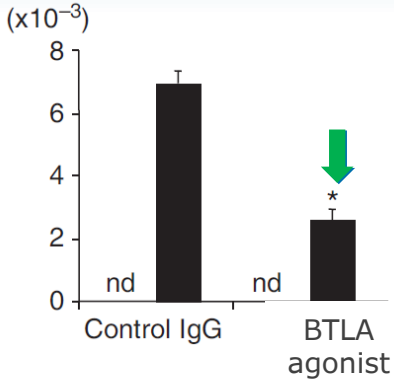
Proliferating CD8 T cell IFNγ



Infiltrating CD4 T cells (similar for CD8)



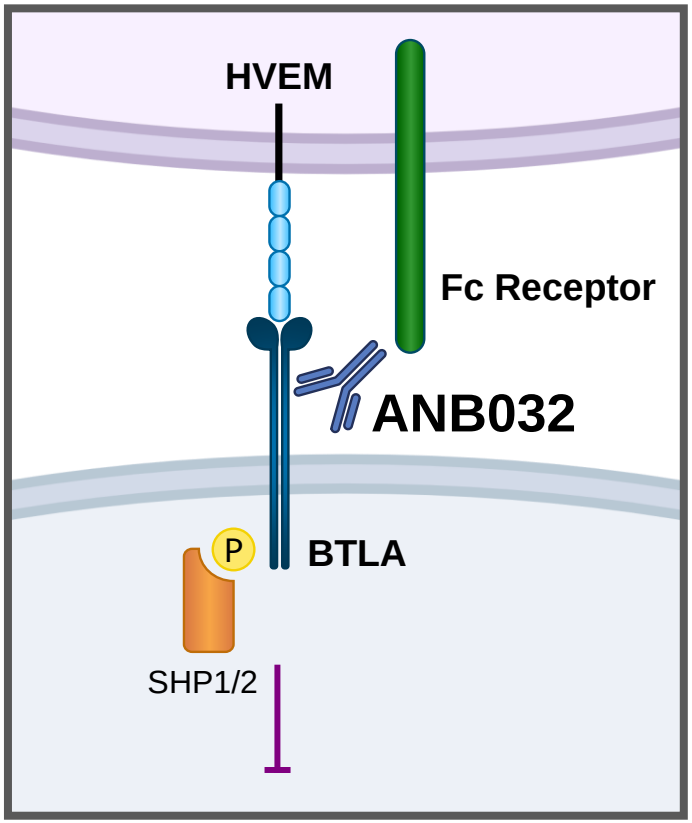
Infiltrating CD8 T cell IFNγ



Vehicle DNFB

Nakagomi et al, Journal of Investigative Dermatology, 2013; Surrogate Murine BTLA agonist third-party data

ANB032: Best-in-class BTLA agonist antibody



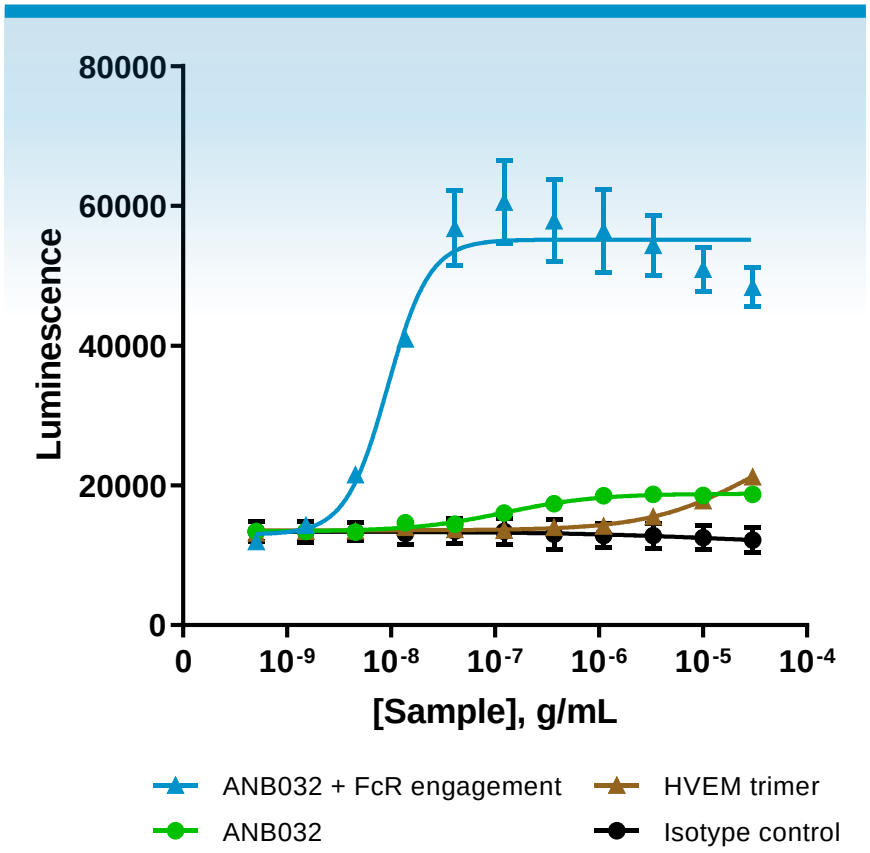
ANB032: IgG4 antibody (non-depleting)

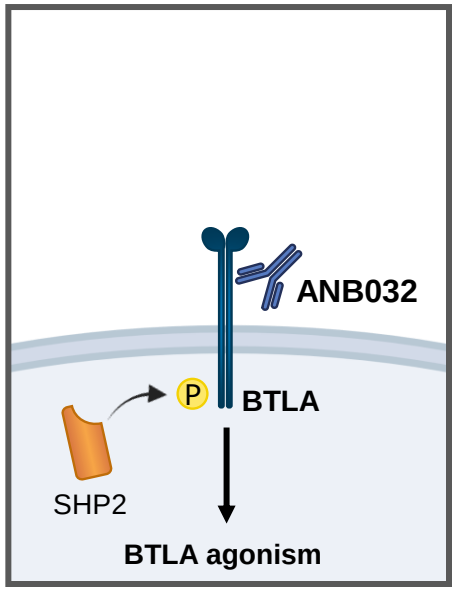
- Binds to BTLA on epitope proximal to immune cell
- Fc receptor binding profile contributes to differentiated potency
- Non-blocking of HVEM engagement with optimized antigen binding affinity

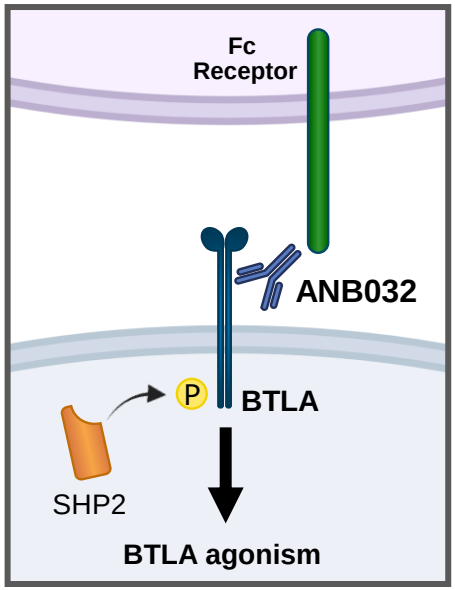
ANB032's agonist signal modulates immune cells

- Inhibits activated T cell proliferation
- Reduces inflammatory cytokine secretion
- Modulates DC function, including inducing Tregs

ANB032's optimized Fc receptor engagement significantly enhances BTLA agonism

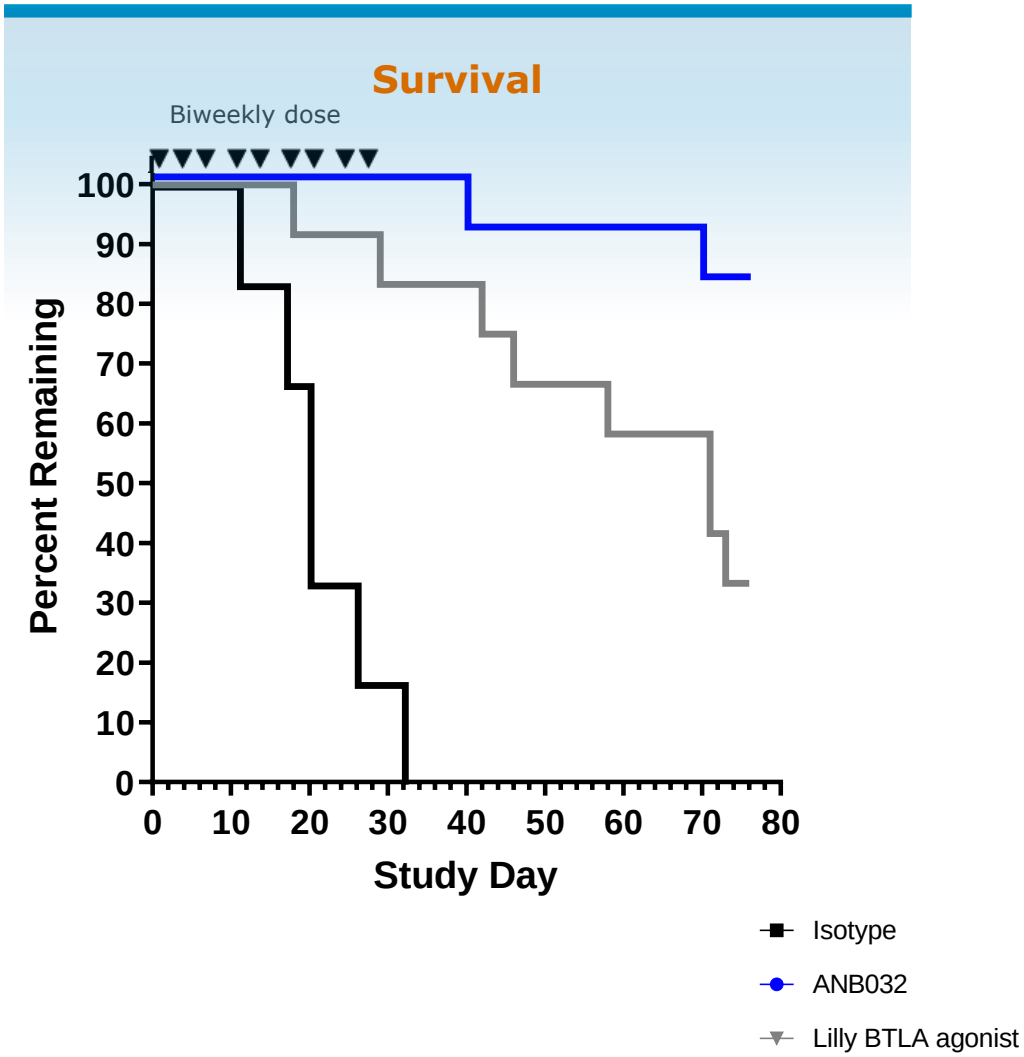


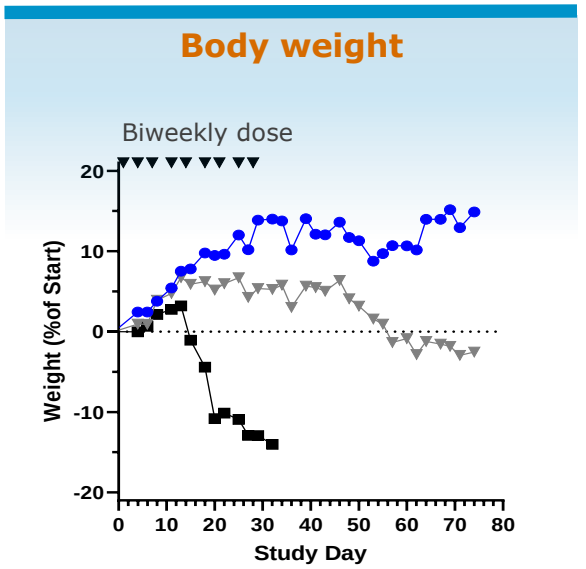


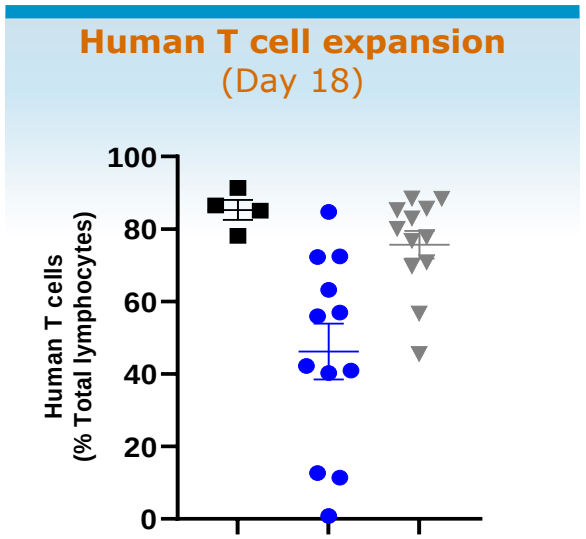


Jurkat BTLA SHP2 Recruitment Assay methodology: BTLA and SHP2 are fused with complementary enzyme fragments, when SHP2 is recruited to activated phosphorylated BTLA, the enzyme donor and enzyme acceptor form active β-gal that is detected by chemiluminescence.

ANB032 demonstrates best-in-class in-vivo efficacy in humanized murine model of GvHD







All antibodies given at the same biweekly dose.

ANB032 modulates immune cell activity with optimized agonistic signaling

- Checkpoint agonism leverages natural immune regulatory mechanisms to safely resolve inflammation and restore balance
- BTLA is a key checkpoint that modulates activity of T, B and dendritic cells
- ANB032 optimized Fc receptor engagement and proximal binding epitope significantly enhances BTLA agonism
- ANB032 has demonstrated preclinical proof of mechanism and best-in-class efficacy in vivo

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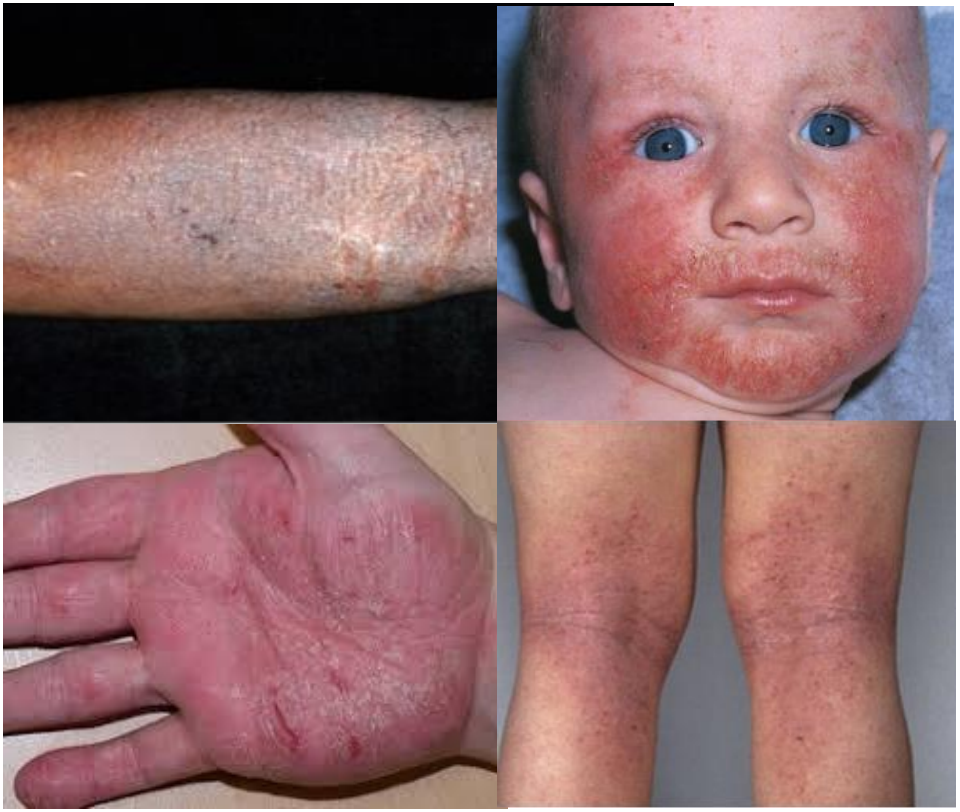
Q&A	AnaptysBio

Unmet Needs in Atopic Dermatitis and Need for Additional Targets

Emma Guttman-Yassky, M.D., Ph.D.
Waldman Professor of Dermatology
System Chair, Department of Dermatology
Icahn School of Medicine at Mount Sinai, New York, NY
Immediate Past President, International Eczema Council

Mount Sinai

Atopic dermatitis



- Most common inflammatory skin disease (~7% of adults in US, 15% of children)
- 20-30% of patients have moderate-to-severe disease
- Large unmet need for long-term disease control

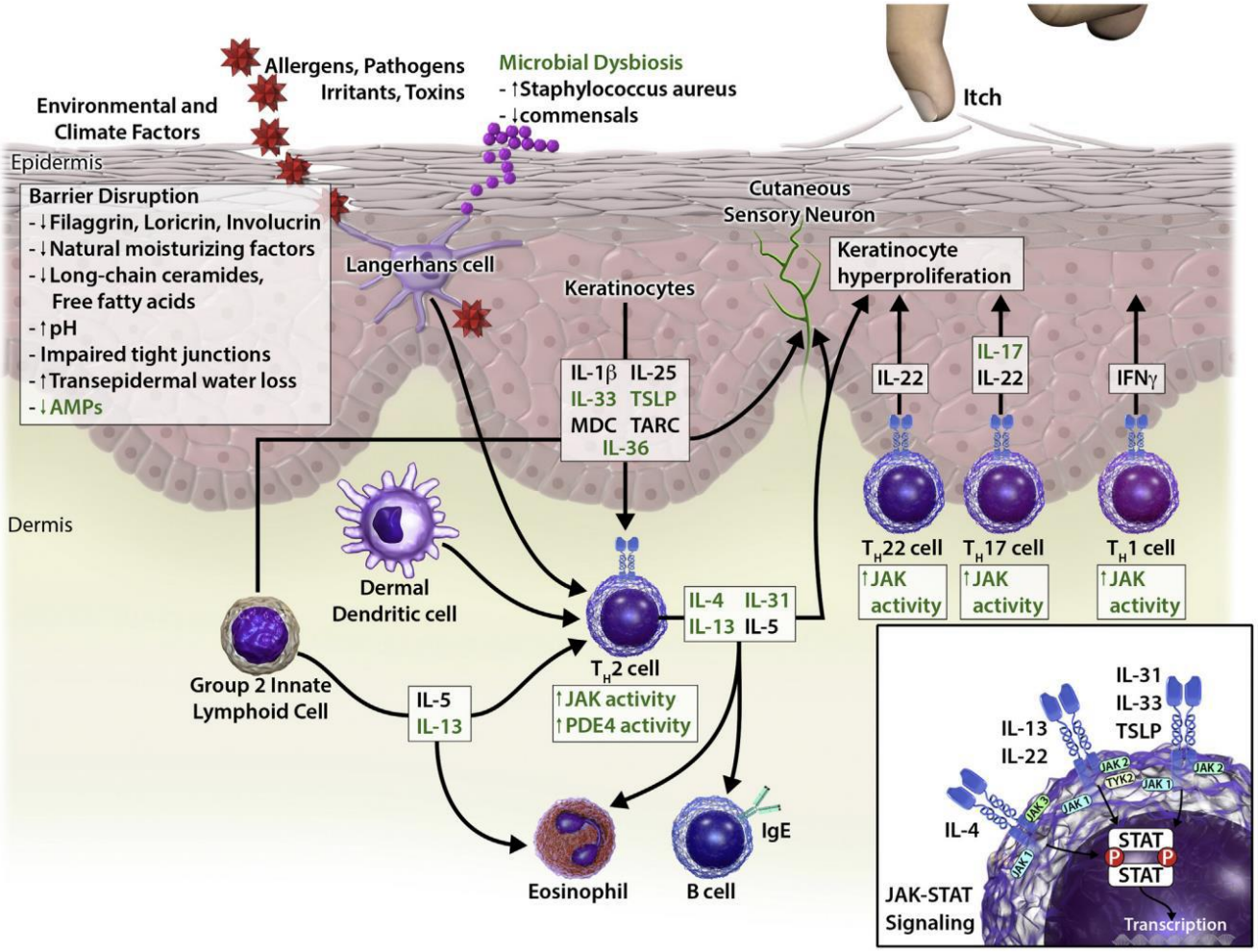
The therapeutic drought is finally ending!

Impact of moderate-to-severe AD[1-6]

AD = atopic dermatitis; QoL = quality of life

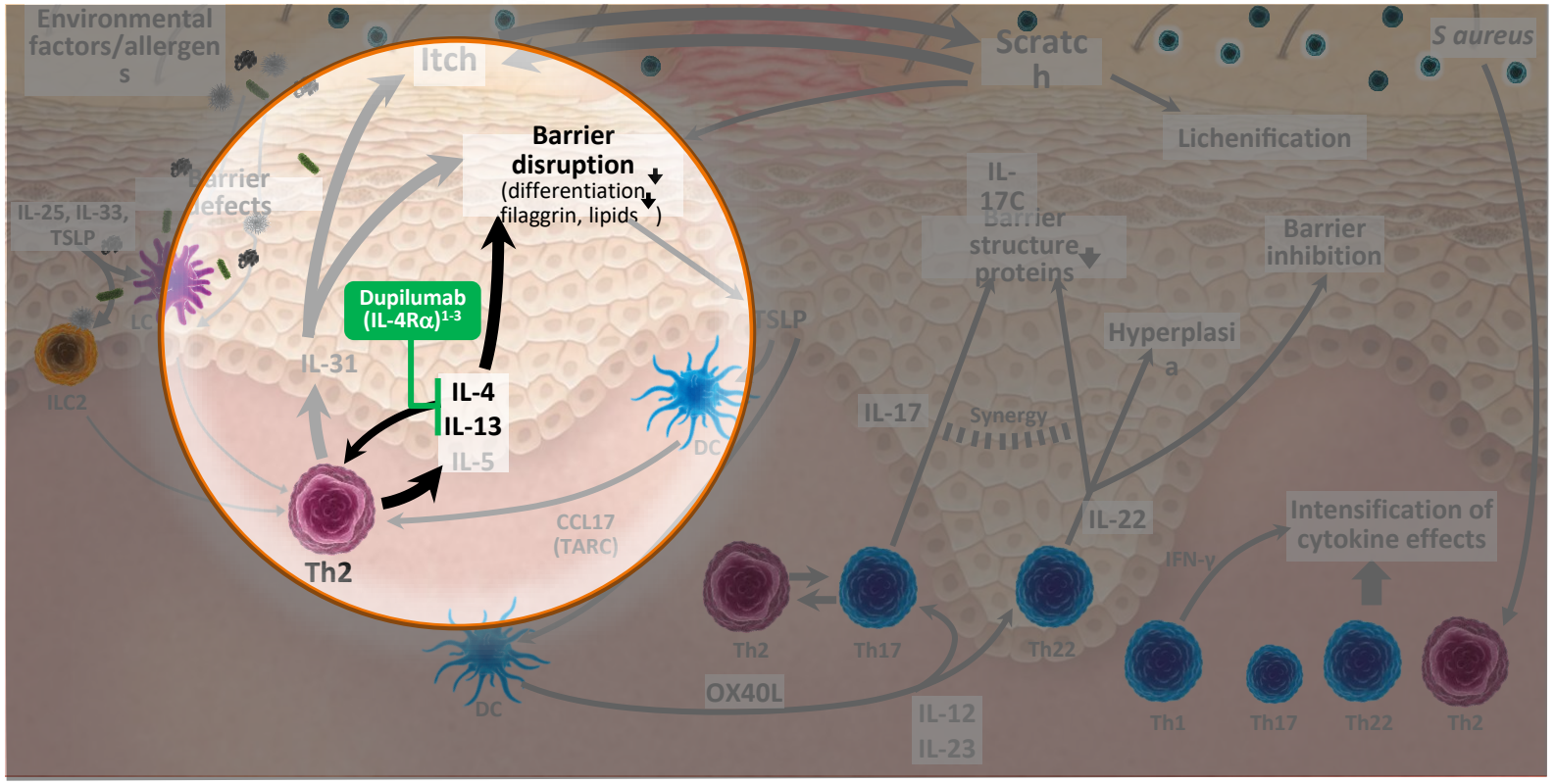
1. Silverberg JI, et al. *Ann Allergy Asthma Immunol* 2018;121(3):340–347; 2. Chrostowska-Plak D, et al. *Acta Derm Venereol* 2009;89(4):379–383;
3. Zuberbier T, et al. *J Aller Clin Immunol* 2006;118(1):226–232; 4. Patel KR, et al. *J Am Acad Dermatol* 2019;80(2):402–410;
5. Silverberg JI, et al. *Ann Allergy Asthma Immunol* 2019;123(2):144–151; 6. Schmidt SAJ, et al. *JAMA Dermatol* 2021;157(6):1–9.

Greater understanding of disease pathogenesis is enabling development of novel therapies

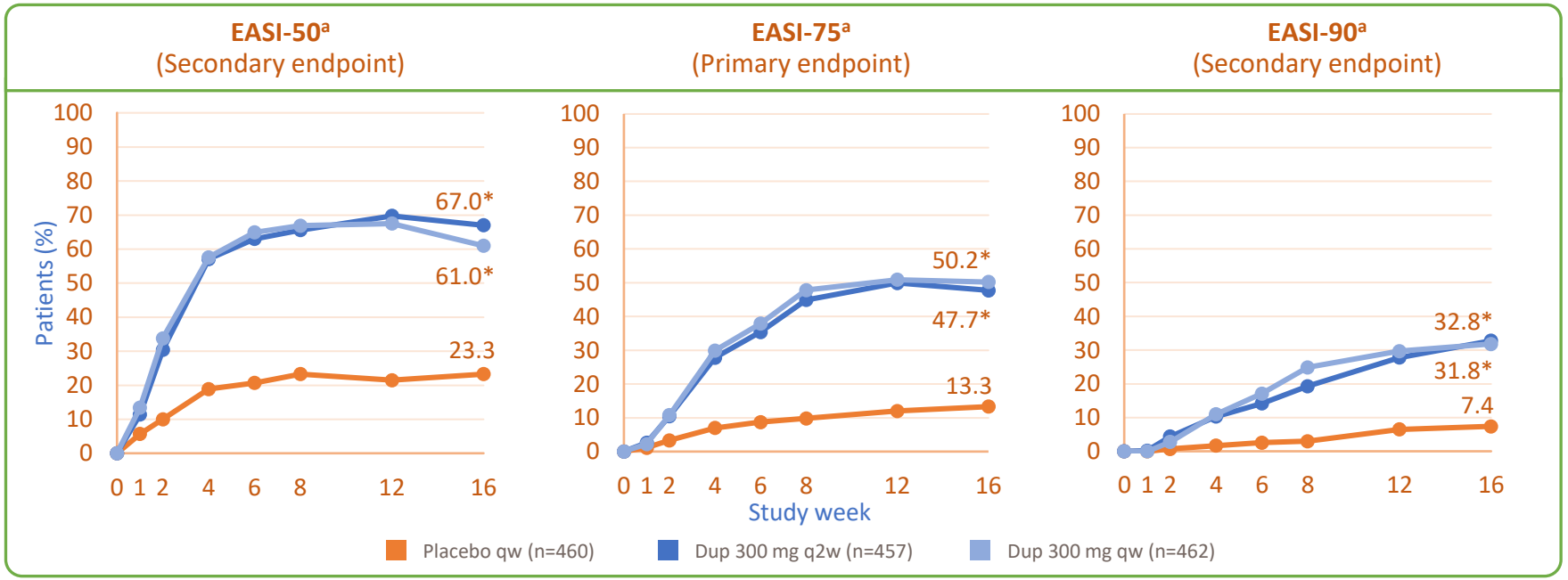


Paller et al. JACI 2019

Dupilumab (anti-IL-4Rα) targets IL-4 and IL-13



EASI-50/75/90 in pooled solo 1 & 2 monotherapy 16-week studies



*P<0.0001 vs placebo.

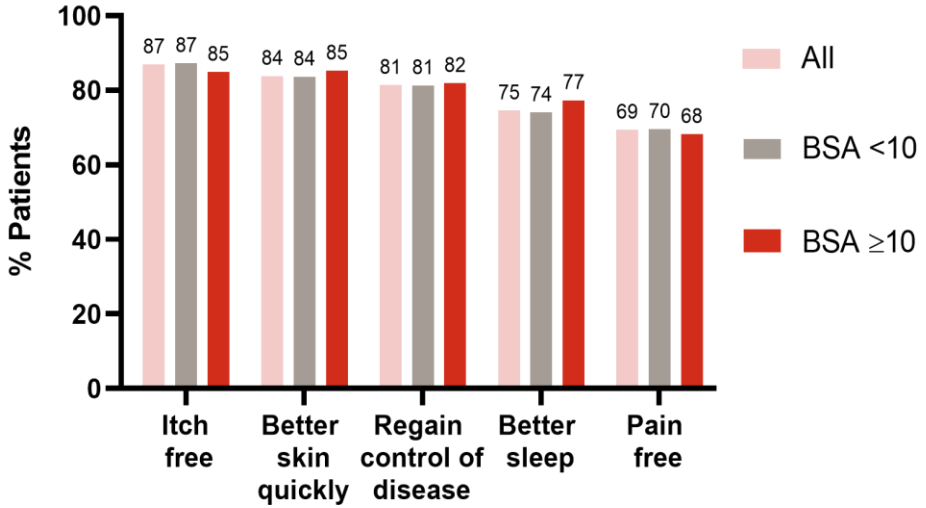
The only licensed dose for dupilumab in moderate-to-severe AD patients is 300 mg Q2W.

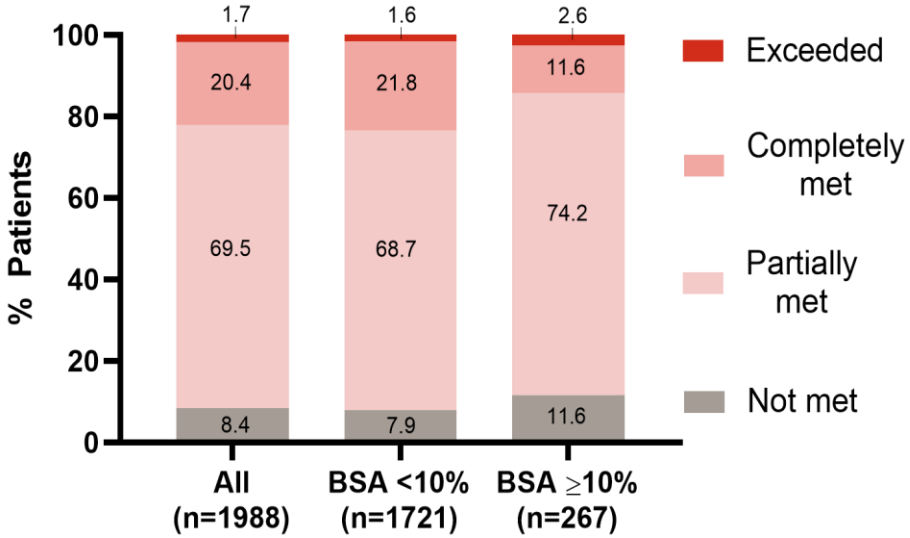
Baseline EASI mean scores (SD): placebo, dupilumab 300 mg q2w, and dupilumab 300 mg qw were 34.0 (14.4), 32.4 (13.3), and 32.5 (13.3), respectively.

Dup=dupilumab; EASI=Eczema Area and Severity Index; EASI-50/75/90=proportion of patients with ≥ 50%/75%/90% improvement in EASI score from baseline; q2w=every 2 weeks; qw=weekly; SD=standard deviation.

1. Ferrándiz C, et al. Dupilumab in moderate-to-severe atopic dermatitis: pooled efficacy results from two identically designed randomized phase 3 trials (SOLO 1 & 2). Presented at: EADV 2017; September 13-17, 2017; Geneva, Switzerland.

Where are we: Less than 25% AD patients are completely satisfied with SOC





AD = atopic dermatitis; BSA = body surface area

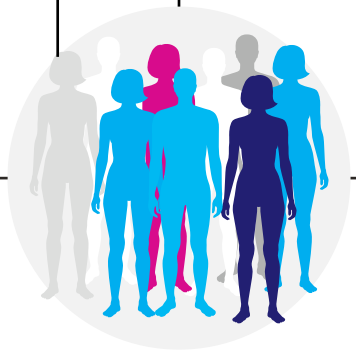
Griffiths CM, *EADV* 2020.

AD is highly heterogeneous and involves multiple immune cytokines (e.g., IFN-γ, IL-4, IL-13, IL-17, IL-22)

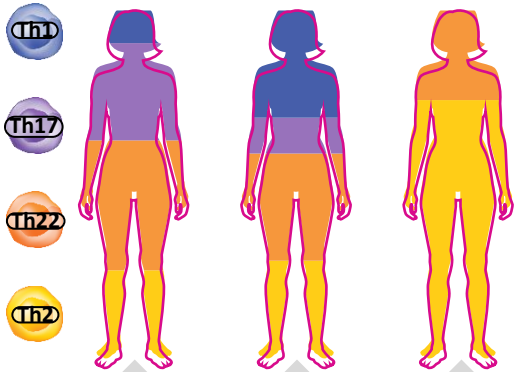
AD exhibits variable patterns

- Disease activity[1]
- Disease severity[2,6]
- Location (eg, face, genitals)[2]

Immune activation may vary between age of onset[2], disease duration, and ethnicity[4,5], resulting in heterogeneous presentation

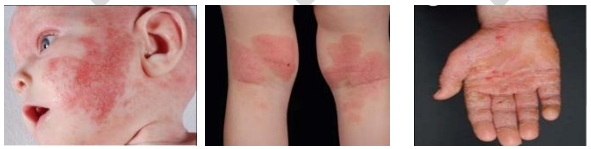


Immune Pathway Skewing



Morphology and distribution of AD lesions differ between age groups[2,3]

Infantile phase | Childhood phase | Adult phase

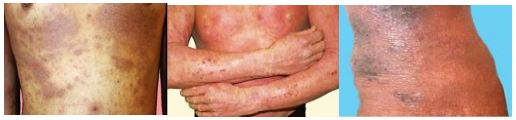


- Adult-onset AD has **greater Th1 activation** compared to pediatric-onset[2]

Other factors varying between patients

- Response to therapy[2,6,7]
- IgE status[7]
- Trigger-induced events[3]
- Impact on itch, sleep, and QoL[3]

Asian AD | European-American AD | African-American AD



AD, atopic dermatitis; IgE, immunoglobulin E; QoL, quality of life; Th, T helper cell

1. Chovatiya R, Silverberg JI. Am J Clin Dermatol 2022. Online ahead of print; 2. Facheris P….and Guttman-Yassky E. Allergy 2023; ; 3. Spergel JM, et al. J Allergy Clin Immunol 2003;112:S118–27; 4. Renert-Yuval Y, et al. Ann Allergy Asthma Immunol 2020;124:28–35; 5. Czarnowicki T, et al. J Allergy Clin Immunol 2019;143:1–11; 6. Weidinger S, et al. Nat Rev Dis Primers 2018;4:1; 7. Bieber T. Ann Dermatol 2010;22:125–37

What's in the pipeline for AD patients?

	Target	Compound	Phase	Sponsor
Single cytokine blockers	IL-13	Tralokinumab	FDA-, EMA-approved	LEO
	IL-13	Lebrikizumab	3	Lilly
	IL-13	Cendakimab	2	BMS
	IL-13RA1	ASLAN004	2	ASLAN
	IL-4RA	CBP201	3	Connect Biopharma
	IL-31	Nemolizumab	3	Galderma /Chugai
	IL-22RA1	LEO138559	2	LEO
Immune cell modulator	OX40	KHK4083/ AMG 4051	3	Amgen (KHK)
	OX40L	Amlitelimab/KY1005	2b	Sanofi
	BTLA agonist	ANB032	2b	AnaptysBio
Small Molecules	CCR4	RPT193	2b	RAPT
	S1PR	Etrasimod	3	Arena/Pfizer

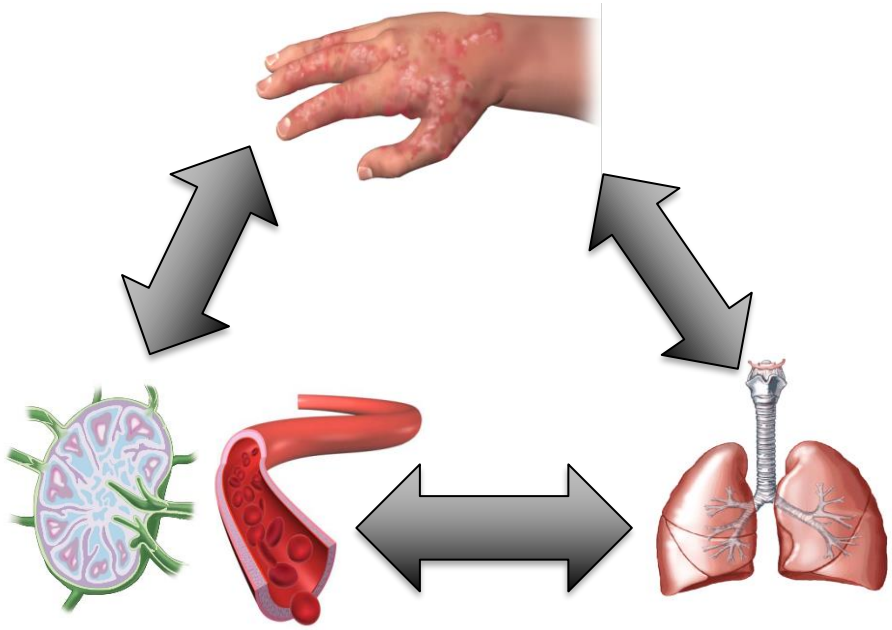
IL-17C, IL-33, TSLP, IL-36, IL-1, IL-17A, IL-5 failed clinical trials in AD

EU=European Union; IL=interleukin; IV=intravenous; TSLP=thymic stromal lymphopoietin.

JAK Inhibitors: Topical: Ruxolitinib (FDA approved in US as of September 2021), Delgocitinib (Approved in Japan)
Other topicals: Roflumilast, Tapinarof (phase 3 in AD)
Oral: Abrocitinib, Upadicitinib, Baricitinib (FDA/EU/UK approval; baricitinib not FDA-approved for AD, only for AA)

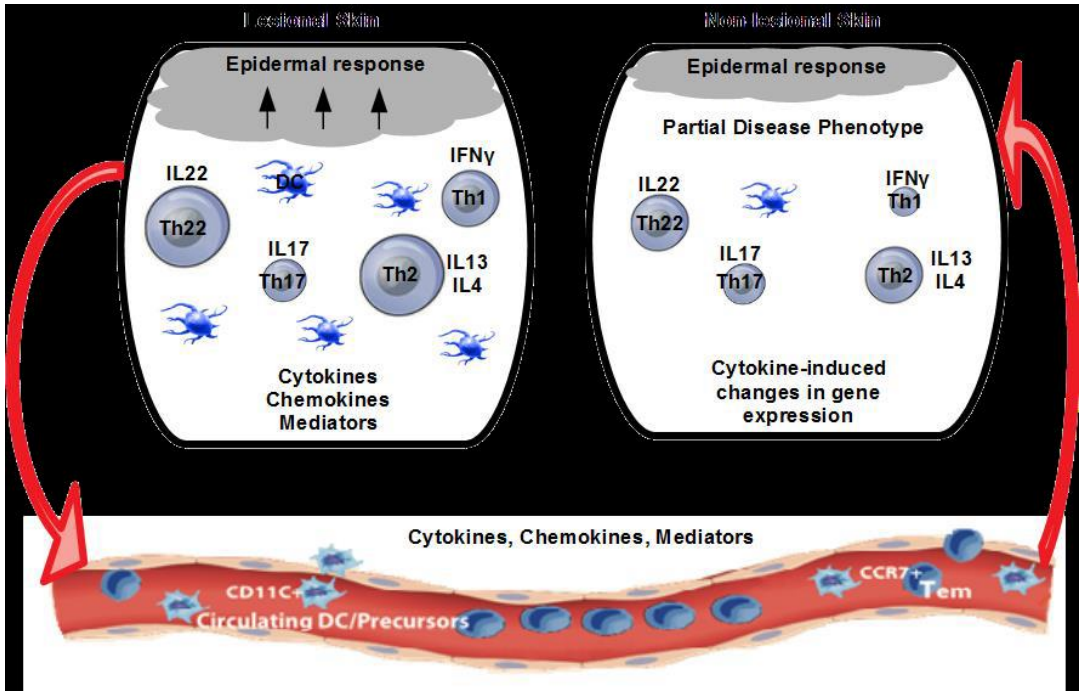
Atopic dermatitis emerges as systemic disease

- Systemic inflammation is well established in psoriasis
- Higher immune activation has been recently reported in peripheral blood from AD vs. psoriasis patients
  - Increased activated T cells
  - Increased circulatory cytokines and cardiovascular associated markers
- Several population-based studies showed increased cardiovascular disease in AD



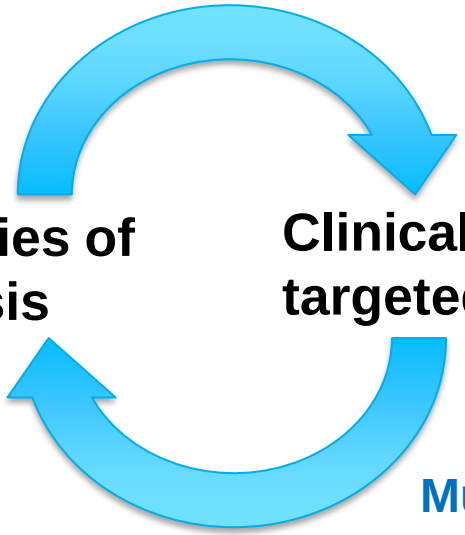
Czarnowicki T….and Guttman-Yassky E..: J Allergy Clin Immunol. 2015 Jul;136(1):208-11;
Ungar B….and Guttman-Yassky E et al.: J Invest Dermatol 2016
Brunner PM…Guttman-Yassky E. Sci Reports 2017.
Silverberg JI Allergy 2015 70: 1300–1308

To fully understand the effect of a given treatment, we need an integrated model of skin and blood AD biomarkers



How are we facilitating therapeutic development in AD

Skin and blood profiling helped formulate pathogenic hypotheses

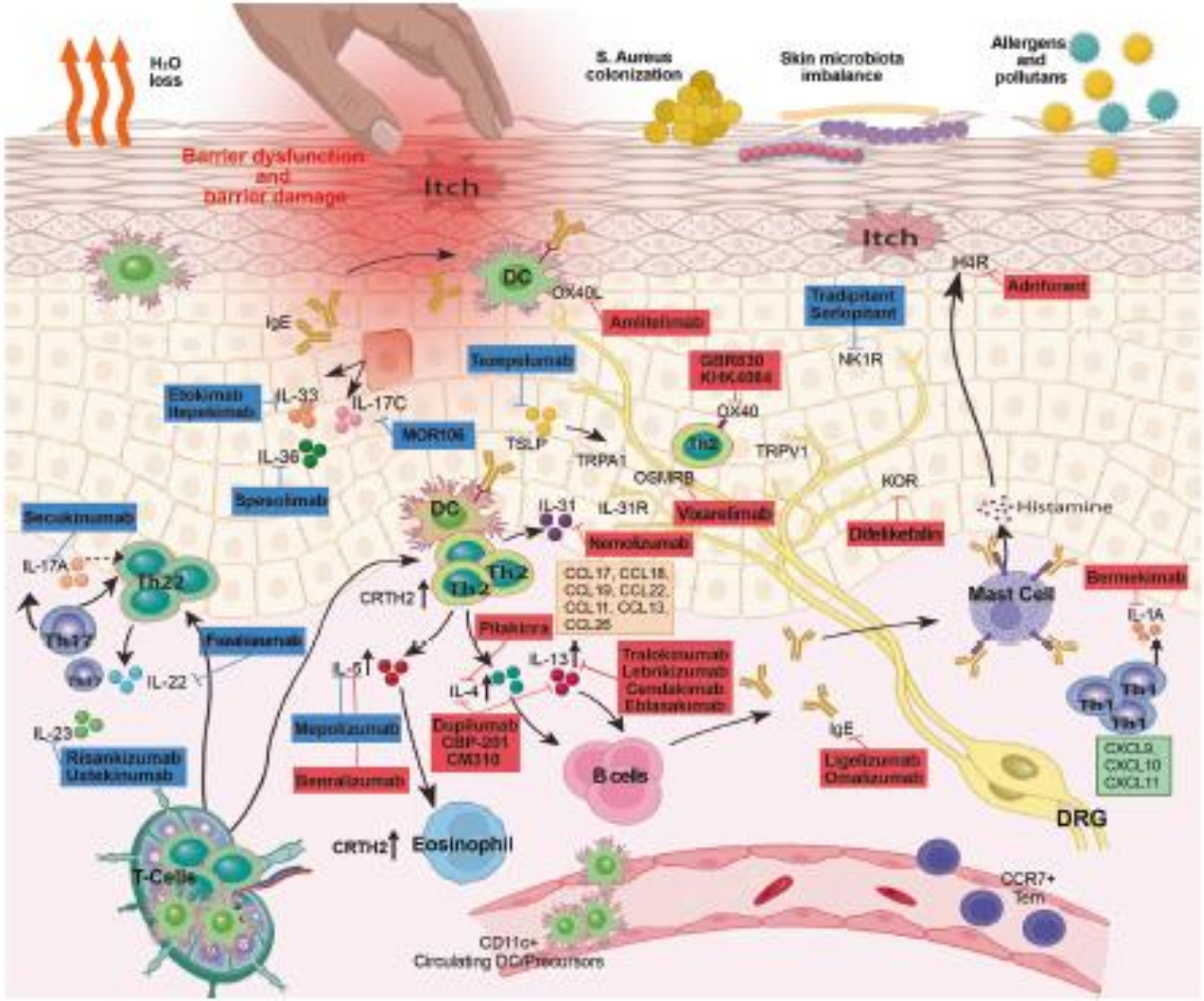


Skin and blood profiling ➔ biomarkers of therapeutic response

Multiple cycles ➔ Novel therapeutics

Both successes and failures have helped to frame pathogenic concepts and therapeutic directions

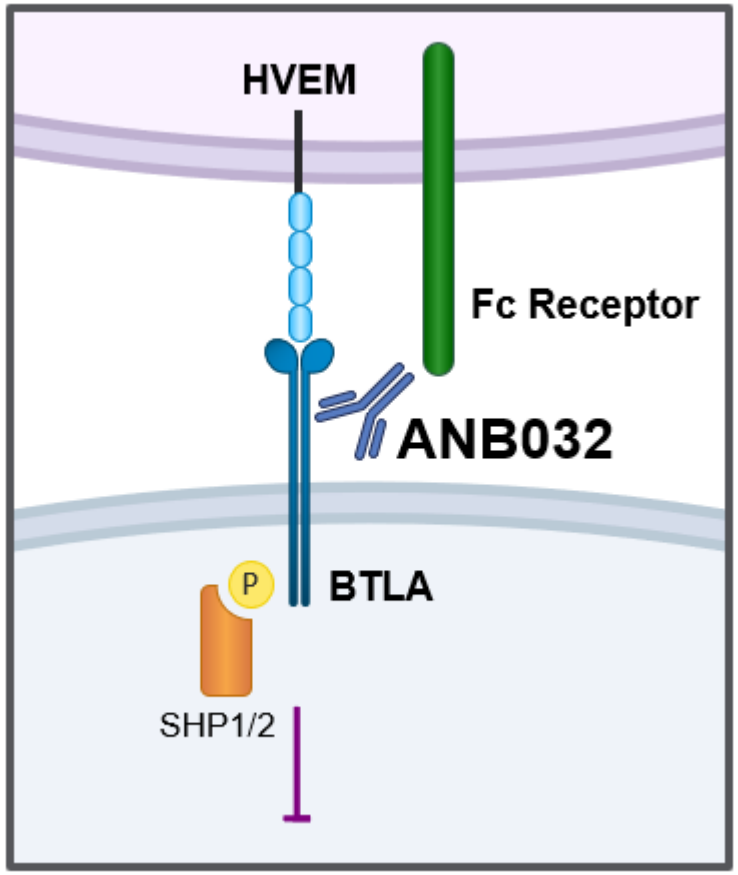
AD biology is heterogenous and uniquely fits BTLA MOA



Pathogenesis linked broadly to Th2, Th1, Th17, Th22 as well as dendritic cells

BTLA expressing cells (Th1, Th2, Th17, Th22, B, Dendritic) are clinically validated drivers of disease in atopic dermatitis

ANB032: Best-in-class BTLA agonist antibody



ANB032: IgG4 antibody (non-depleting)

- Binds to BTLA on epitope proximal to immune cell
- Fc receptor binding profile contributes to differentiated potency
- Non-blocking of HVEM engagement with optimized antigen binding affinity

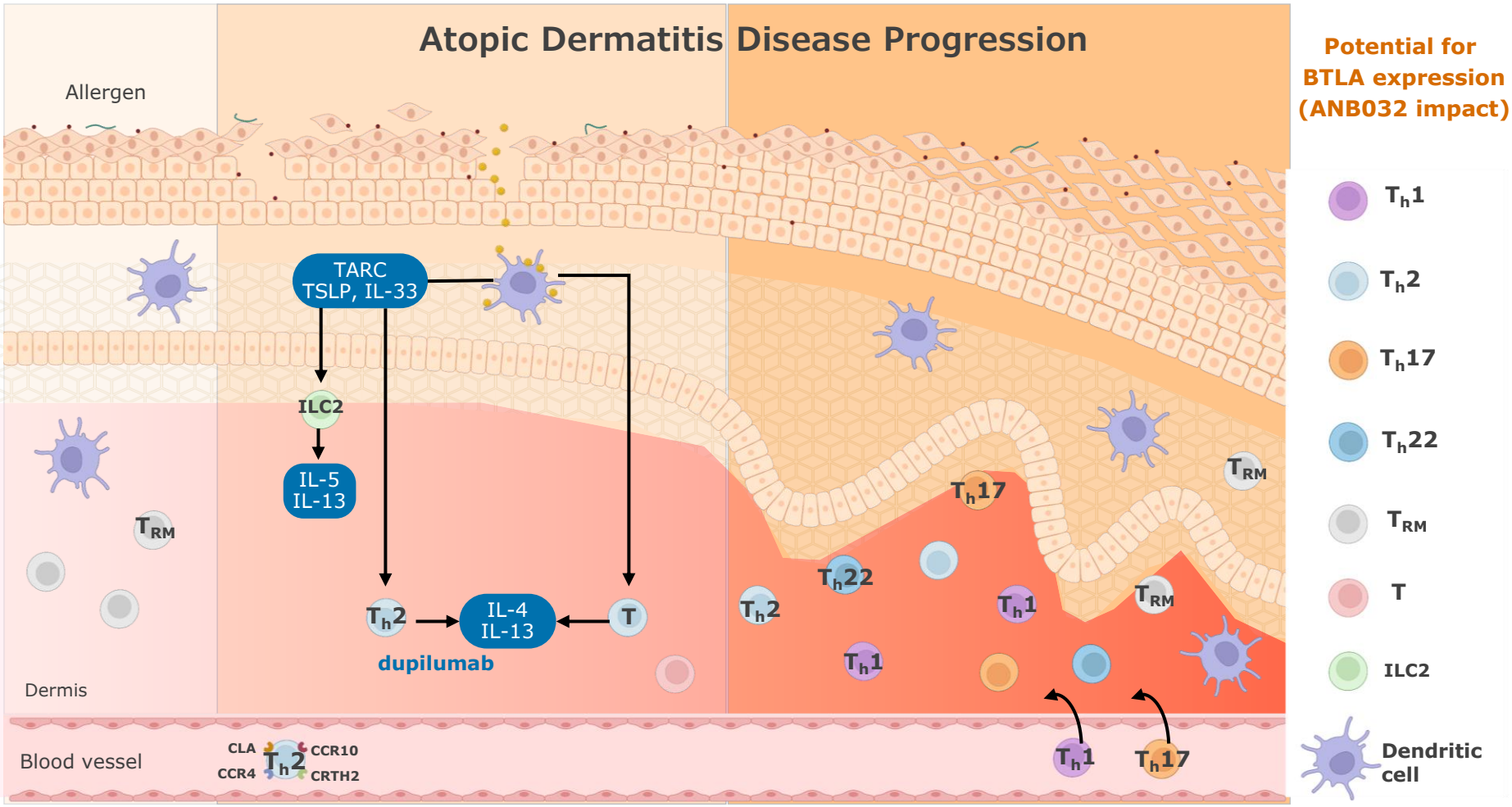
ANB032's agonist signal modulates immune cells

- Inhibits activated T cell proliferation
- Reduces inflammatory cytokine secretion
- Modulates DC function, including inducing Tregs

Agenda for today

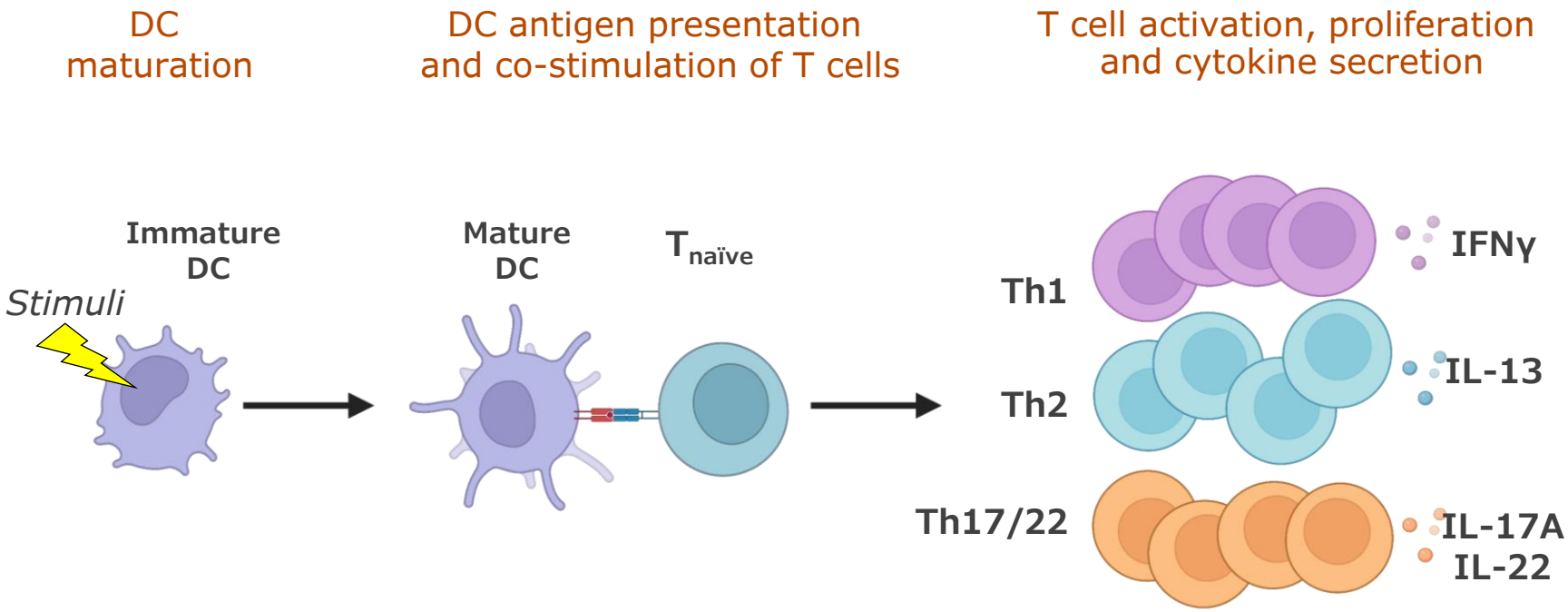
TOPIC	SPEAKER
AnaptysBio: Best-In-Class Immune Cell Modulators	Dan Faga Chief Executive Officer
Checkpoint Agonism and ANB032, a BTLA Agonist	Martin Dahl, Ph.D. Senior Vice President, Research
Unmet Needs in Atopic Dermatitis (AD) and Need for Additional Targets	Emma Guttman, M.D., Ph.D. Professor and Chair of Dermatology Icahn School of Medicine, Mount Sinai
Targeting AD with ANB032: Translational Data	Martin Dahl, Ph.D. Senior Vice President, Research
ANB032: Phase 1 Results and AD Phase 2b Trial	Paul Lizzul, M.D., Ph.D. Chief Medical Officer
Closing Remarks	Dan Faga Chief Executive Officer
Q&A	AnaptysBio

Th1, Th2, Th17, Th22 and dendritic cells in tissue and periphery drive AD pathogenesis

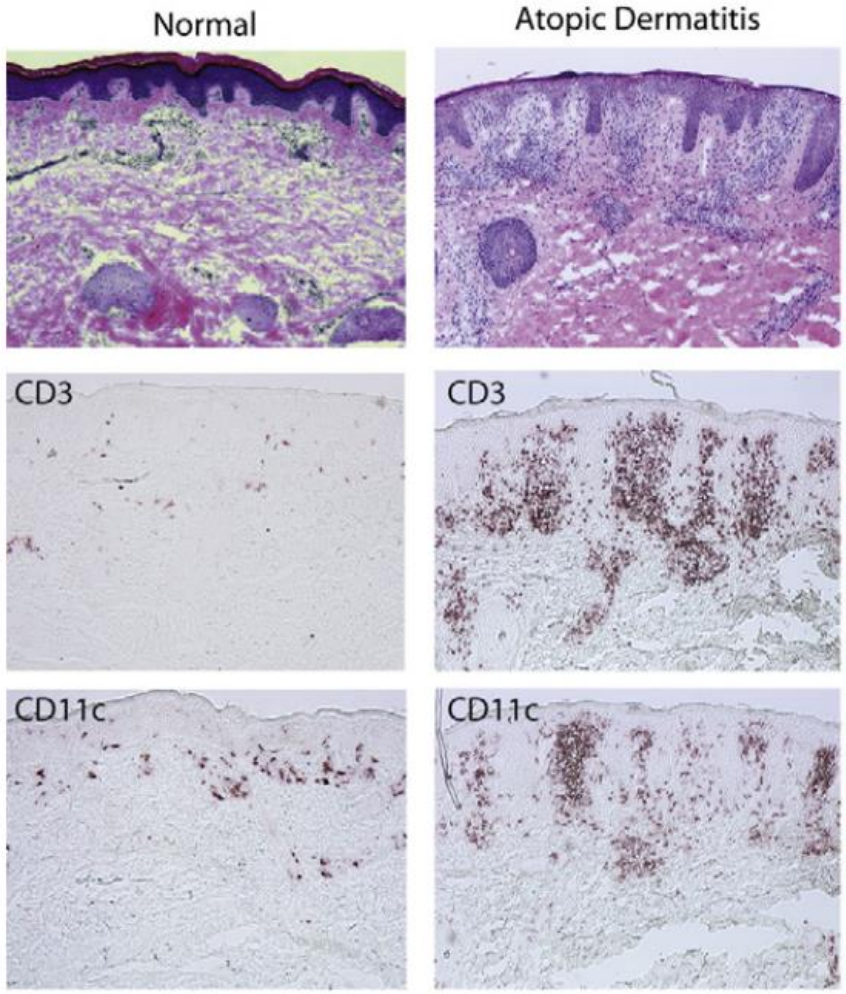


Adapted from Nature Reviews Disease Primers volume 4, Article number: 1 (2018).

BTLA is broadly expressed on immune cells driving atopic dermatitis pathogenesis



T cells and dendritic cells are hallmarks of AD skin



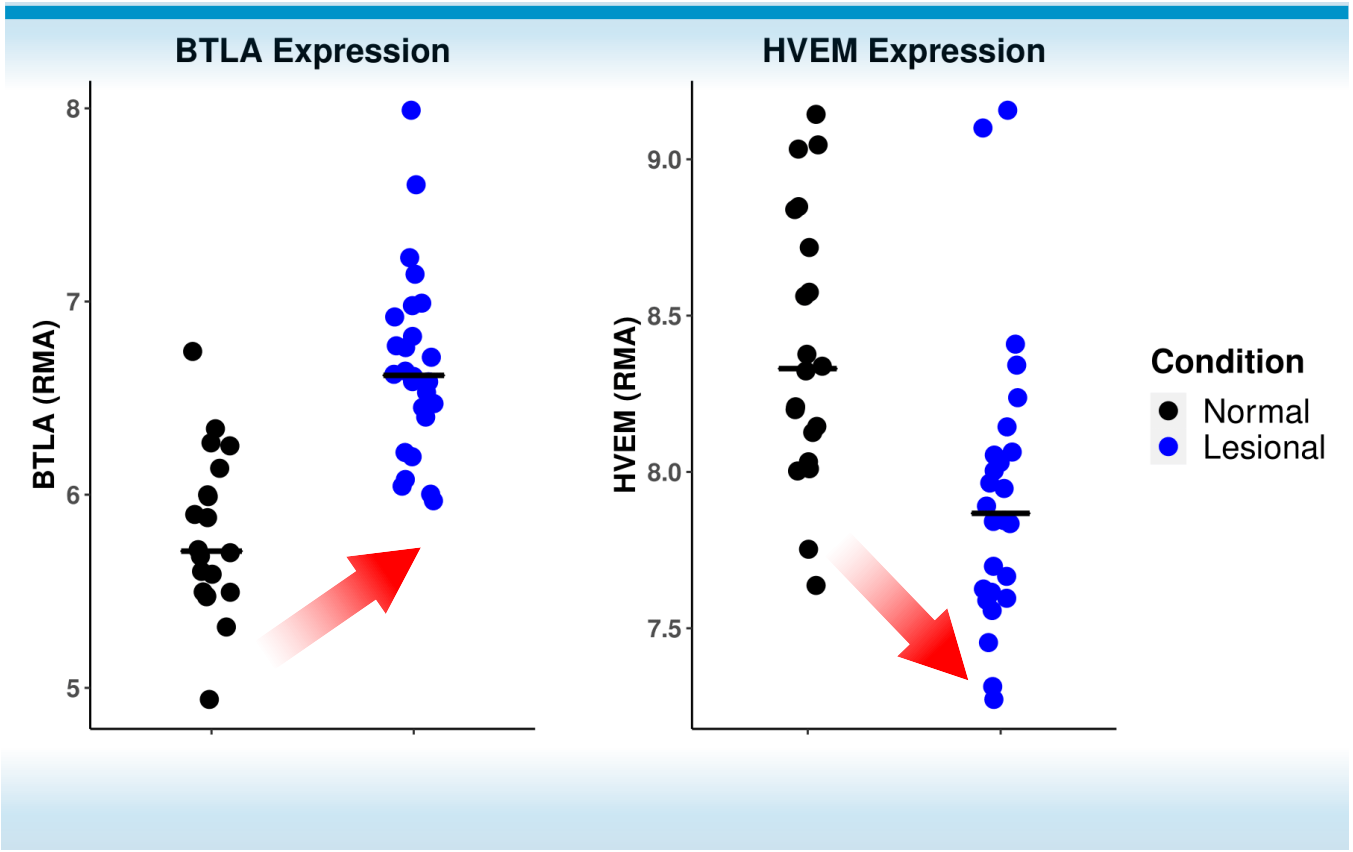
T cells

DCs

Guttman-Yassky et al, JACI, 2011.

BTLA pathway is dysregulated in AD tissue…

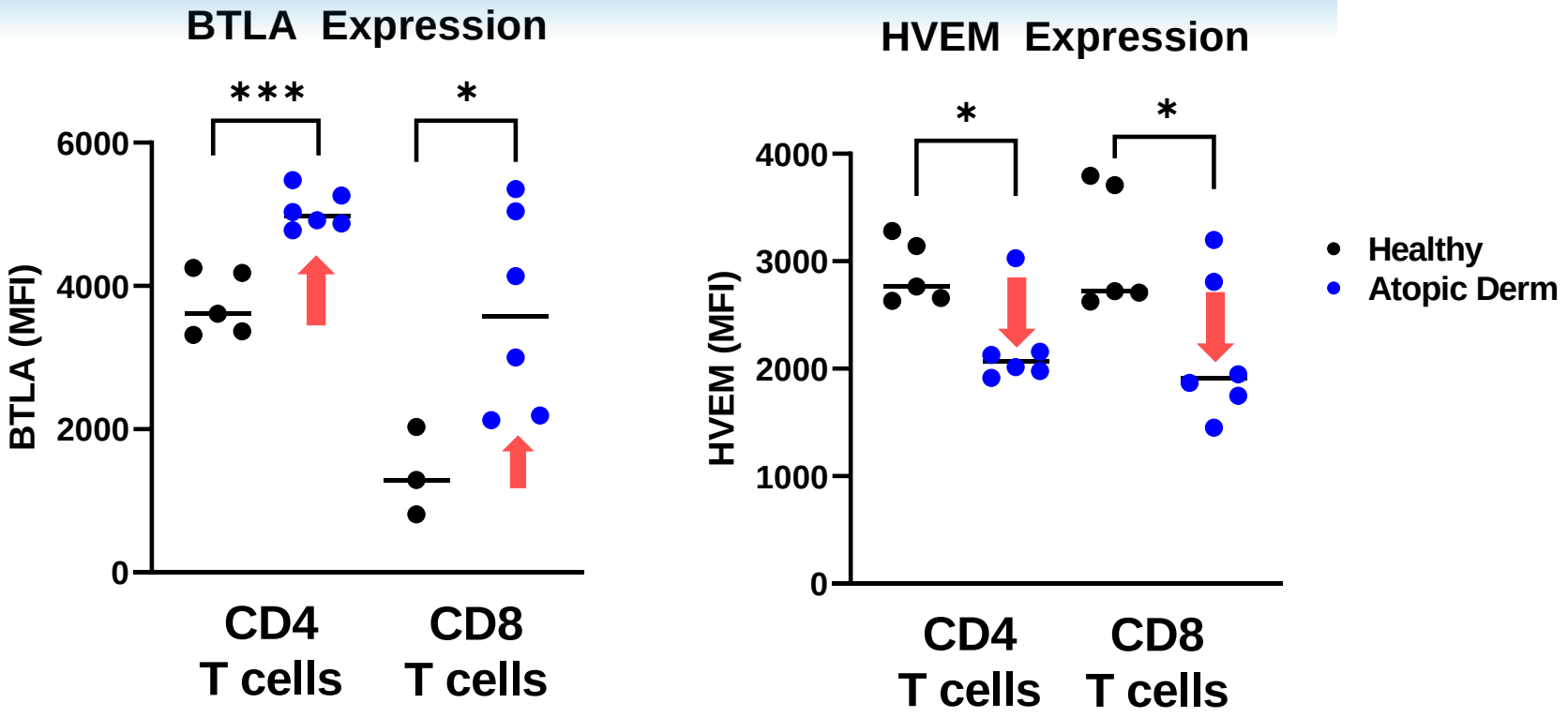
BTLA is elevated and HVEM is decreased,
suggesting insufficient agonism in diseased skin



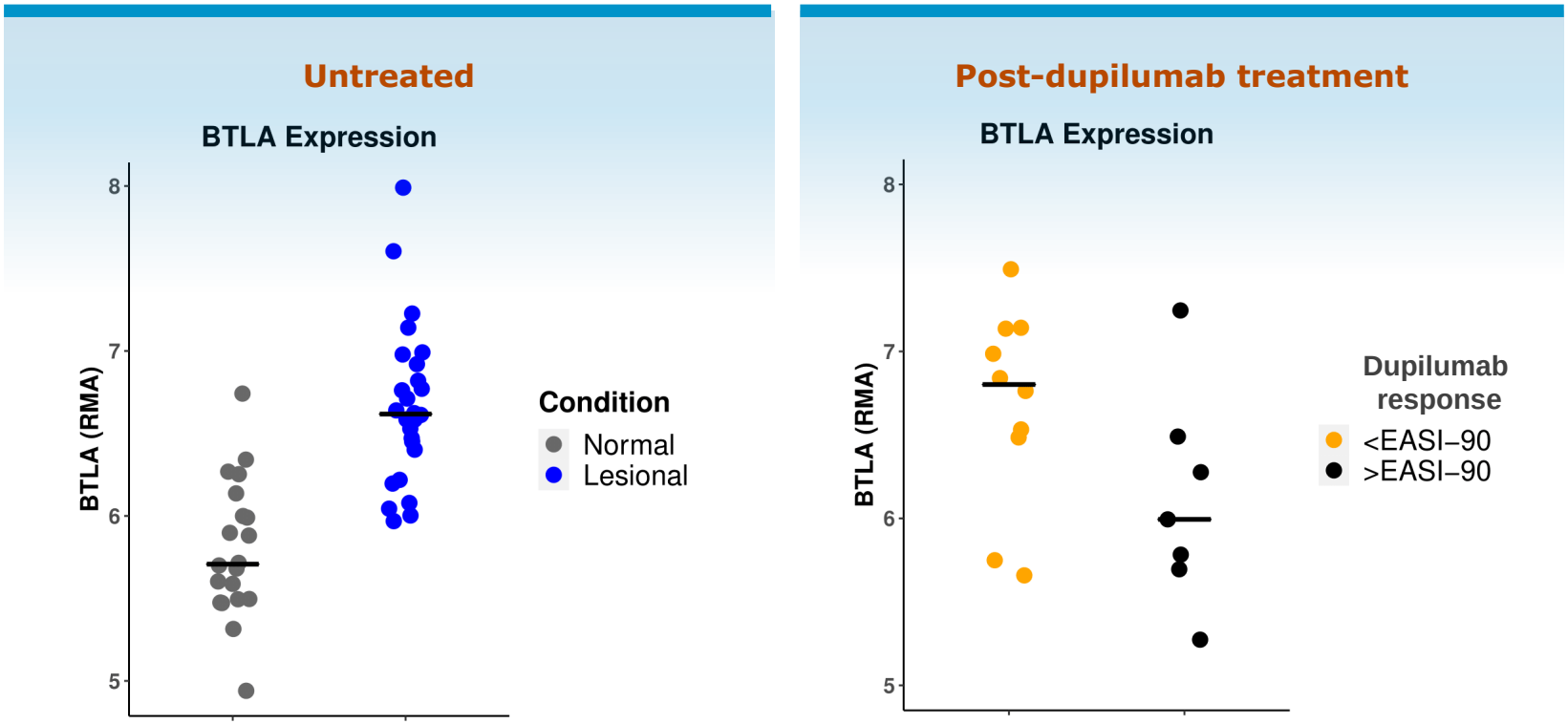
Adapted from Guttman et al, Journal of Allergy and Clinical Immunology, 2018.

...and in the periphery

BTLA is elevated and HVEM is decreased,
suggesting insufficient agonism in circulating T Cells



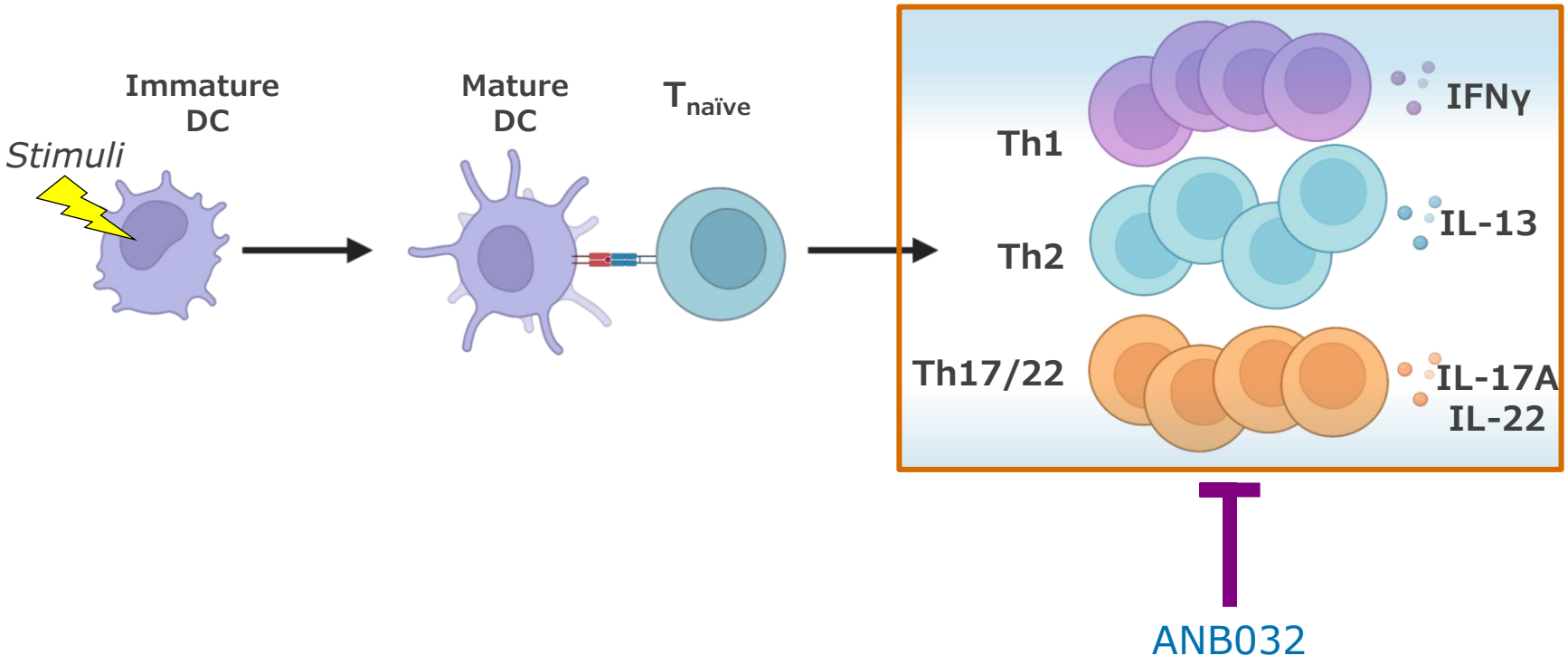
BTLA expression remains elevated after treatment with IL-13 targeted biologic



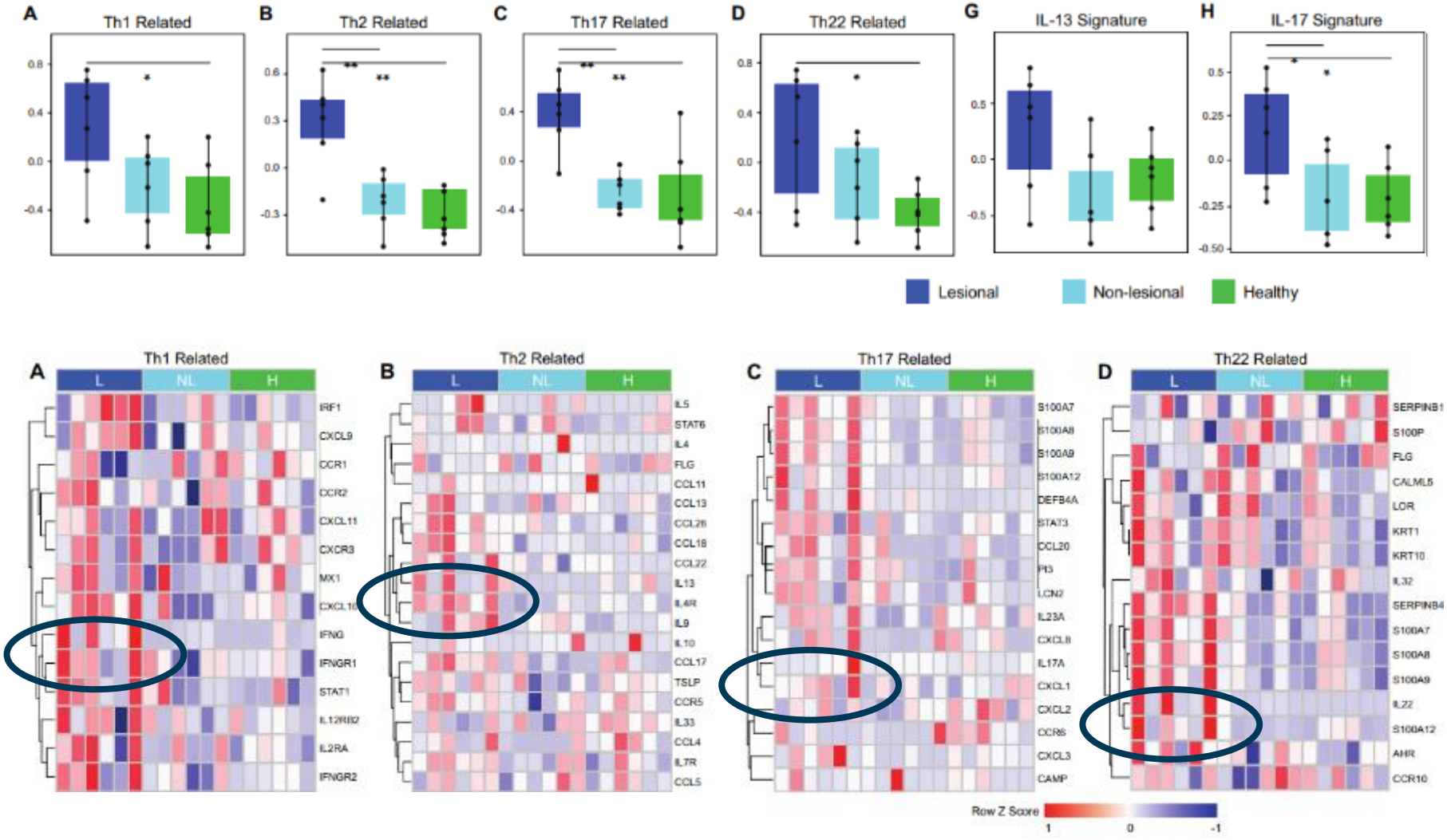
Analysis based on data sourced from Guttman et al, Journal of Allergy and Clinical Immunology, 2018.

ANB032 inhibits pathogenic T cell amplification in both periphery and skin

Inhibition of activated T cell proliferation
and reduction of inflammatory cytokine secretion

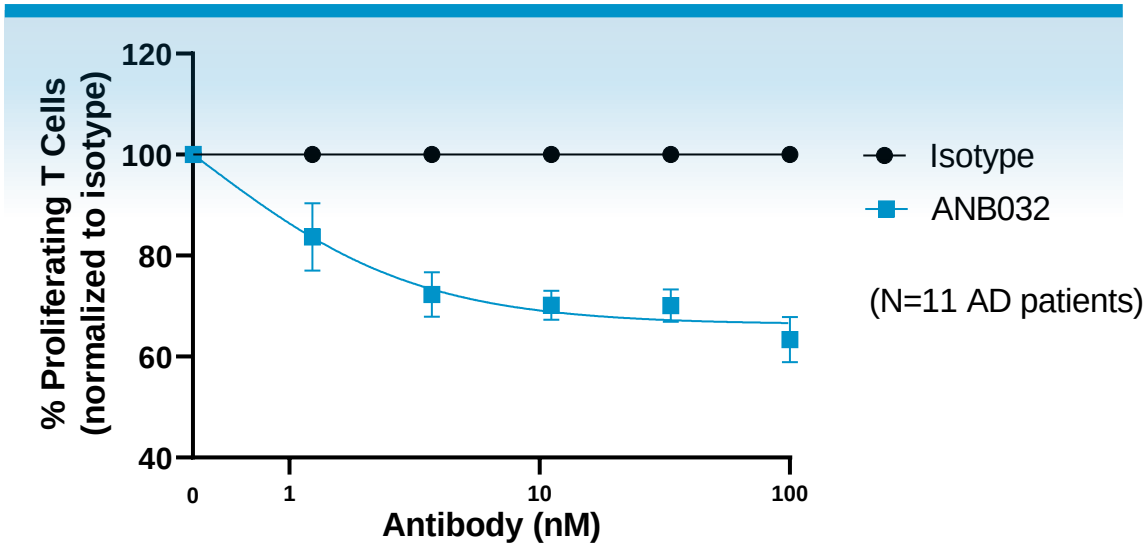


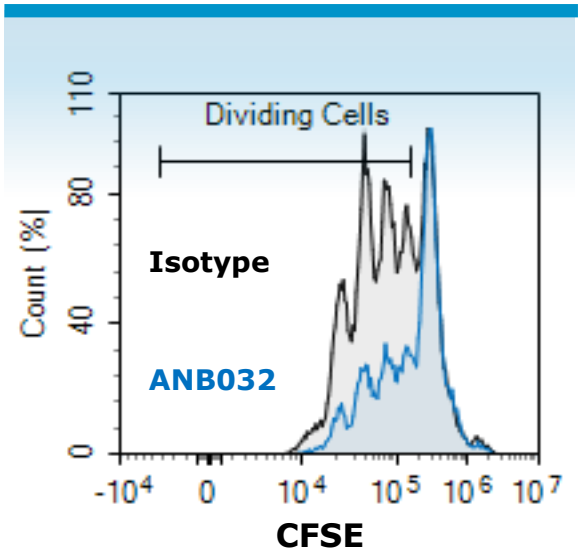
Broad T cell (Th1, Th2, Th17 and Th22) signatures characterize atopic dermatitis



Wongvibulsin et al, Sci Rep. 2021 May 27;11(1):11175.

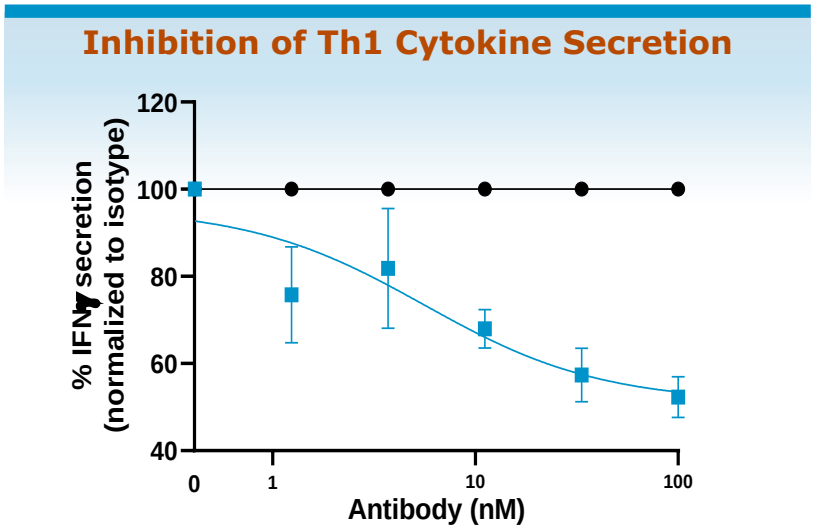
ANB032 inhibits T cell proliferation in AD patient-derived PBMCs

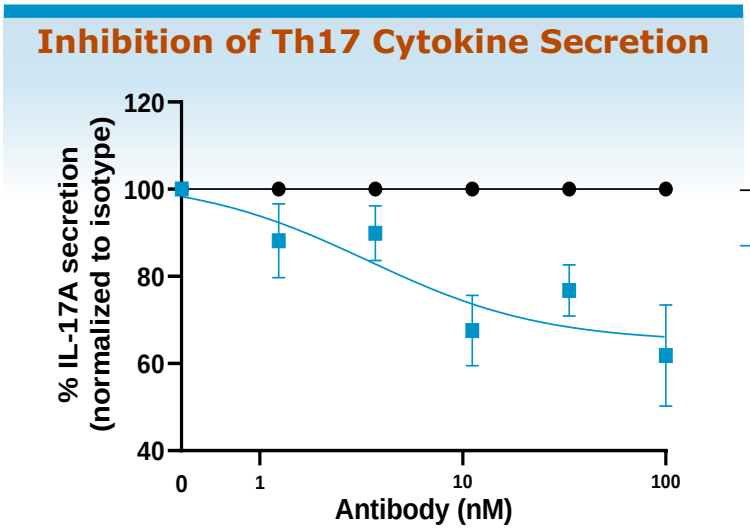


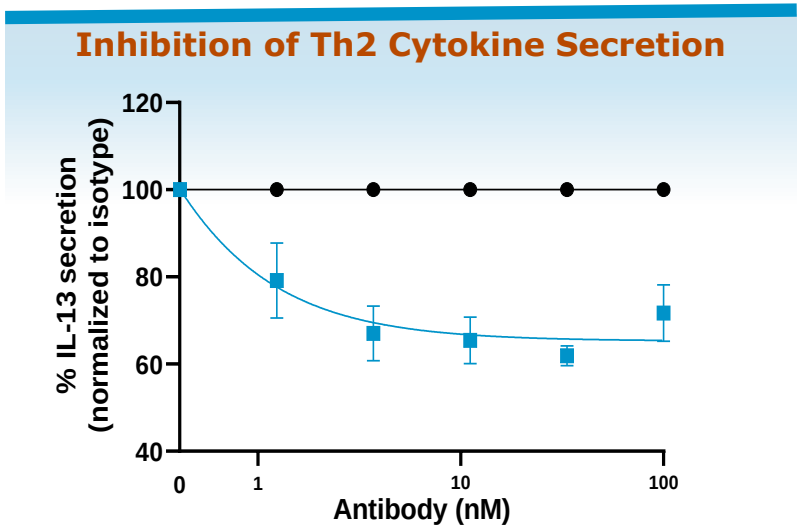


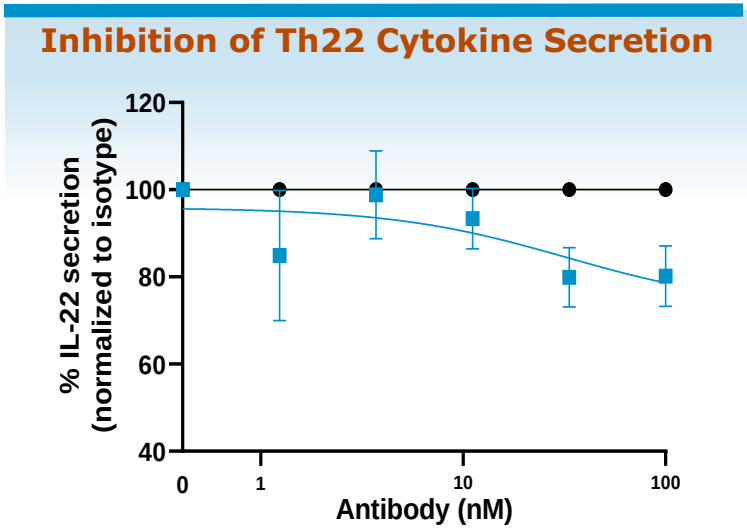
Assay protocol includes incubating AD-patient derived PBMCs with ANB032 and analyzing samples for T cell proliferation three days later.

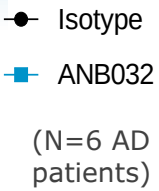
ANB032 inhibits Th1, Th2, Th17 and Th22 cytokine secretion in AD patient-derived PBMCs





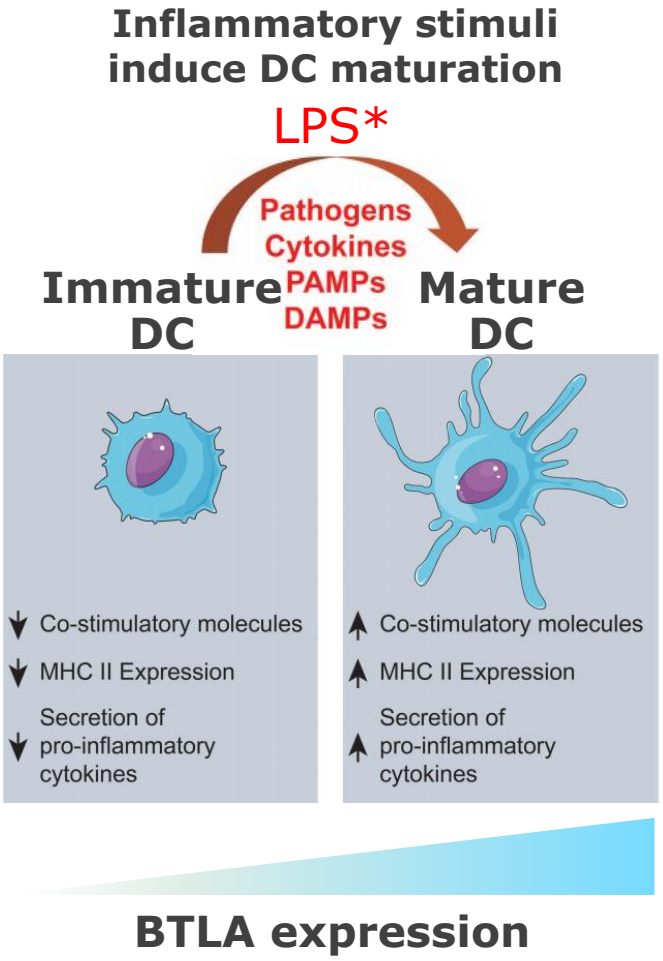


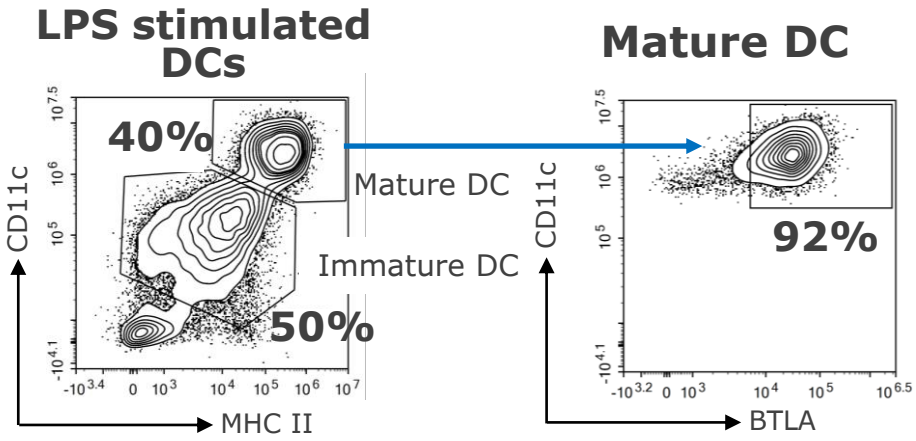




(N=6 AD patients)

BTLA highly expressed on mature DCs

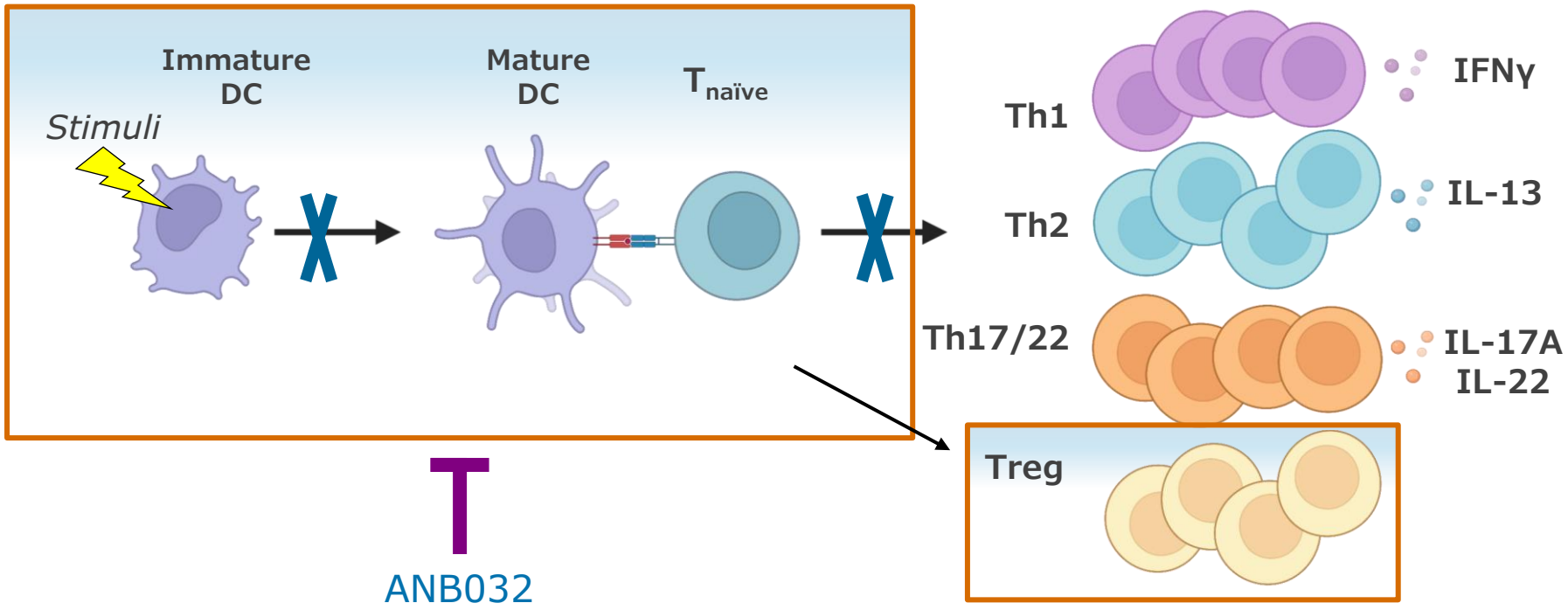




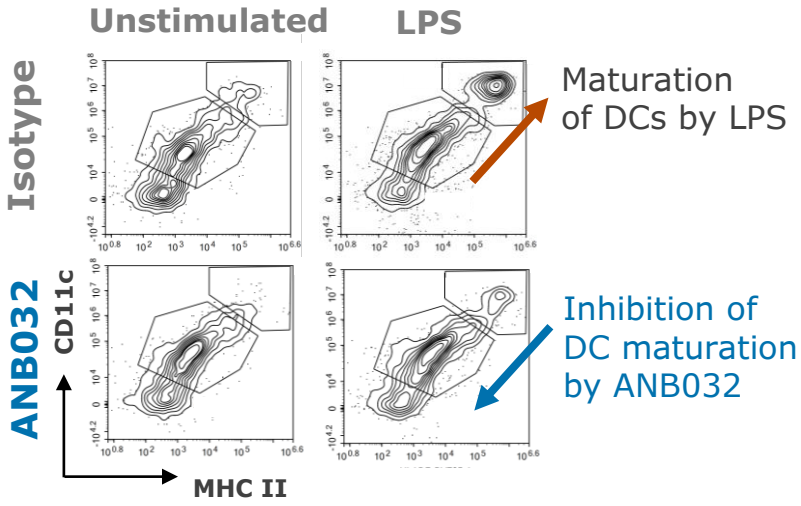
Adapted from Frontiers in Immunology. 2019 Jan 21;9:3176 ; * LPS=Lipopolysaccharide (a TLR4 agonist).

ANB032 modulates dendritic cell function

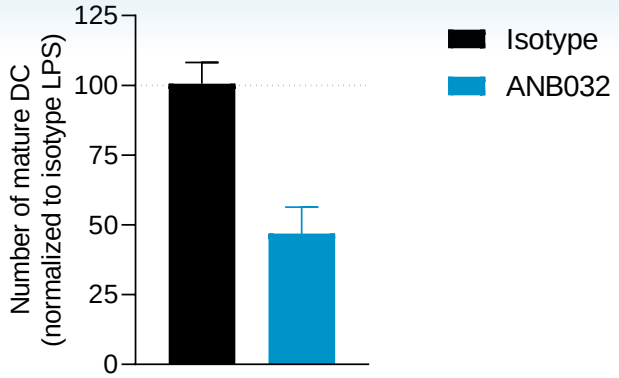
Reduction of mature dendritic cells (DCs) enhances the direct inhibitory effect on T cells and induces CD4+ FOXP3+ Tregs



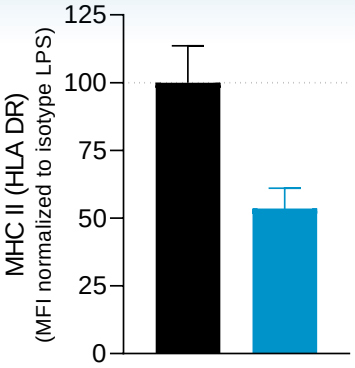
ANB032 inhibits DC maturation and reduces antigen presentation and co-stimulatory molecules



ANB032 inhibits DC maturation

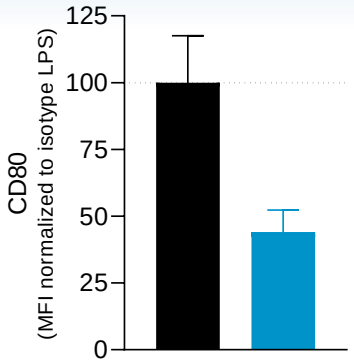


Antigen presentation
(Gated on total CD11c+ DCs)
MHC II

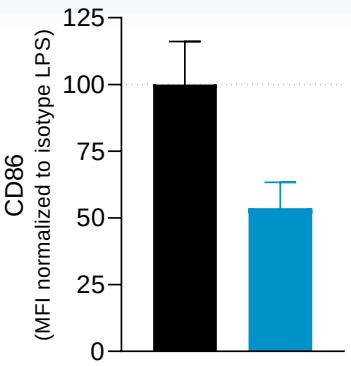


Co-stimulatory molecules on DCs
(Gated on total CD11c+ DCs)

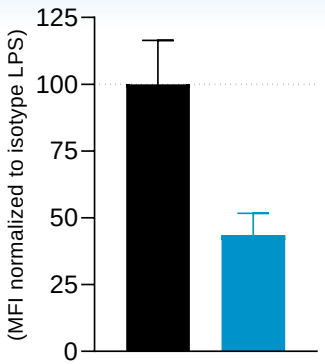
CD80



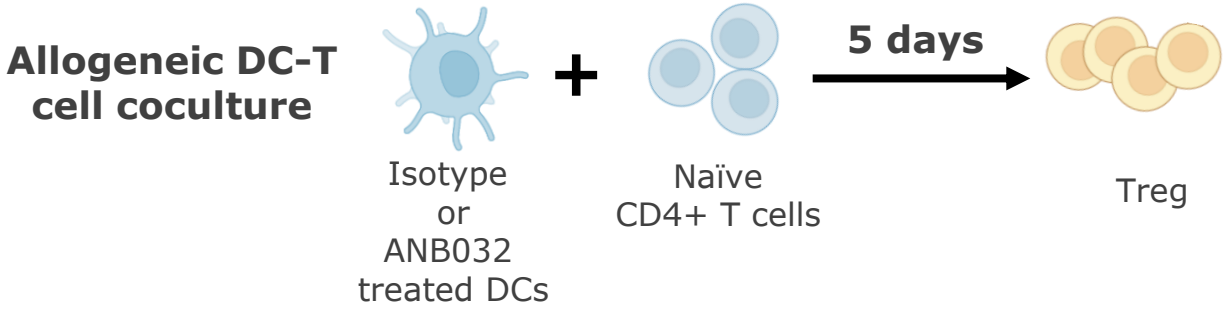
CD86

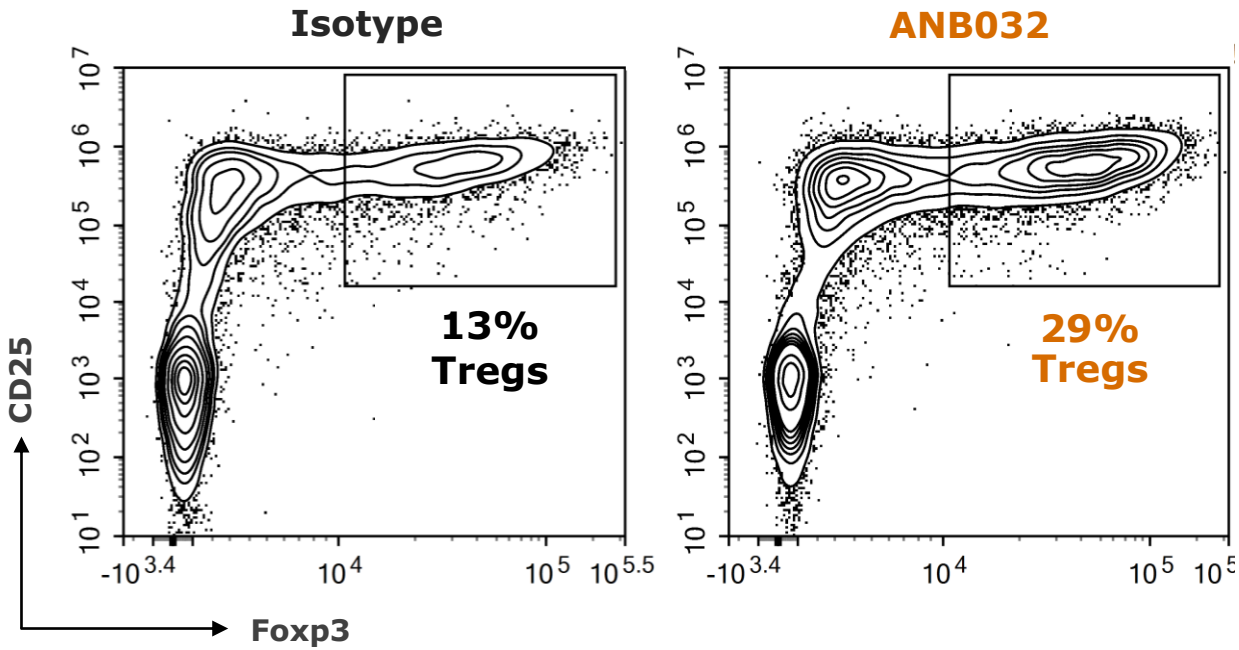


CD40



ANB032-treated DCs induce functional Tregs offering potential to restore immune balance





ANB032 inhibits T- and DC-mediated inflammation that are validated drivers of AD pathogenesis

BTLA pathway is dysregulated and BTLA is broadly expressed on T cells and dendritic cells that drive all phases of AD pathogenesis

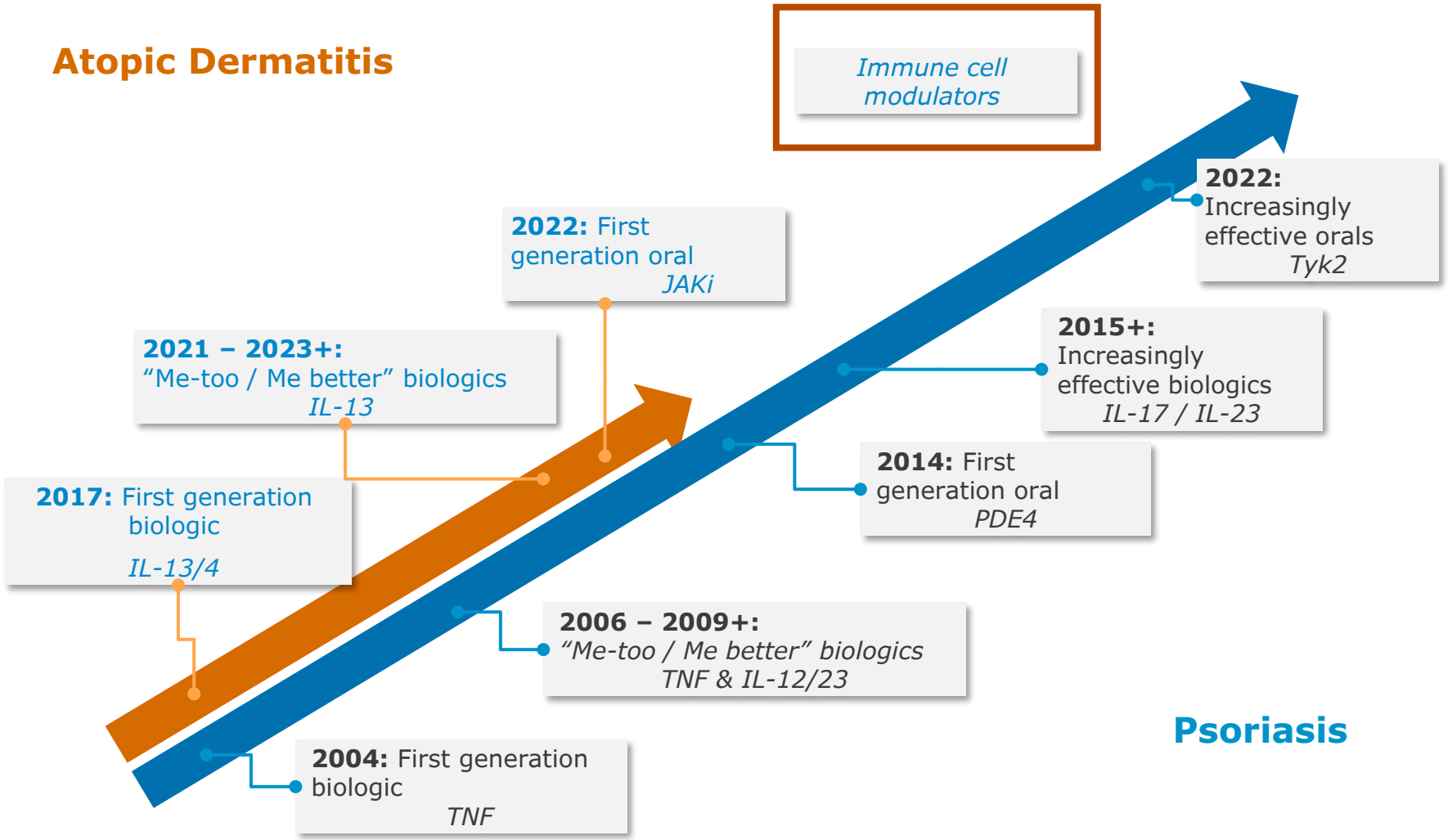
ANB032 directly inhibits proliferation of and cytokine secretion from AD patient-derived pathogenic Th1, Th2, Th17, Th22 cells

ANB032 modulates dendritic cells which enhances the direct inhibitory effect on T cells and induces Tregs

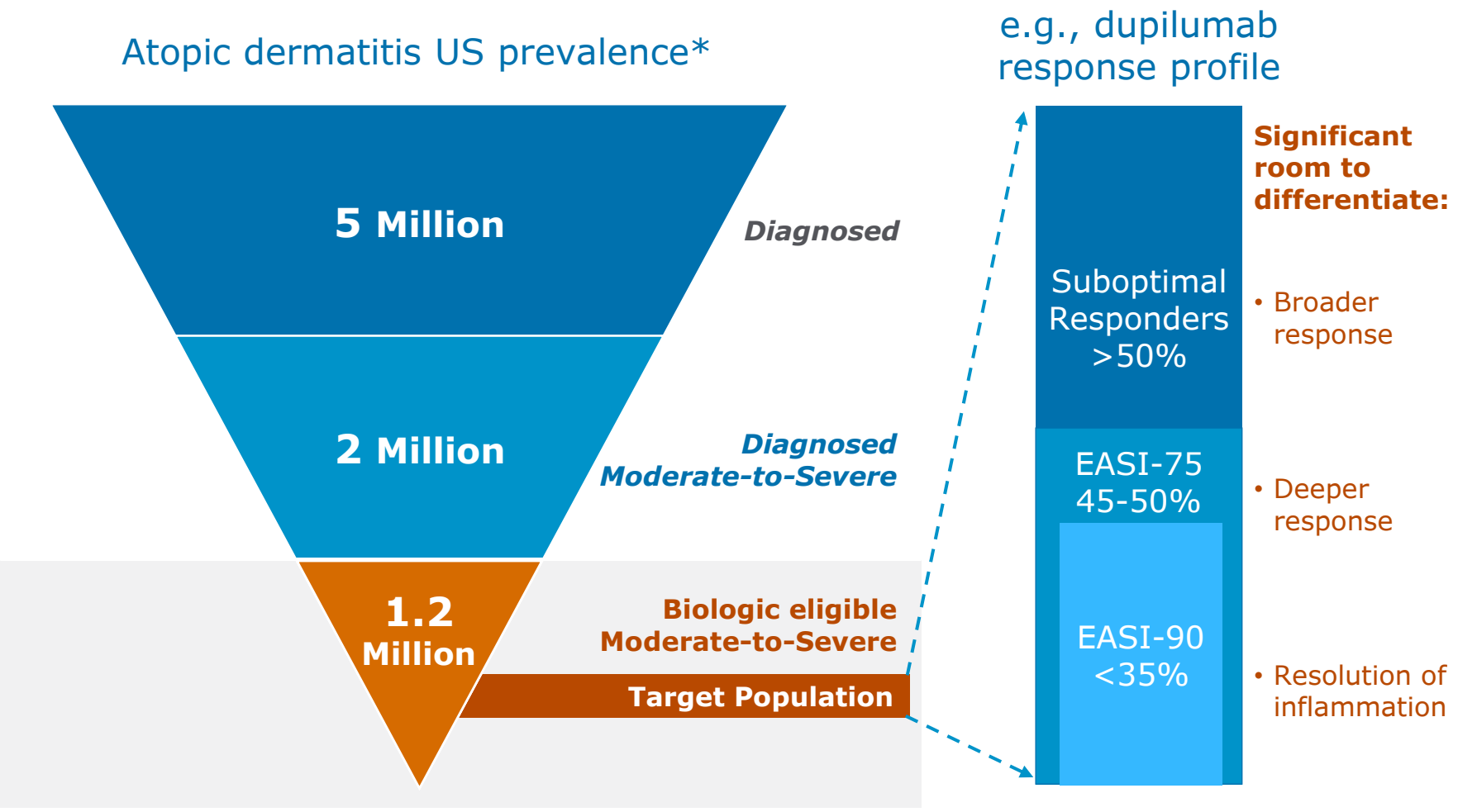
Agenda for today

TOPIC	SPEAKER
AnaptysBio: Best-In-Class Immune Cell Modulators	Dan Faga Chief Executive Officer
Checkpoint Agonism and ANB032, a BTLA Agonist	Martin Dahl, Ph.D. Senior Vice President, Research
Unmet Needs in Atopic Dermatitis (AD) and Need for Additional Targets	Emma Guttman, M.D., Ph.D. Professor and Chair of Dermatology Icahn School of Medicine, Mount Sinai
Targeting AD with ANB032: Translational Data	Martin Dahl, Ph.D. Senior Vice President, Research
ANB032: Phase 1 Results and AD Phase 2b Trial	Paul Lizzul, M.D., Ph.D. Chief Medical Officer
Closing Remarks	Dan Faga Chief Executive Officer
Q&A	AnaptysBio

Understanding disease pathology enables better therapies: AD mirrors psoriasis a decade ago

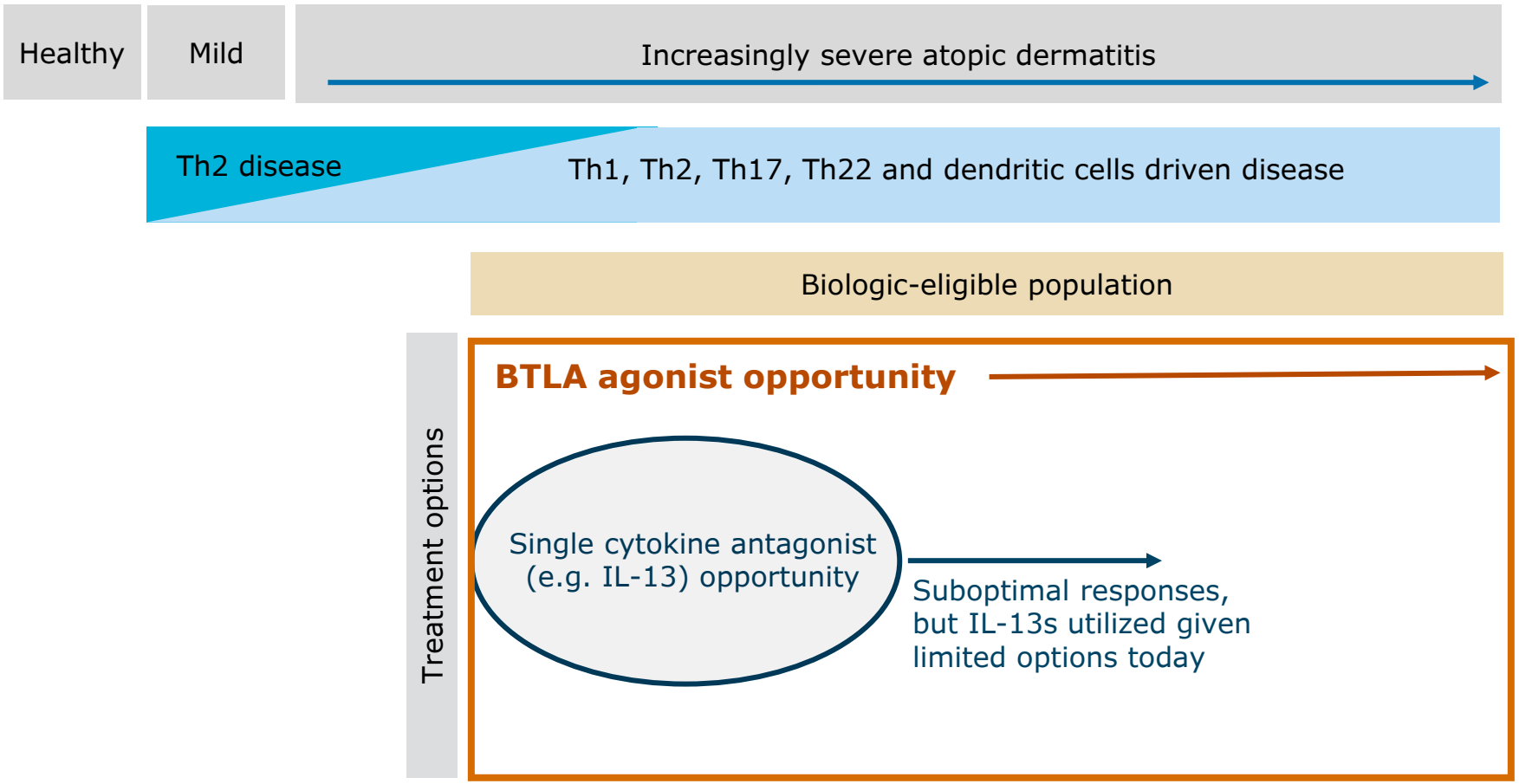


Opportunity for new biologic class with differentiated outcomes in AD regardless of prior treatment



*Claims analysis to determine market size based on 5 years of claims history; dupilumab responsive profile per prescribing information (label (fda.gov).

BTLA agonists may treat all stages of AD



ANB032 demonstrated favorable safety and tolerability with rapid and sustained PK/PD activity

96 healthy volunteers in SAD and MAD cohorts in Phase 1 study

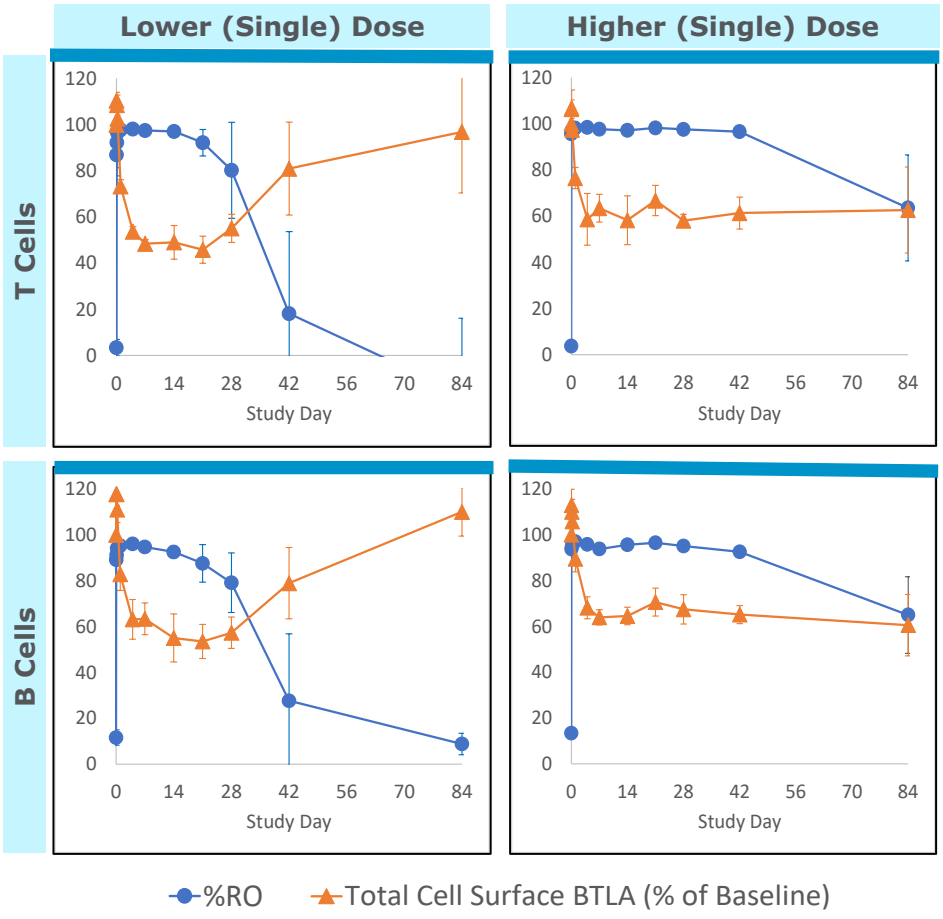
- Favorable ~2-week half-life with IV and SQ dosing
- Full receptor occupancy (RO) within hours and maintained for >30 days

Rapid and sustained target engagement on both T and B cells

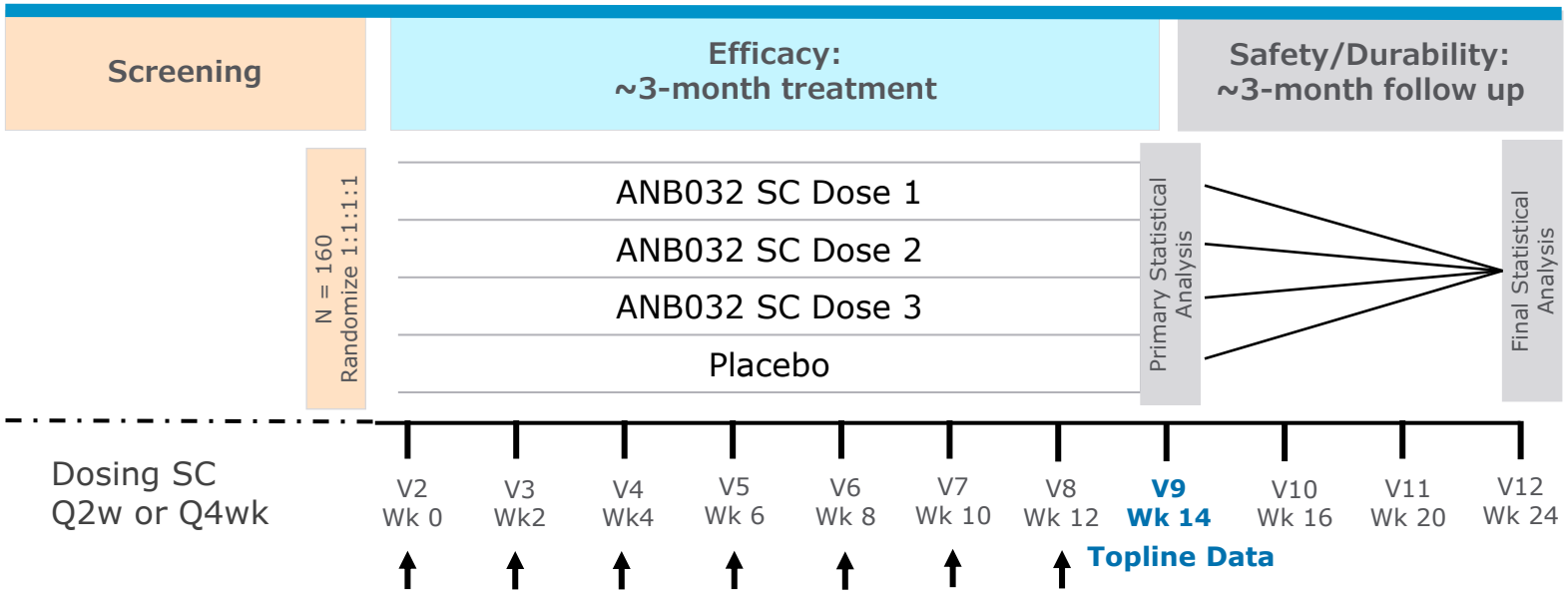
- Duration of reduced BTLA expression persisted in dose-dependent manner

Well-tolerated with no dose limiting tox

- No SAEs
- Most AEs mild-to-moderate, short duration, dose independent and resolved without sequelae
- No evidence of infection risk or cancer risk to date



ANB032 Phase 2b in IL-13 mAb naïve and experienced AD patients



Patient population		• Adults with moderate-to-severe atopic dermatitis* • Include both dupilumab/IL-13 naïve and experienced patients
Endpoints	Primary	• Mean change from Baseline in EASI at Week 14
	Secondary	• EASI-75 • vIGA-AD 0 (clear) or 1 (almost clear) and a ≥ 2-point reduction (improvement) • PNRS (itch), DLQI, SCORAD • Safety
Exploratory endpoints		• Th1/Th2/Th17 cytokines and other soluble biomarkers, tissue samples (tape strips, biopsies)

* Moderate-to-severe atopic dermatitis: at least 10% of their total body surface area (BSA), an Eczema Area and Severity Index (EASI) score ≥ 16, and a validated Investigator Global Assessment for Atopic Dermatitis (vIGA) score ≥ 3.

Explore predictive markers of treatment response

Goals

- Apply precision medicine to further development of ANB032 in AD and other diseases
- Profile immune impact and mechanistic markers of BTLA agonism
- Map immune changes from baseline onto current and future disease signatures

Periphery biomarkers

- T cell stimulation assay
- Transcriptomics
- Immunophenotyping (e.g., TBNK, Tregs)
- Soluble biomarkers and cytokines relevant to Th1/Th2/Th17/Th22 biology

Tissue biomarkers (tape strips and biopsies)

- Proteomics including cytokines
- Transcriptomics
- Immunophenotyping (e.g. TBNK, Tregs)

Top-line Week 14 data anticipated by YE 2024

Clinical team has deep experience
conducting dermatology clinical trials, including in AD

Global Trial	Extensive AD relationships	Recruitment initiatives
40+ dermatology investigators with proven track record enrolling AD trials	Long history of relationships with global KOLs in AD	Robust training program
North America and Europe trial sites	Strong medical society engagement	Community outreach/ advocacy group engagement
	Collaboration with experienced and proven CROs in AD	Social media campaign, patient resources and local advertising

ANB032 may address need for improved treatment outcomes for AD patients

- AD disease heterogeneity is not adequately addressed by standard of care
- ANB032 mechanism could enable broader and deeper responses to resolve inflammation and restore immune balance
- Phase 2b dose-finding study initiated in both IL-13 mAb naïve and experienced patients with moderate-to-severe AD
- Top-line Phase 2b results expected in Q4 2024

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ANB032: Phase 1 Results and AD Phase 2b Trial	Paul Lizzul, M.D., Ph.D. Chief Medical Officer
Closing Remarks	Dan Faga Chief Executive Officer
Q&A	AnaptysBio

ANB032's mechanism of action matches atopic dermatitis disease pathogenesis

Checkpoint agonism may deliver differentiated outcomes while restoring immune balance in inflammatory and autoimmune diseases

BTLA agonism inhibits activated T cell proliferation, reduces inflammatory cytokine secretion and modulates DC function including induction of Tregs

Th1, Th2, Th17, Th22 and dendritic cells drive AD pathogenesis

ANB032, supported with translational preclinical and Phase 1 safety data, has potential for deep responses across a broad patient population in AD

Global Phase 2b trial initiated with top-line data expected by year-end 2024

Immune cell modulator development: Three phase 2 initiations in 2023

Antibody Program	Therapeutic Indication	Development Stage and Anticipated Milestones				
		Lead Optimization	IND Enabling	Phase 1	Phase 2	Phase 3
Rosnilimab (PD-1 agonist)	**Rheumatoid Arthritis**				Phase 2b initiation Q3 2023 Top-line data mid 2025	
	Inflammatory Disease				Trial initiation YE 2023	
ANB032 (BTLA agonist)	**Atopic Dermatitis**				Phase 2b initiated Q2 2023 Top-line data YE 2024	
ANB033 (CD122 antagonist)	**Inflammatory Diseases**		IND submission H1 2024			