Breakfast Symposium ARGX-113 Phase 2 Study in Pemphigus Vulgaris

John Stanley, MD, University of Pennsylvania
Peter Verheesen, PhD, argenx
Patrick Dupuy, MD, argenx
Tim Van Hauwermeiren, CEO, argenx

November 10, 2017, New York

Forward Looking Statements

THIS PRESENTATION HAS BEEN PREPARED BY ARGENX SE ("ARGENX" OR THE "COMPANY") FOR INFORMATIONAL PURPOSES ONLY AND NOT FOR ANY OTHER PURPOSE. NOTHING CONTAINED IN THIS PRESENTATION IS, OR SHOULD BE CONSTRUED AS, A RECOMMENDATION, PROMISE OR REPRESENTATION BY THE PRESENTER OR THE COMPANY OR ANY DIRECTOR, EMPLOYEE, AGENT, OR ADVISER OF THE COMPANY. THE MATERIALS PRESENTED BY DR. STANLEY WERE PREPARED BY HIM INDEPENDENTLY AND CONTAIN HIS OWN VIEWS. AS A RESULT, THE COMPANY TAKES NO RESPONSIBILITY FOR DR. STANLEY'S MATERIALS.THIS PRESENTATION DOES NOT PURPORT TO BE ALL-INCLUSIVE OR TO CONTAIN ALL OF THE INFORMATION YOU MAY DESIRE. THIS PRESENTATION ALSO CONTAINS ESTIMATES AND OTHER STATISTICAL DATA MADE BY INDEPENDENT PARTIES AND BY US RELATING TO MARKET SIZE AND GROWTH AND OTHER DATA ABOUT OUR INDUSTRY. THIS DATA INVOLVES A NUMBER OF ASSUMPTIONS AND LIMITATIONS, AND YOU ARE CAUTIONED NOT TO GIVE UNDUE WEIGHT TO SUCH ESTIMATES.

Safe Harbor: Certain statements contained in this presentation, other than present and historical facts and conditions independently verifiable at the date hereof, may constitute forward-looking statements. Examples of such forward-looking statements include those regarding our investigational product candidates and preclinical and clinical trials and the status and related results thereto, future results of operations and financial positions, business strategy, plans and our objectives for future operations. When used in this presentation, the words "anticipate," "believe," "can," "could," "estimate," "expect," "intend," "is designed to," "may," "might," "will," "plan," "potential," "predict," "objective," "should," or the negative of these and similar expressions identify forward-looking statements. Such statements, based as they are on the current analysis and expectations of management, inherently involve numerous risks and uncertainties, known and unknown, many of which are beyond the Company's control. Such risks include, but are not limited to: the impact of general economic conditions, general conditions in the biopharmaceutical industries, changes in the global and regional regulatory environments in the jurisdictions in which the Company does or plans to do business, market volatility, fluctuations in costs and changes to the competitive environment. Consequently, actual future results may differ materially from the anticipated results expressed in the forward-looking statements. In the case of forward-looking statements regarding investigational product candidates and continuing further development efforts, specific risks which could cause actual results to differ materially from the Company's current analysis and expectations include: failure to demonstrate the safety, tolerability and efficacy of our product candidates; final and quality controlled verification of data and the related analyses; the expense and uncertainty of obtaining regulatory approval, including from the U.S. Food and Drug Administration and European Medicines Agency; the possibility of having to conduct additional clinical trials and our reliance on third parties such as our licensors and collaboration partners regarding our suite of technologies and product candidates. Further, even if regulatory approval is obtained, biopharmaceutical products are generally subject to stringent on-going governmental regulation, challenges in gaining market acceptance and competition. These statements are also subject to a number of material risks and uncertainties that are described in the Company's filings with the U.S. Securities and Exchange Commission ("SEC"), including in the final prospectus related to the Company's initial U.S. public offering filed with the SEC pursuant to Rule 424(b) of the Securities Act of 1933, as amended, as well as subsequent filings and reports filed by the Company with the SEC. The reader should not place undue reliance on any forward-looking statements included in this presentation. These statements speak only as of the date made and the Company is under no obligation and disavows any obligation to update or revise such statements as a result of any event, circumstances or otherwise, unless required by applicable legislation or regulation.

Agenda

08:30 **Welcome & Introduction**
Tim Van Hauwermeiren, CEO argenx

08:35 **ARGX-113: Phase 2 Study in pemphigus vulgaris**

- Introduction on pemphigus vulgaris
John Stanley, MD, Dermatology University of Pennsylvania School of Medicine

- Preclinical and clinical rationale
Peter Verheesen, PhD, argenx

- Phase 2 clinical trial in pemphigus vulgaris
Patrick Dupuy, MD, argenx

09:35 **ARGX-113: Pipeline-in-product opportunity**
Tim Van Hauwermeiren, CEO argenx

09:45 **Q&A**

Prof. John Stanley

- Since 1995: Professor, Department of Dermatology, University of Pennsylvania, Philadelphia, PA
- Prof. Stanley's contributions to the Science (i.a.):
  - Pemphigus antigens: Molecularly cloned the pemphigus vulgaris antigen and showed it was a new member of the desmoglein gene family, desmoglein 3. These antigen characterizations led to the clinical use of ELISA assays to diagnose and evaluate severity of disease.
  - Pemphigus pathophysiology: Developed and validated the "Desmoglein Compensation Theory" in pemphigus. This concept explains the blister localization in pemphigus vulgaris.
  - Defining the antibody repertoire in pemphigus: Genetically and proteomically characterized the IgG-B cell antibody repertoire in pemphigus vulgaris and shown that the same non-tolerant (i.e. anti-desmoglein) B cells and antibodies persist over a decade in patients, and that patients rarely develop new non-tolerant anti-desmoglein B cells emerging from the marrow.

Pemphigus vulgaris: Introduction

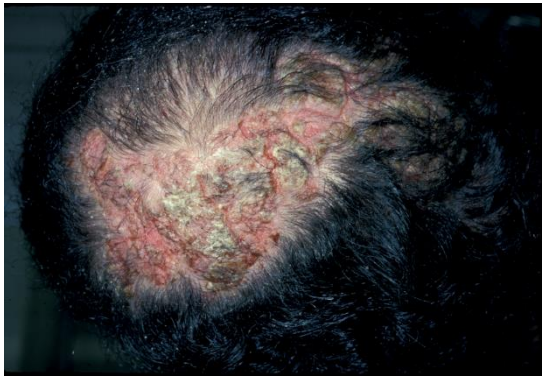
Therapies for Pemphigus

Pemphigus vulgaris

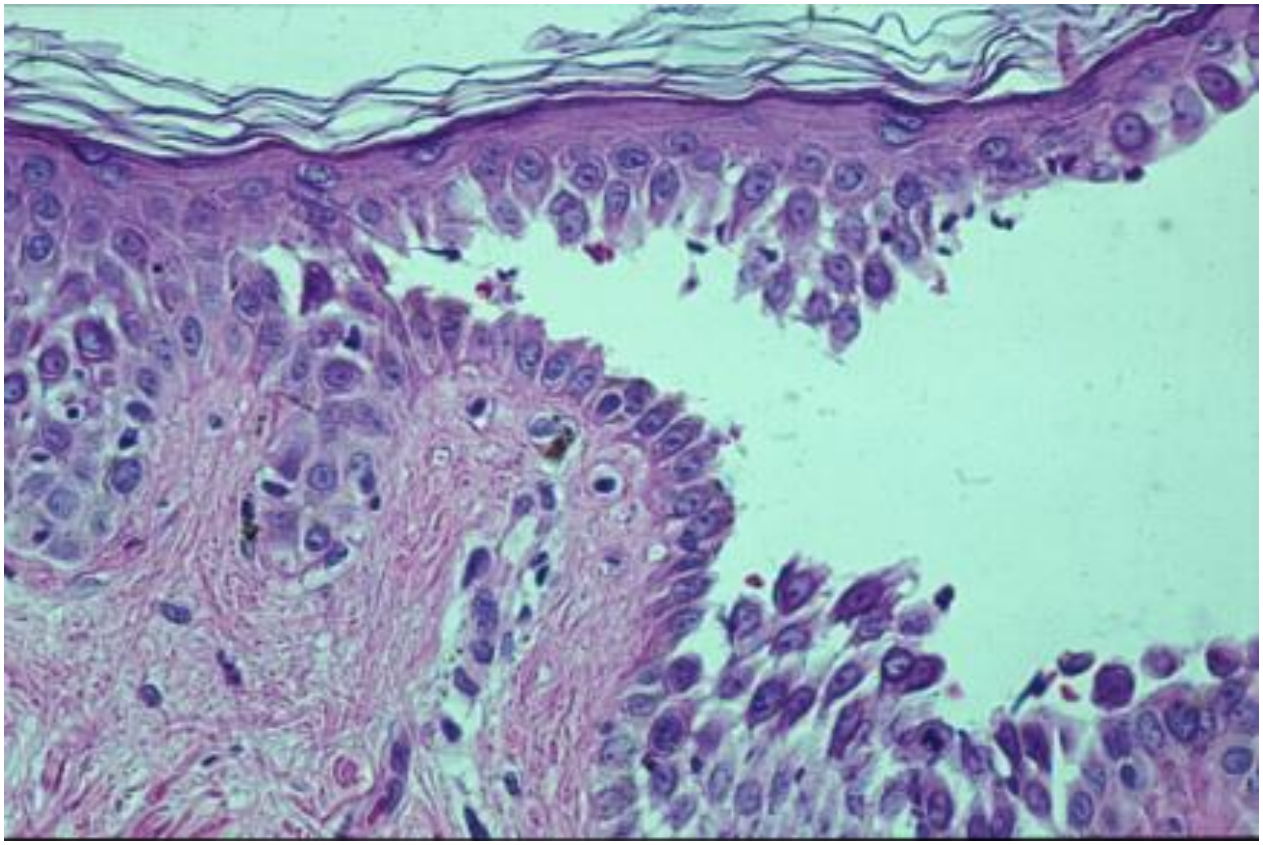
- Acutely can be severe and life-threatening
- After therapy can remit and relapse
- May become chronic with low level disease
- With current therapy some patients have long term remissions off therapy (? Cures)

Pemphigus vulgaris (PV)

PV chronic and persistent



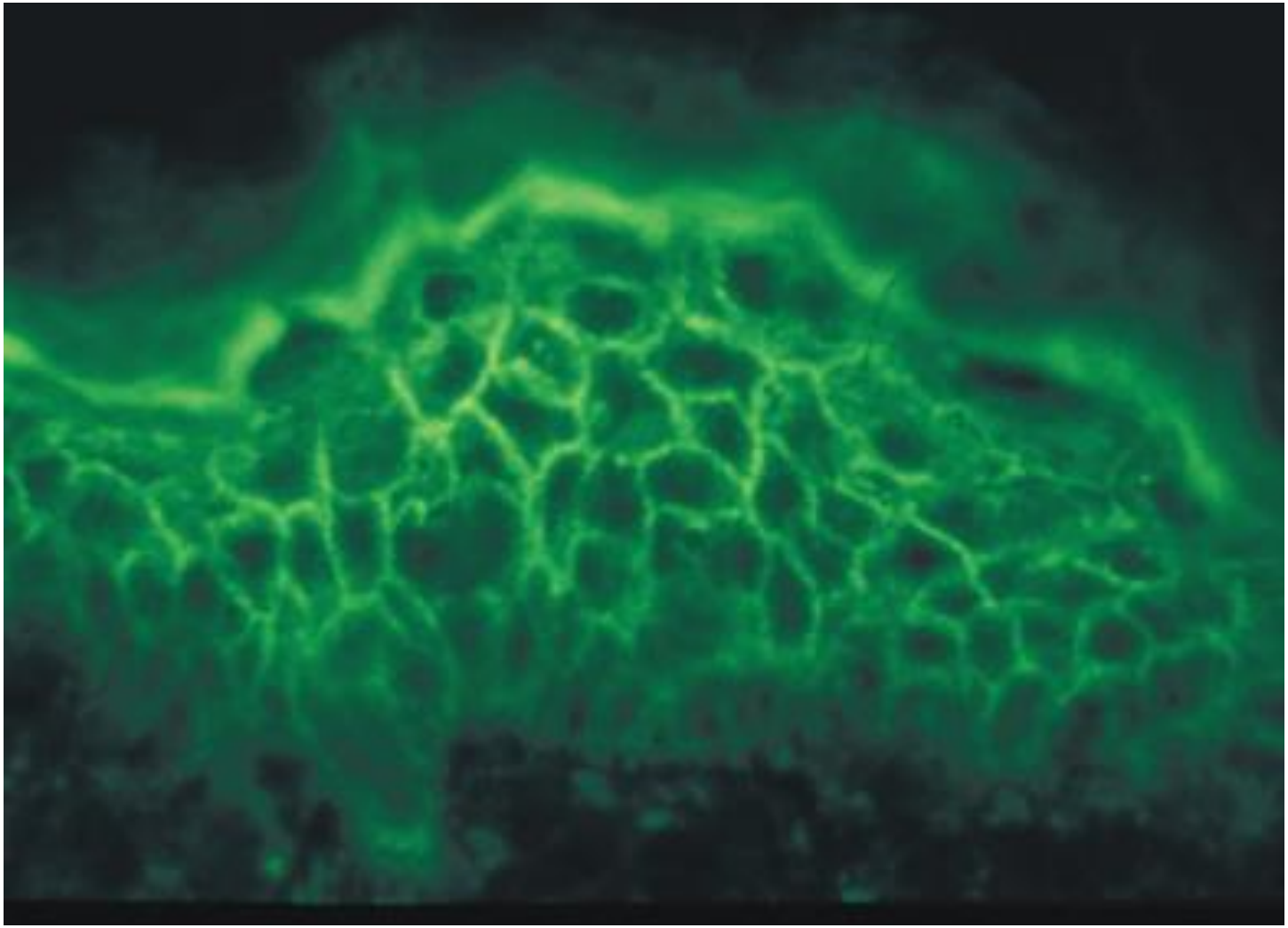
PV: histology



Epidemiology

- Age of onset: peak 50-70 years
- Incidence PV per 1M/year
  - Jerusalem: 16 (Ashkenazi Jews HLA-DRB1*0402)
  - UK: 7 (BMJ Langan SM et al, 2008)
  - France, Germany, Switzerland, Finland: 0.5-1
  - Connecticut: 4.2, Jewish pop 32 (Arch Derm Simon DG et al, 1980)
- Approx. 30-40,000 cases in US (IPPF Website)
- Deaths PV in US
  - 1979-2002: 1226
  - Mortality 2000-2002: 0.2 per 1M per year
- Hospitalization US (JAMA Derm Hsu D et al, 2016)
  - 8 per 1 million hospitalizations
  - Cost per pt: $14-16,000 (cf. $18,200 for MI)
  - Total hosp costs 2012: PV 1^0 dx: $7 mil; PV 2^0 dx: $40 mil

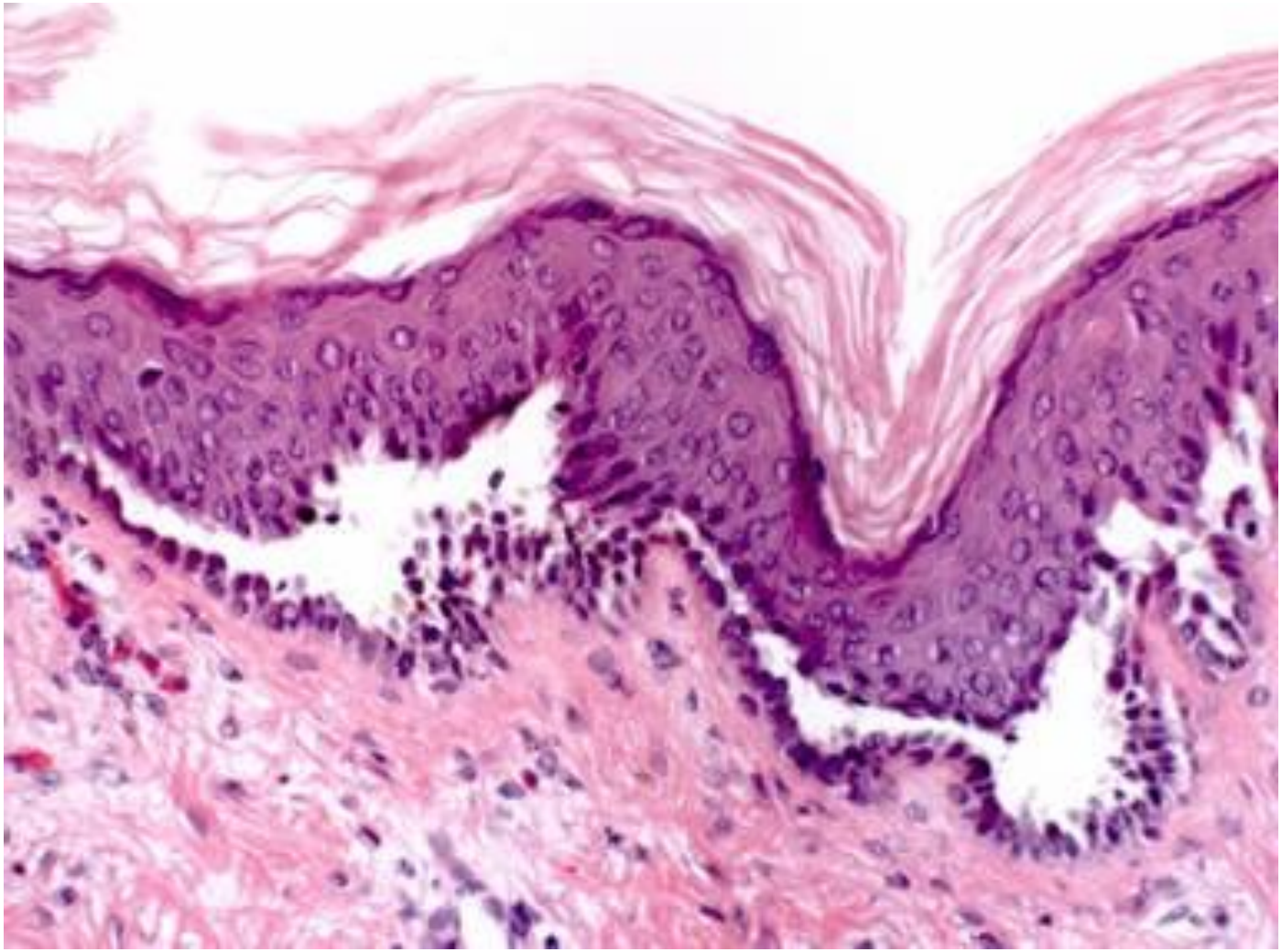
Pemphigus: IIF



Pemphigus autoantibodies are pathogenic

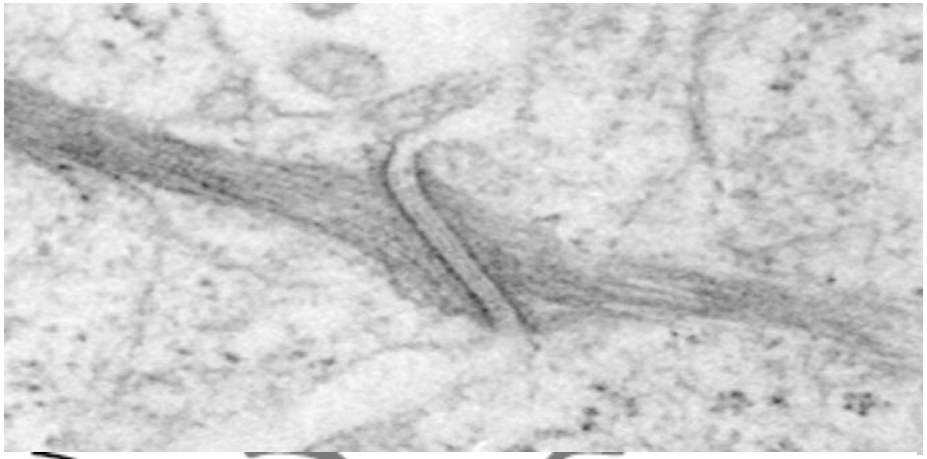
- Clinical observations
  - –antibody titer and disease activity
  - –neonatal pemphigus
- Experimental observation
  - –skin organ culture
  - –passive transfer to neonatal mice

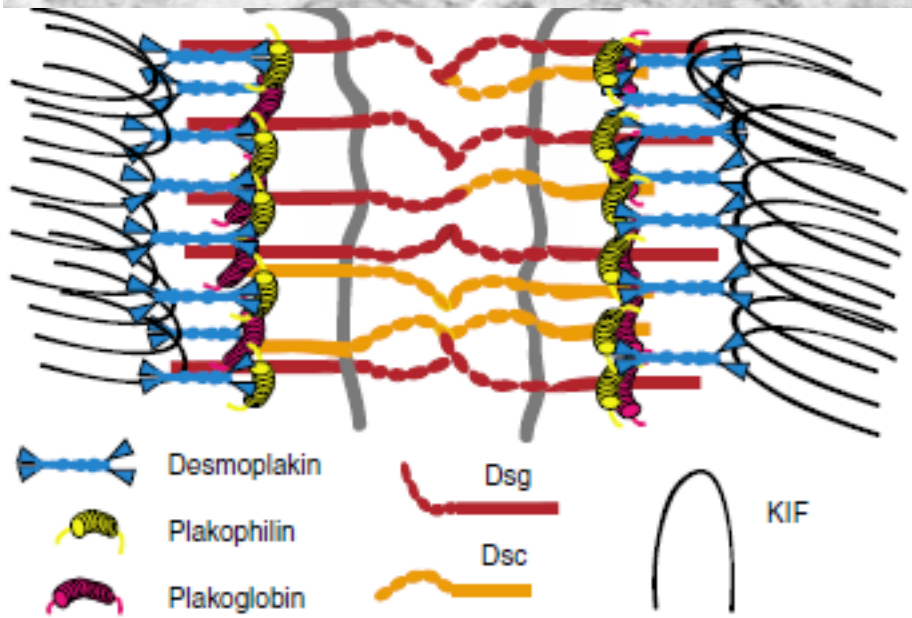
PV antibody injected in normal skin



Discovery that pemphigus is an anti-desmosomal autoimmune disease

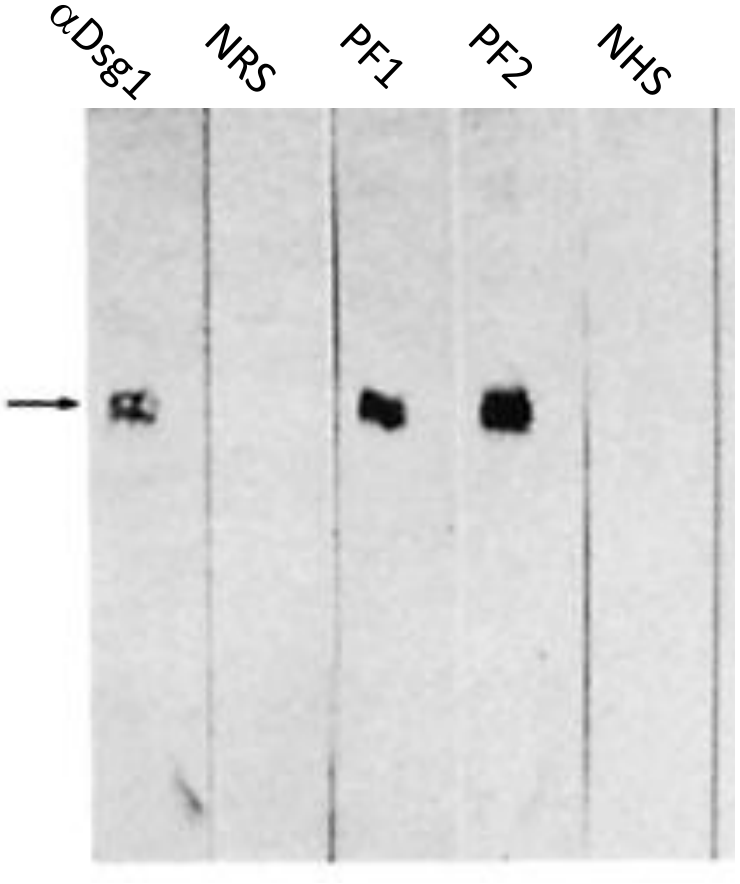
Desmosomes hold cells together; in pemphigus autoantibodies cause cells to fall apart





Kottke MD et al J Cell Sci 119:797, 2006

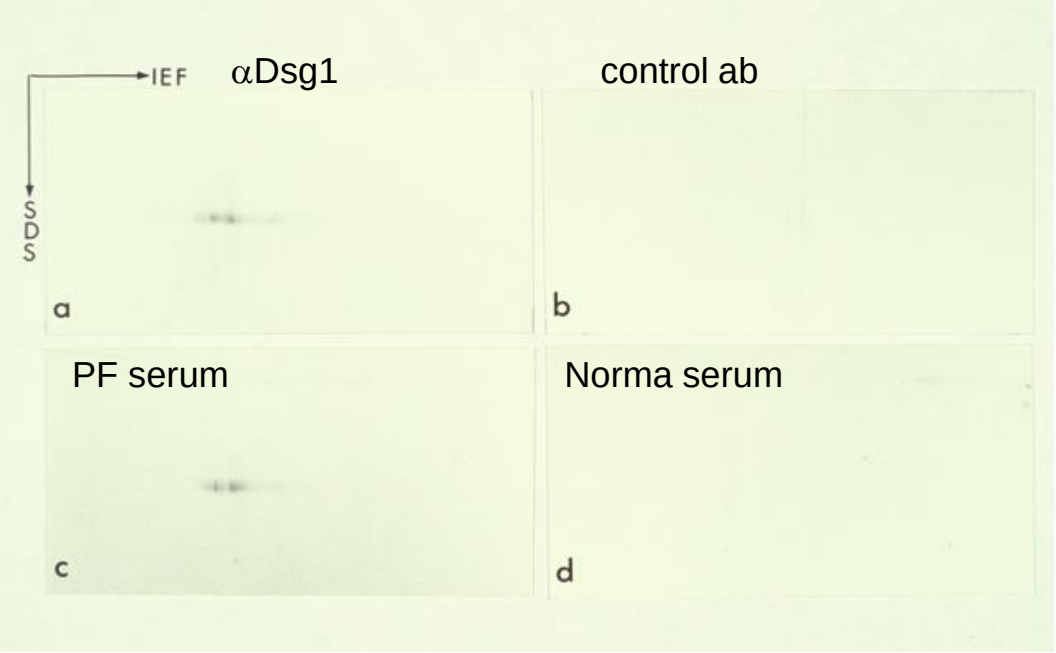
Immunoblot: desmoglein 1 (Dsg1) co-migrates with PF antigen



Koulu , Kusumi, Steinberg, Klaus-Kovtun, Stanley. J Exp Med 160:1509, 1984

PFAg is desmoglein 1 (Dsg1): first autoimmune disease targeting desmosome

2-D immunoblot
of epidermal extract

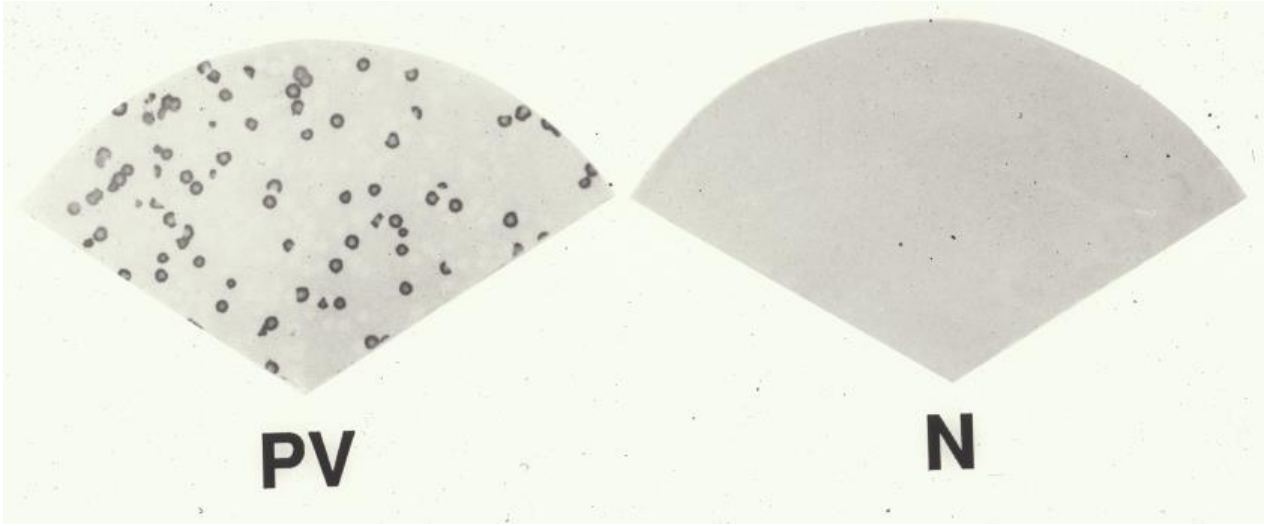


Koulu, Kusumi, Steinberg, Klaus-Kovtun,Stanley, **JExpMed** 1984

Stanley,Koulu,Klaus-Kovtun,Steinberg **J Immunol** 1986

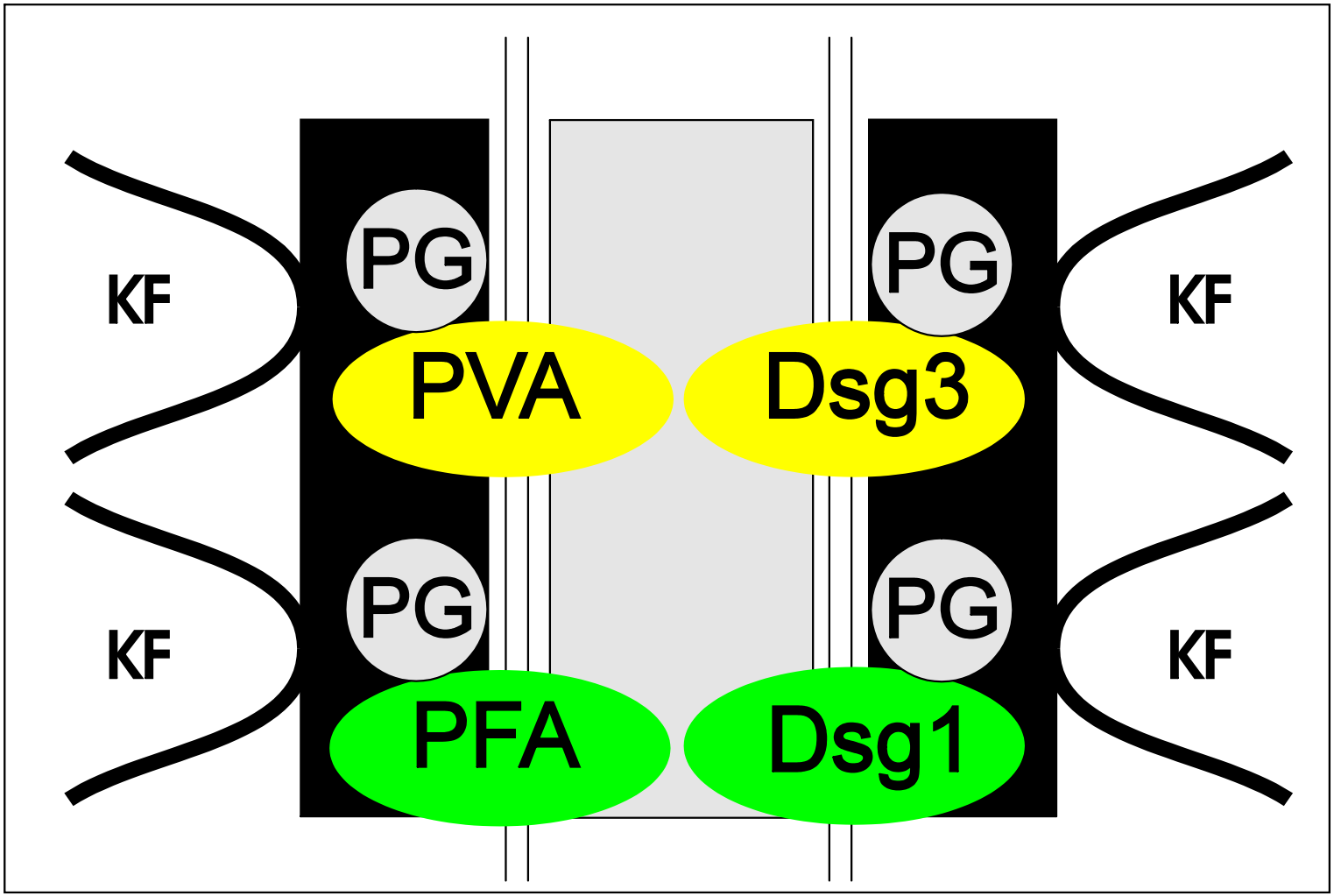
Molecular cloning indicates PV antigen is a previously unknown desmoglein, now called desmoglein 3

λgt11 clone expressing pemphigus antigen



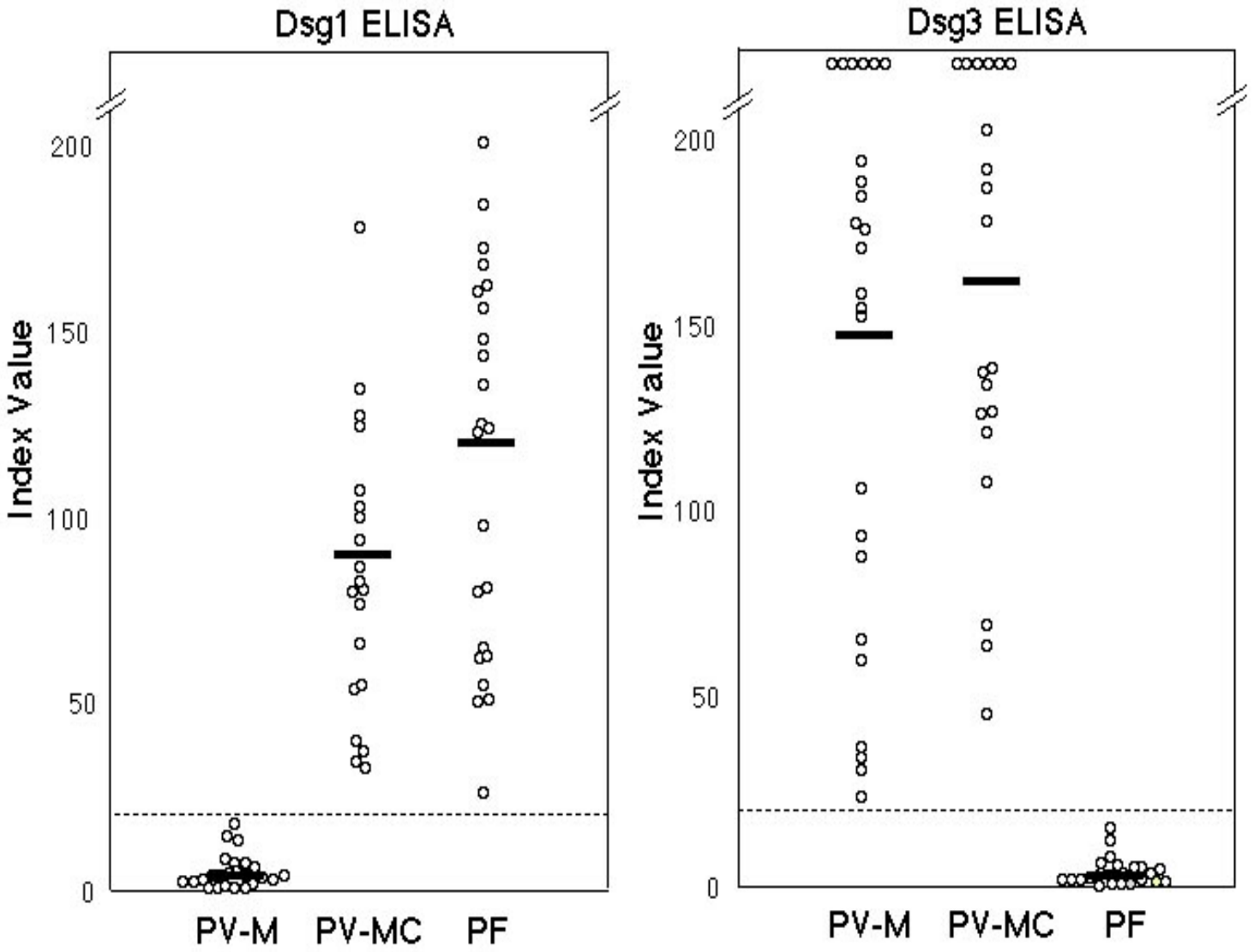
Amagai,Klaus-Kovtun, Stanley, **Cell** 1991

Pemphigus antibodies bind desmogleins, cell adhesion molecules in desmosomes



ELISA

anti-Dsg antibodies in PF and PV



Pemphigus disease activity correlates with anti-desmoglein antibody level

- IIF titer
- ELISA titer (BJD, Cheng SW et al, 2002; JEADV Kwon EJ et al, 2008)
- Effective therapies decrease autoantibodies
- Patients in remission usually have low or undetectable antibodies

Basic principles of therapy of pemphigus

- Decreasing autoantibody levels will decrease disease activity
- Eliminating B cells that produce antibody will cure disease
- Possible: eliminate anti-Dsg3 T cells or T reg therapy

Major current therapies

- Non-targeted therapies induce general immunosuppression with many off target effects and adverse reactions
  - Prednisone
  - Azathioprine/mycophenolate

- Antibody and B-cell directed therapies
  - Plasmapheresis/adsorption
  - IVIg probably decreases autoantibodies by catabolism of all antibodies
  - Rituximab: targets B-cells that make pemphigus antibodies but not plasma cells that make antibodies induced by immunizations and infectious diseases

Rituximab

- Chimeric human-murine anti-CD20
- Binds all B-cells except B stem cells
  - B cells absent from blood for 6-12 mos.
- Does not bind plasma cells
- Pemphigus anti-Dsg3 and anti-Dsg1 antibodies decrease, often to undetectable
  - Anti-tetanus toxoid, V-Z do not
- Rituximab induces long term remission off therapy in about 50% of patients probably because it eliminates all non-tolerant anti-desmoglein B cells necessary to produce pemphigus antibodies

Rituximab as primary therapy

Joly et al Lancet 389:2031, 2017

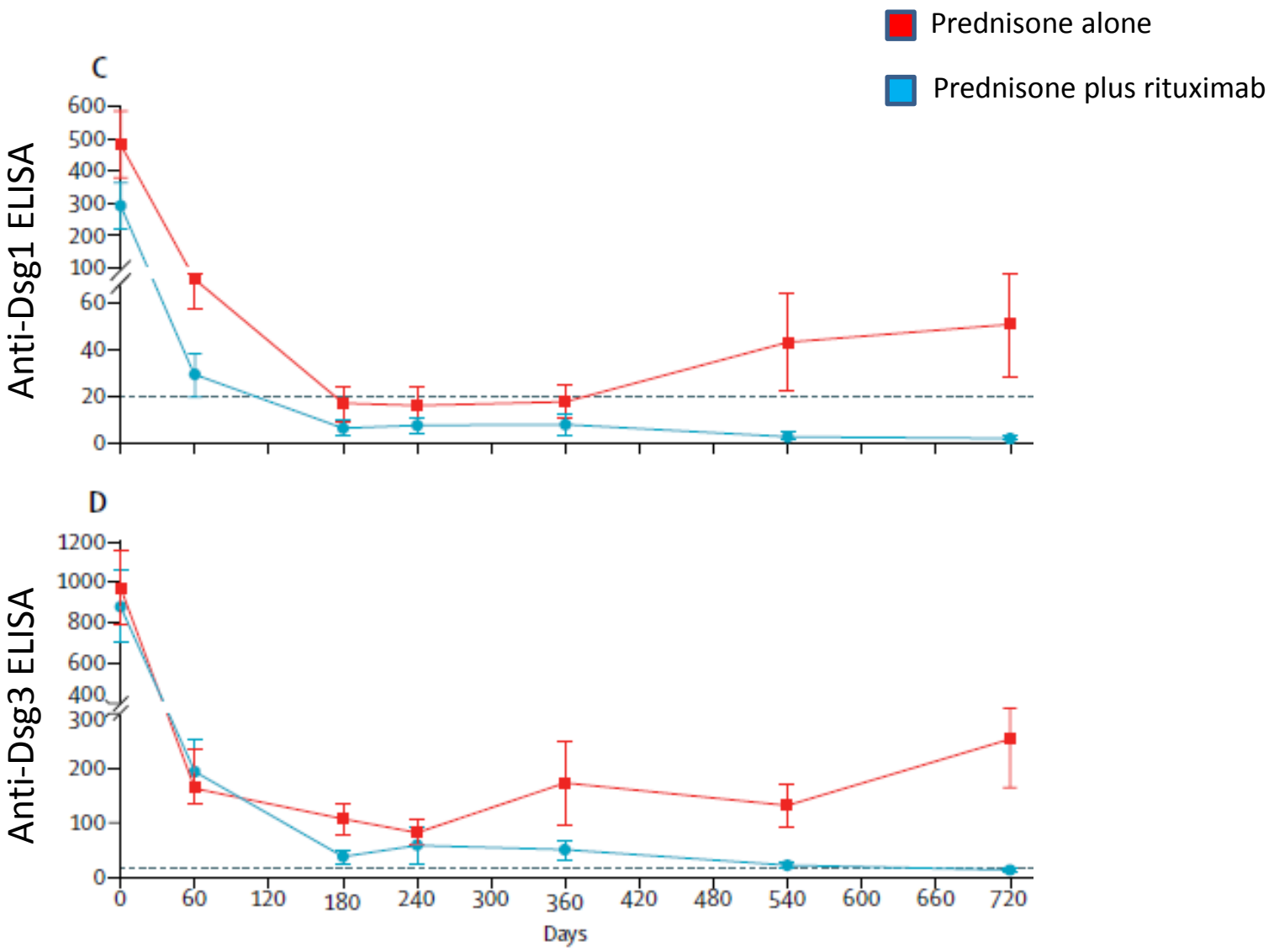
- 90 pemphigus patients (82% PV)
- Therapies
  - Rituximab plus prednisone taper from 0.5-1 mg/kg/day
  - Prednisone taper from 1-1.5 mg/kg/day
- Primary end point: CR of therapy (CROT) at month 24

Rituximab as primary therapy

Joly et al Lancet 389:2031, 2017

	Pred alone	Rituximab plus pred
CROT at 24 mos	34%	89%
Median time to CROT (d)	677	277
Median duration CROT (d)	62	446
Relapse	45%	24%
2 yr disease free	37%	75%
Severe adverse effects per pt *	1.2	0.59

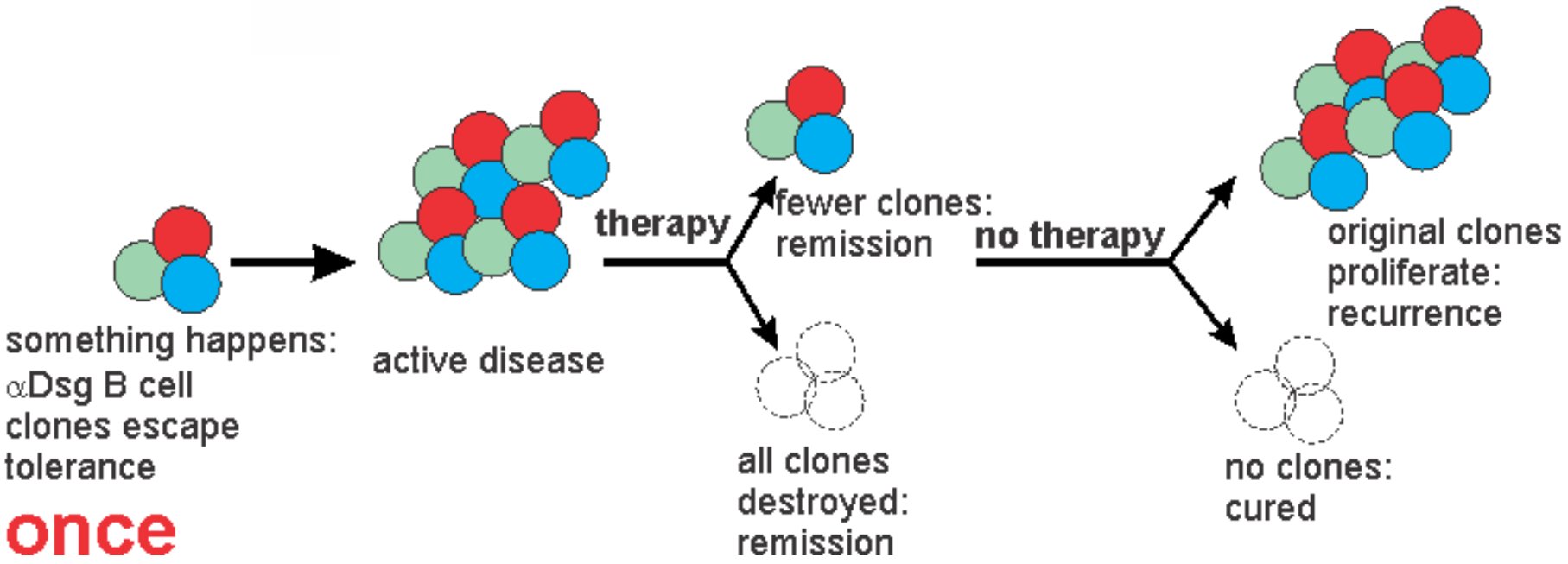
*diabetes, myopathy, bone disease most common; no deaths in either group


Prednisone alone
Prednisone plus rituximab
C
Anti-Dsg1 ELISA
600
500
400
300
200
100
60
40
20
0
D
Anti-Dsg3 ELISA
1200
1000
800
600
400
300
200
100
0
0
60
120
180
240
300
360
420
480
540
600
660
720
Days

Rituximab adverse side effects

- 0-6.4% serious adverse effects
- Infection
  - reactivation HBV
  - other viral
  - bacterial
- Progressive multifocal leukoencephalopathy
  - JC virus (50% of population seropositive)
- TEN
- Neutropenia
  - one to several months after therapy
  - usually asymptomatic but if concomitant immunsuppression may have infectious complication
- Immediate hypersensitivity

Why pemphigus recurs after therapy



Potential cure in pemphigus

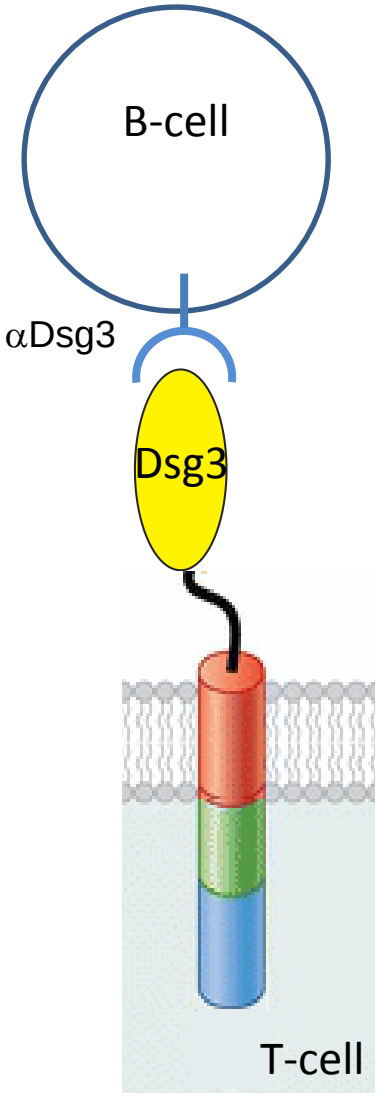
- Pemphigus patients do not have an ongoing defect in tolerance to desmoglein
- Pemphigus probably results in a one time “hit” with development of a few non-tolerant B cell clones
- If the oligoclonal B cells can be eliminated, disease can be cured

Therapeutic goals for pemphigus

- Decrease or eliminate autoantibodies with minimal off target effects on immune system
- To induce cure treatment aim is to eliminate all anti-Dsg B cells and/or T-cells
- Therapies that only decrease autoantibody titer (without eliminating anti-Dsg B and/or T cells) will be effective but probably not induce cure
  - Valuable in acute, severe disease

Future therapies

- argenx
- Chimeric autoantigen receptor cell based therapy
- Target anti-desmoglein T cell or use regulatory T cells



ARGX-113: Preclinical and clinical rationale for pemphigus vulgaris

Pemphigus vulgaris autoantibodies induce skin lesions

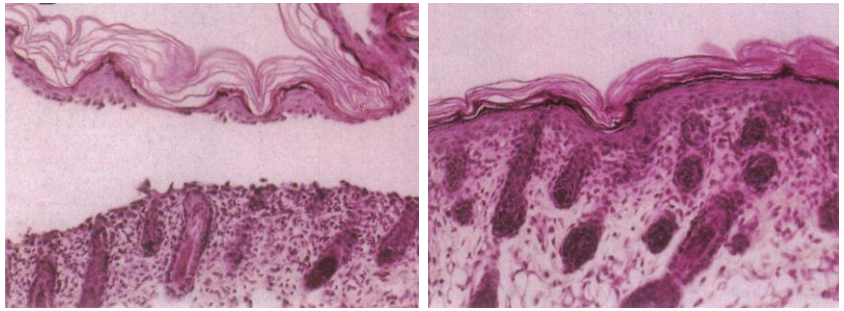
Autoantibody levels correlate with disease severity

Patient serum derived IgG with PV autoantibodies

Patient serum derived IgG with non-active anti-Dsg3

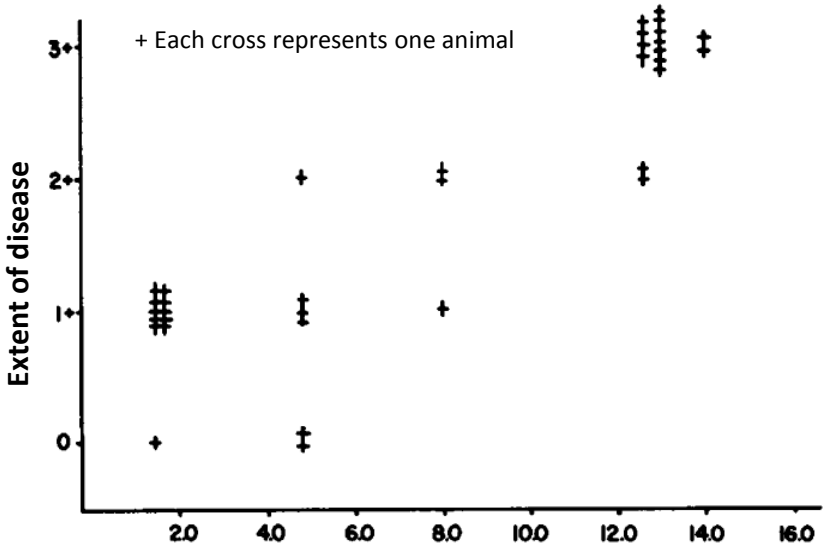
Blisters and erosions | **Healthy skin**

Acantholysis | **No apparent acantholysis**



Amagai et al. 1994, J. Clin Invest.

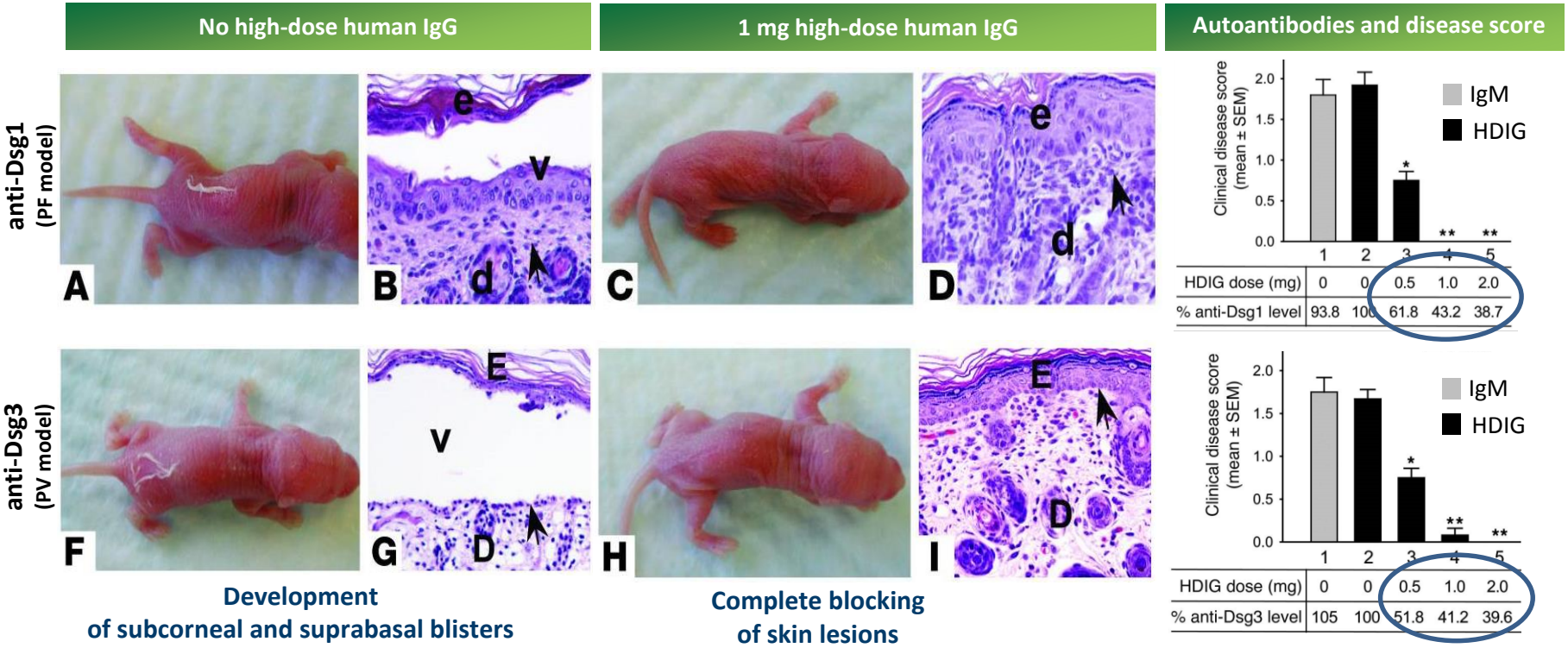
Mice injected with patient derived IgG with PV autoantibodies



Anhalt et al. 1982, NEJM

- Autoantibodies (anti-Dsg3 and anti-Dsg1) cause blisters and erosions in mice
- Direct correlation between human autoantibodies levels and disease severity in mice

High-dose human IgG prevents skin lesion formation in mice

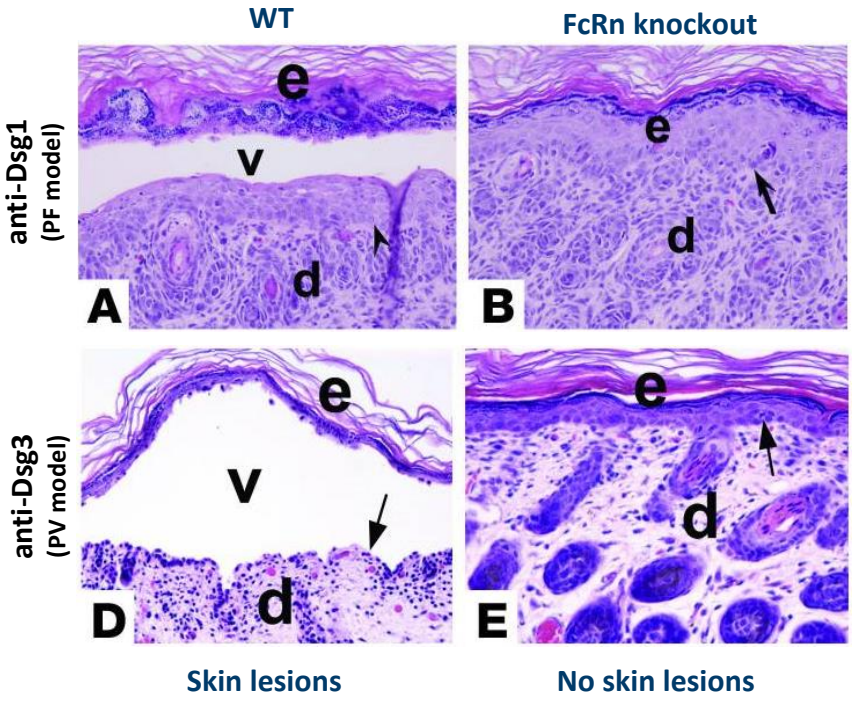


- Pre-treatment of mice with high-dose human IgG:
  - accelerates catabolism of pathogenic anti-Dsg1 and anti-Dsg3
  - prevents skin blisters
  - inhibits experimental pemphigus
- Lowering of autoantibodies levels correlates with reduction of clinical disease score

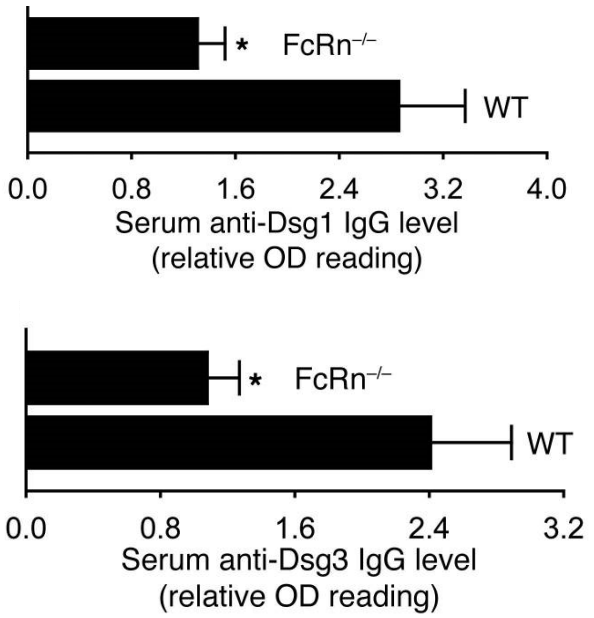
Li et al. 2005, J. Clin Invest.

FcRn-deficient mice resistant to experimental pemphigus

FcRn-deficient mice + anti-Dsg1/3



Lower anti-Dsg1/3 levels in FcRn-deficient mice



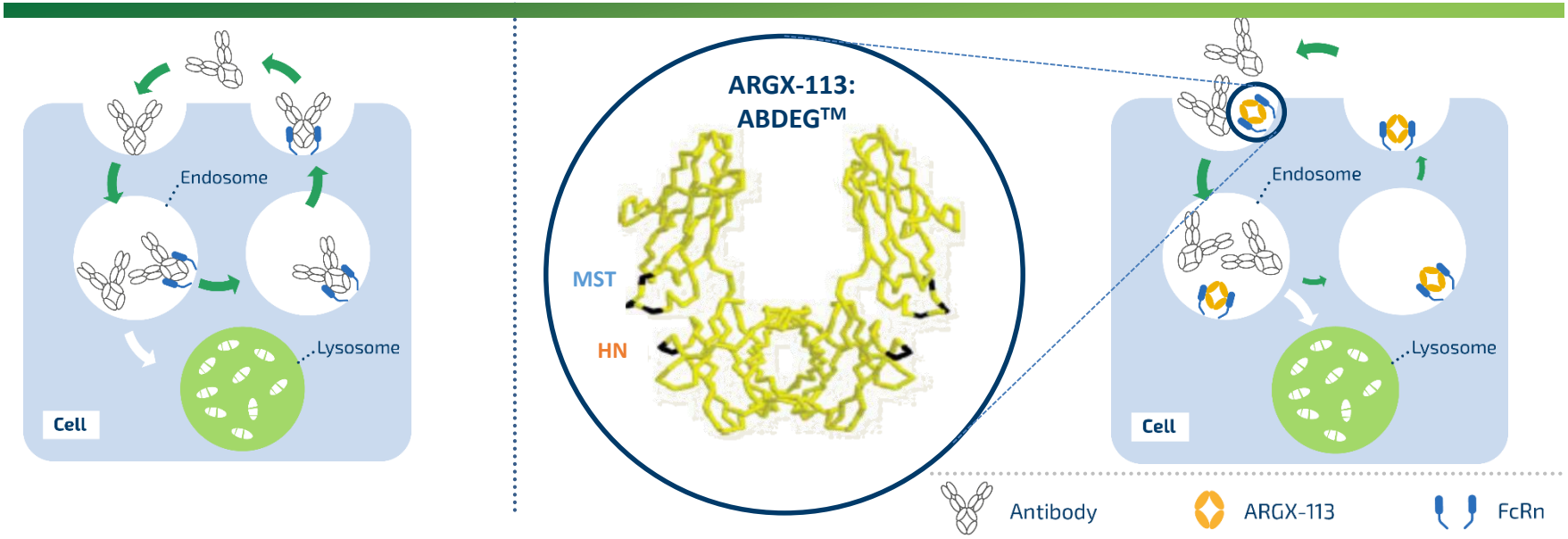
- FcRn-deficient mice resistant to anti-Dsg1/3 → reduction in circulating pathogenic IgG
- Faster degradation prevents pathogenic IgG from reaching skin tissue targets → skin blistering abolished

Li et al. 2005, J. Clin Invest.

ARGX-113: Lead program based on novel target FcRn

An innovative approach to eliminate IgG autoantibodies

IgG antibodies recycle through FcRn(1)… **…ARGX-113 blocks FcRn…** **…leading to IgG elimination**



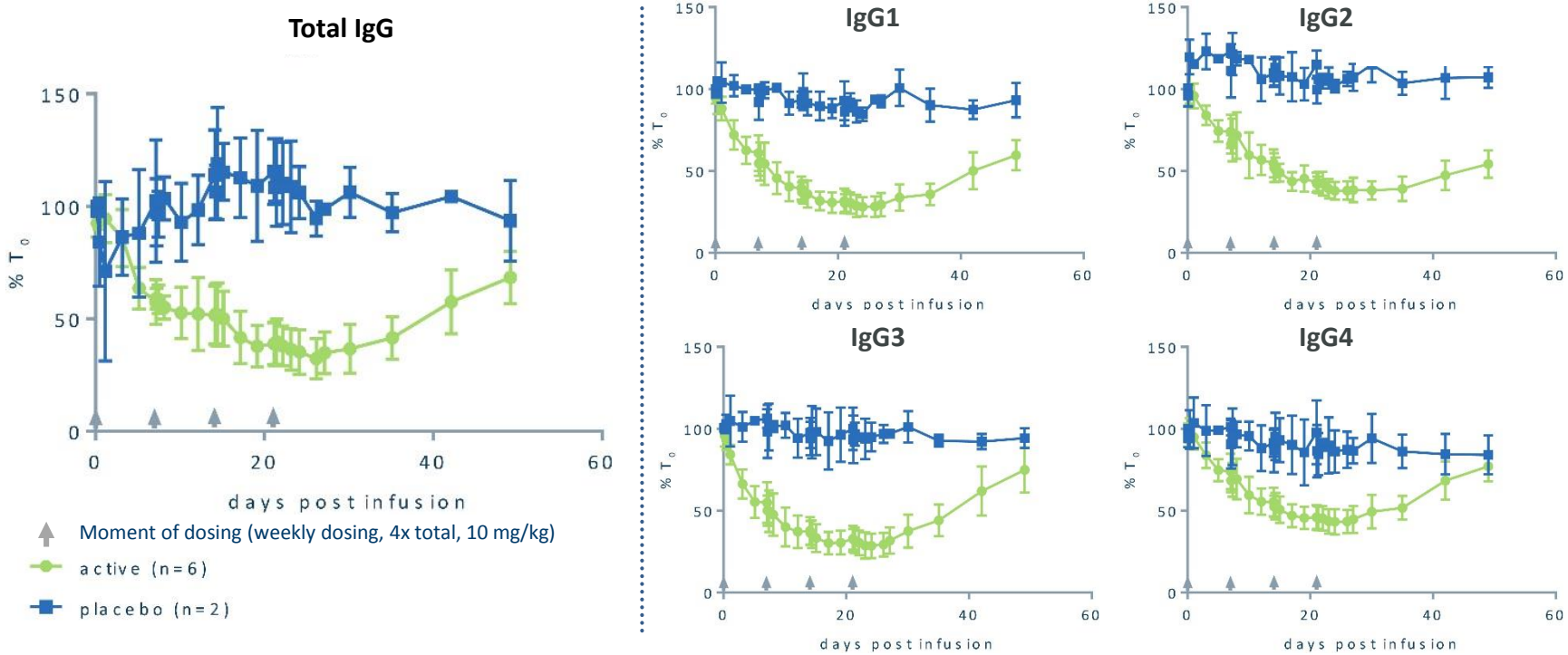
- ARGX-113 is a human IgG1 Fc-fragment that utilizes ABDEG™ Fc engineering technology(2)(3)
- ARGX-113 targets and binds to FcRn blocking the recycling of IgG leading to an elimination of IgG antibodies
- Demonstrated 50% to 85% reduction of circulating IgG antibody levels in Phase 1 trial
- Pathogenic IgG antibodies mediate multiple autoimmune diseases
- There is a correlation between disease severity and autoantibody levels in pemphigus vulgaris

(1) Roopenian et al. 2007, Nat Rev Immunol.
(2) Vaccaro et al. 2005, Nat Biotech.
(3) argenx data

ARGX-113: IgG reduction seen in Phase 1

PD data multiple ascending dose (MAD) study in healthy volunteers

Dosing 10 mg/kg; every 7 days

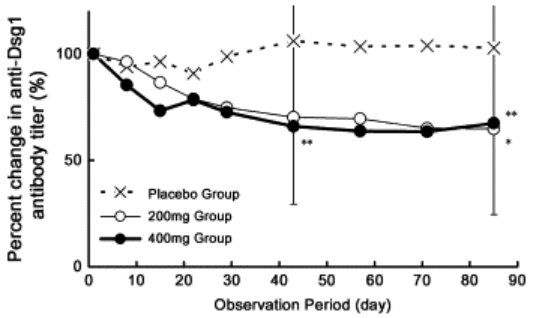


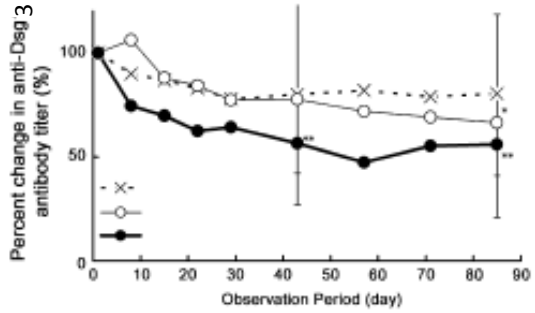
- IgG reduction across subclasses
- IgG reduction: 50% achieved in 1 week; up to 85% maximum reduction
- Selective IgG reduction, not affecting IgM/IgA and albumin levels (data not shown)
- Pathogenic anti-Dsg antibodies in PV are from IgG1 and IgG4 subclasses
- Total IgG reduction of 10 mg/kg dosing every 7 days for 4 weeks (median 69%; range: 55-83%)

Pemphigus vulgaris patients benefit from lowering autoantibodies

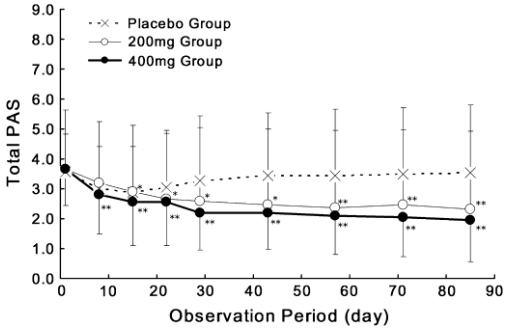
IVIg treatment

anti-Dsg1 and anti-Dsg3 levels after IVIg treatment

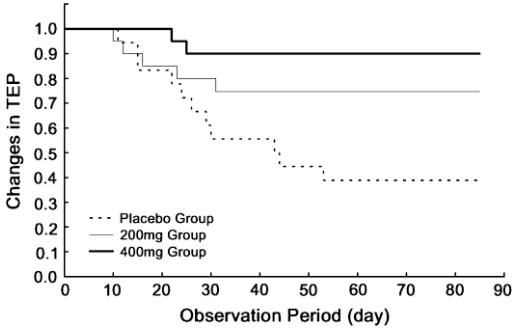




PAS after IVIg treatment



TEP after IVIg treatment

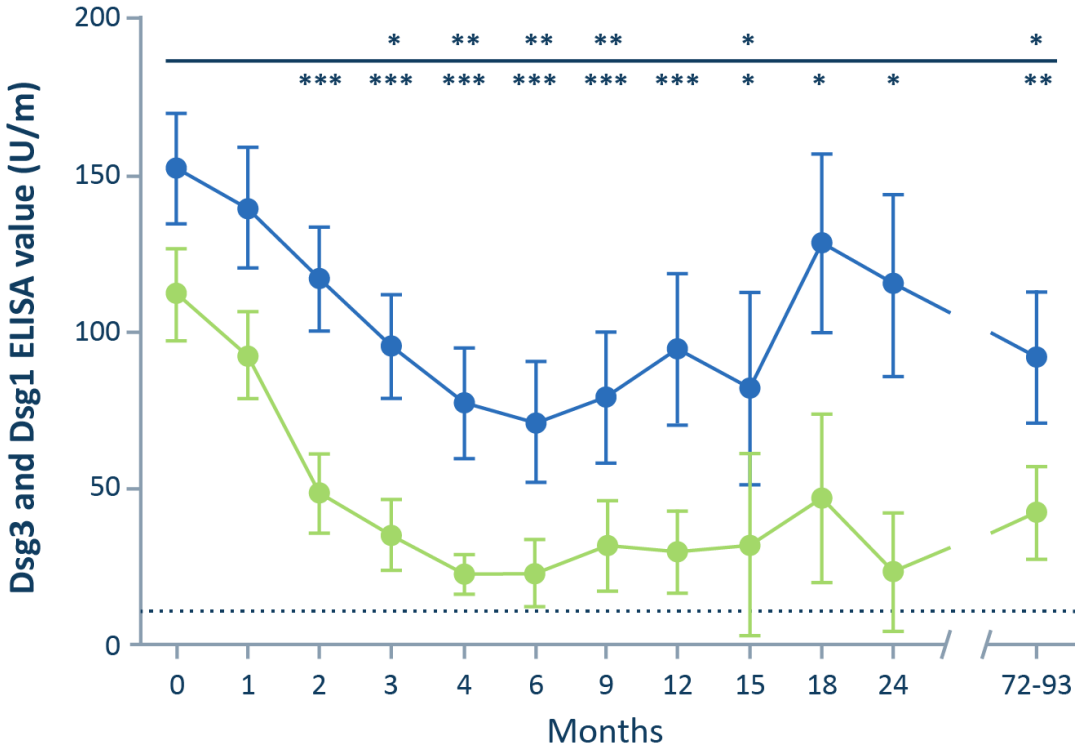


- IVIg treatment lowers anti-Dsg1 and anti-Dsg3 significantly
  - Correlates with lower total Pemphigus Activity Score (**PAS**)
  - Prolongs time to escape from the protocol (**TEP**) 10% and 25% after 90 days

Amagai et al. 2009, J. Am. Acad. Dermatol

Pemphigus vulgaris patients benefit from lowering autoantibodies

Rituximab treatment



- Rituximab treatment: B-cell depletion results in significant lower anti-Dsg1 and anti-Dsg3 levels
- Autoantibodies levels reduced after several weeks to months

Colliou et al. 2013, Autoimmunity

ARGX-113: favorable safety and tolerability profile

Phase 1 study in healthy volunteers

	Placebo	SAD					MAD		
ARGX-113 (mg/kg)		**0.2**	**2**	**10**	**25**	**50**	**10 (q4d)**	**10 (q7d)**	**25 (q7d)**
N (total number of subjects)	18*	4	4	4	4	4	6	6	12**
INVESTIGATIONS									
Diff. WBC count abnormal					3	4			
C-Reactive protein increased					2	4			1
NERVOUS SYSTEM DISORDERS									
Headache	4				1	3	1		3
Dizziness	1					2			
Somnolence									1
MUSCULOSKELETAL AND CONNECTIVE TISSUE DISORDERS									
Back pain	2					1			
Myalgia						1			
Pain in extremity						1			
GASTROINTESTINAL DISORDERS									
Nausea						1			
Abnormal discomfort	1								1
GENERAL DISORDERS AND ADMINISTRATION SITE CONDITIONS									
Chills						1+ 1			2
Fatigue	2								2
Feeling cold	2								1
Malaise									1
Pyrexia									1
EYE DISORDERS									
Photophobia						1			
Eye paresthesia								1	
SKIN AND SUBCUTANEOUS TISSUE DISORDERS									
Hyperhidrosis	1					1			
Rash macular								1	
Rash maculo-papular									1
BLOOD AND LYMPHATIC SYSTEM DISORDERS									
Lymphadenopathy								1	

Moderate AE

Other= Mild AE

AEs that were considered possibly, probably, or likely-related to treatment (ARGX-113 vs. placebo)

ARGX-113: Phase 2 clinical trial in pemphigus vulgaris

ARGX-113: Proof-of-concept study in pemphigus vulgaris

Pemphigus vulgaris

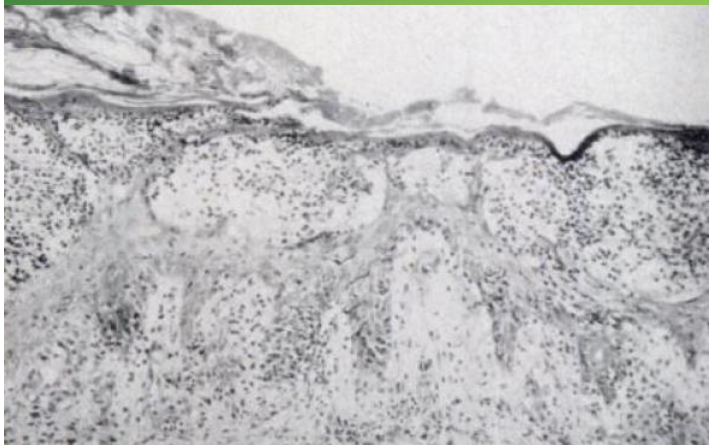
- Antibody-driven disease
- Strong biomarkers (serum IgG, anti-Dsg 3 +/- 1) can be used to anticipate clinical activity
- High medical needs:
  - Early disease control
  - Keep patients under remission safely
  - Current treatments (e.g. high dose corticosteroids, immunosuppressant) = co-morbidity factors

For updated review: Kasperkiewicz et al. 2017, Nature Reviews

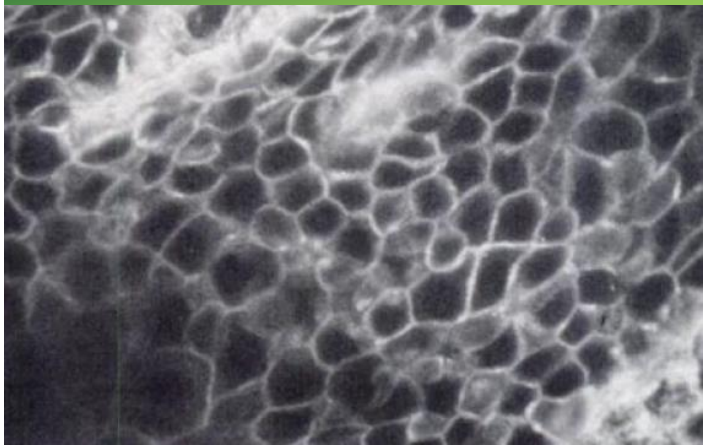
Neonatal pemphigus vulgaris: Passive transfer of maternal IgG causing the disease

Erosive lesions

Acantholysis



Maternal IgG in newborn skin

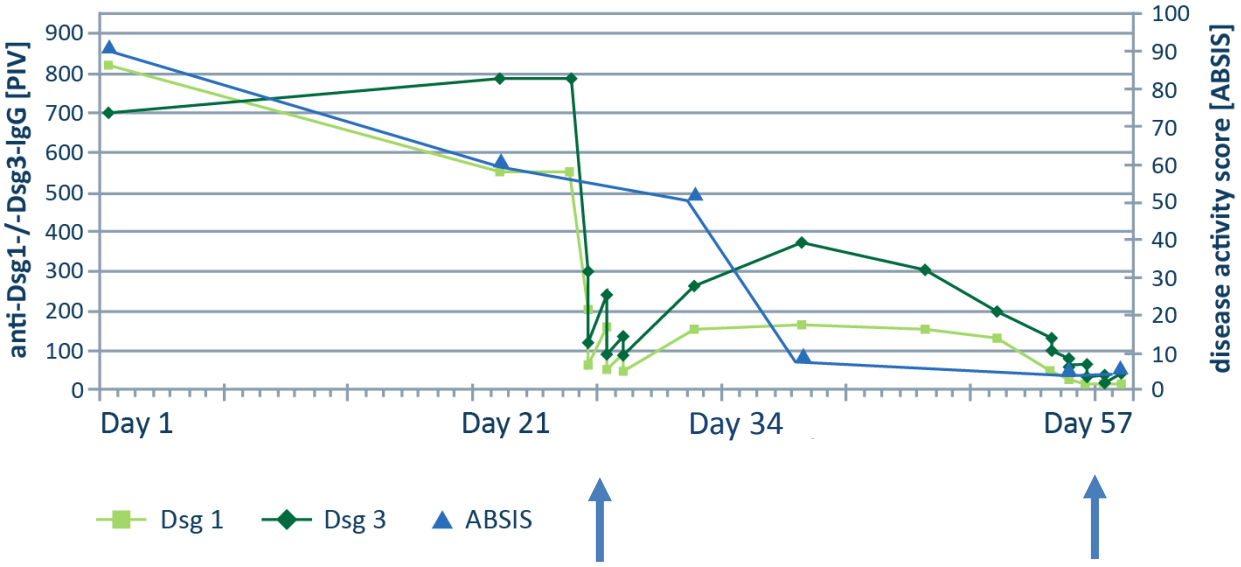


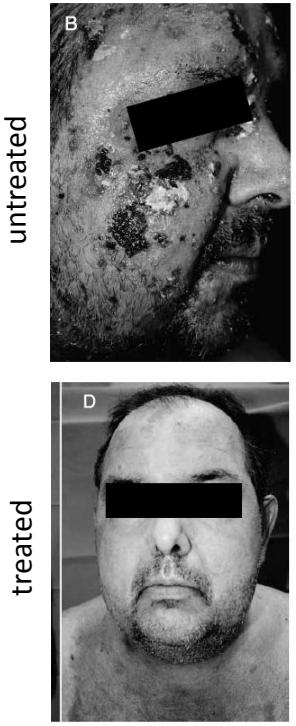
Dupuy and Metzger, 1985, Communication Franch Society of Dermatology

Pemphigus vulgaris patients benefit from lowering autoantibodies

Immunoadsorption treatment

anti-Dsg1 and anti-Dsg3 levels after immunoadsorption treatment





- Immunoadsorption (2 cycles) reduces circulating autoantibodies (anti-Dsg1 and anti-Dsg3) rapidly
  - 70-80 % reduction of total IgG
  - 50-70% reduction of autoantibodies
  - Disease control within 4-8 weeks

Eming et al. 2006, Autoimmunity

Current PV treatment can manage the disease, but carries significant health risks and damages quality of life

I just sat down for a snack and I realized I had a huge blood blister on my palate. Being a dentist I knew there could be a serious reason for this

My health deteriorated quickly, and in only five weeks I moved from being strong and healthy to being unable to eat and even walk around the city. I kept thinking, "This can't be true". I had no idea how living with a chronic disease would change my life

After several months of seeing doctors, three biopsies, and numerous hospital waiting rooms, I found out I had Pemphigus Vulgaris. I was started on high dose Prednisone

There's no cure, no smart drug to take it all away. It is nearly always controllable, but that control comes at a heavy cost.

The slightest change to my medication regimen could disturb the precarious balance, and instead of controlling the disease I'd be tumbling down into another crisis. Once I started Azathioprine, it did get a little easier though.

I found it very hard to accept that my health was badly damaged and I couldn't return to my old self. High dose corticosteroids and azathioprine had been necessary, and saved my life, but I was now disabled with problems walking, pain, and poor eye sight.

Two years after starting steroids, I'm finally off them. But, if I stop taking small doses of azathioprine, the blisters always come back.

At a conference, I heard about Rituximab. I didn't want to rock the boat and risk my health. But, I deserve to be symptom free. Few doctors have expertise with Rituximab, so it was difficult finding the right doctor to go through treatment with me.

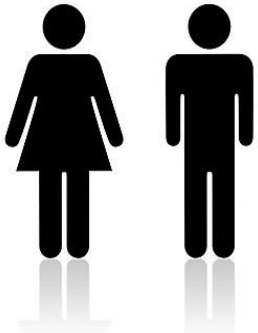
©2016 Collective Acumen
Quotes from patient blogs

ARGX-113 Phase 2 study: Objectives

- Demonstrate pharmacologic effect of ARGX-113 in an IgG-driven disease
- Assess the safety and efficacy of ARGX-113 in pemphigus vulgaris (disease control, treatment consolidation)
- Prepare design of further trials in the indication

ARGX-113 Phase 2 study: Exploratory, open-label, non-controlled

12 pemphigus vulgaris adults
Mild to moderate



Newly diagnosed
or relapsing

ARGX-113

9 weeks treatment + Follow up

Primary study assessment

- Incidence & severity of adverse events
- Physical examination
- Vital signs
- Electrocardiogram
- Laboratory values

Secondary study assessments

- Pharmacodynamics (serum total IgG & pathogenic IgG)
- Efficacy (PDAI, time to disease control, time until relapse)
- Pharmacokinetics
- Immunogenicity

- Newly diagnosed and relapsing patients off therapy: ARGX-113 used as monotherapy
- Relapsing patients under minimal prednisone: prednisone kept at stable dosage
- First line, long term treatment: effect of ARGX-113 at induction phase (3 weeks) and maintenance

PDAI: Pemphigus disease area index

Skin	Activity		Damage
Anatomical location	Erosion/Blisters or new erythema		Post-inflammatory hyperpigmentation or erythema from resolving lesion
	0 absent 1 1–3 lesions, up to one >2 cm in any diameter, none > 6 cm 2 2–3 lesions, at least two > 2 cm diameter, none > 6cm 3 >3 lesions, none > 6 cm diameter 5 >3 lesions, and/or at least one >6 cm 10 >3 lesions, and/or at least one lesion >16 cm diameter or entire area	Number lesions if ≤ 3	0 absent 1 present
Ears			
Nose			
Rest of the face			
Neck			
Chest			
Abdomen			
Back, buttocks			
Arms			
Hands			
Legs			
Feet			
Genitals			
Total skin	/120		/12

Scalp

Scalp	Erosion/Blisters or new erythema	Number lesions if ≤ 3	Post-inflammatory hyperpigmentation or erythema from resolving lesion
	0 absent 1 in one quadrant 2 two quadrants 3 three quadrants 4 affects whole skull 10 at least one lesion > 6 cm		0 absent 1 present
Total Scalp (0–10)	/10		/1

Mucous membrane

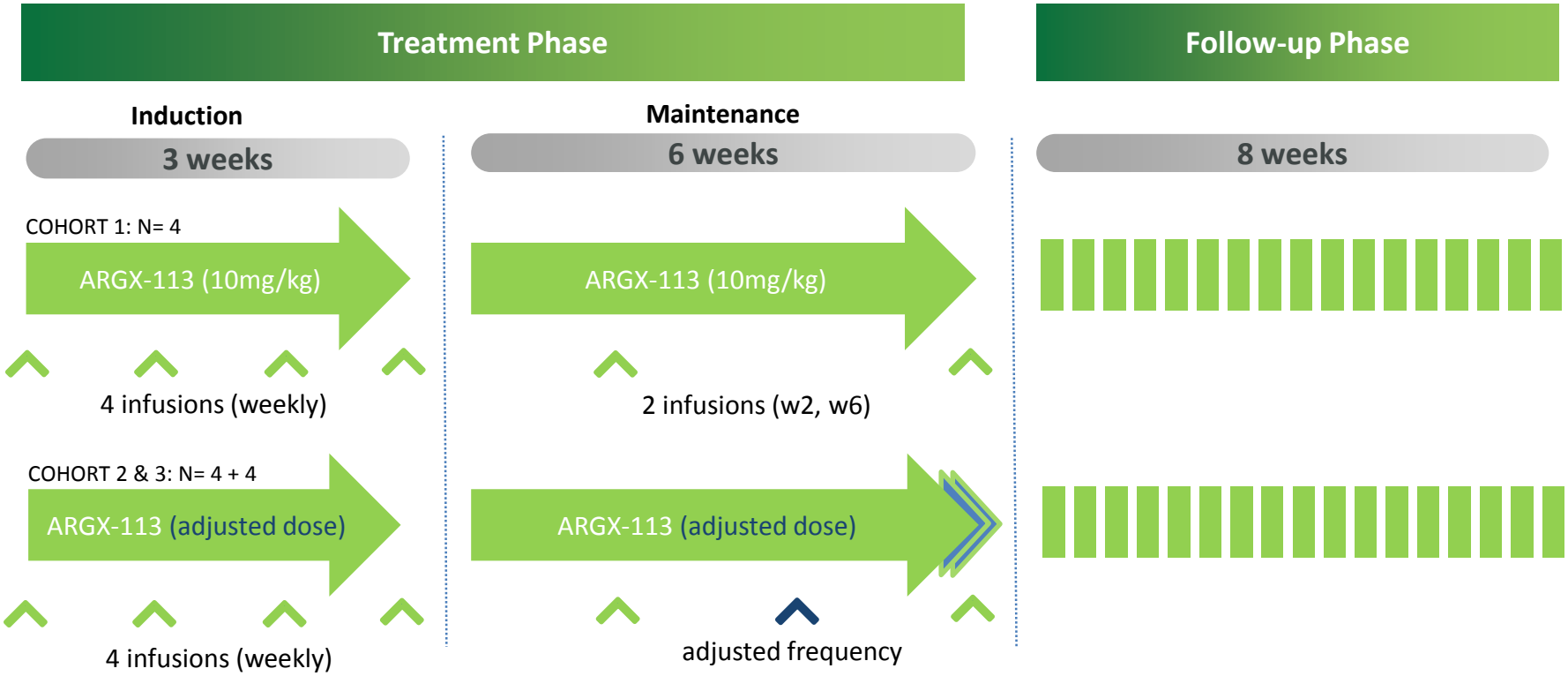
Anatomical Location	Erosion/Blisters	
	0 absent 1 1 lesion 2 2–3 lesions 5 >3 lesions or 2 lesions >2 cm 10 entire area	Number lesions if ≤ 3
Eyes		
Nose		
Buccal mucosa		
Hard palate		
Soft palate		
Upper gingiva		
Lower gingiva		
Tongue		
Floor of mouth		
Labial bucosa		
Posterior pharynx		
Anogenital		
Total Mucosa	/120	

Severity	PDAI[2][3]
Mild	1-14
Moderate	15-44
Severe	≥ 45

- PDAI: international validated score (preferable as severity measure)
- High score denotes worse disease
- Score range: from 0 to 263 (250 points measuring disease activity & 13 points measuring post-inflammatory lesions)
- Disease control: absence of new lesion, and established lesions beginning to heal
- Relapse: appearance of 3 or more lesions within the month not healing spontaneously within one week, or extension of established lesions

(1) Rosenbach et al. 2009, J invest. Dermatol (2) Hanna et al. 2016, Int J Womens Dermatol (3) Boulard et al. 2016, Br J Dermatol (4) Murell et al. 2008, J Am Acad Dermatol

ARGX-113 Phase 2 study : IDMC-driven adaptive design



- Patients enrollment divided in 3 sequential cohorts
- IDMC recommendations for cohorts 2 & 3:
  - Change of dose (max dose of 25mg/kg)
  - Frequency of administration at maintenance (max 2 extra doses after each cohort)
  - Expansion of maintenance duration

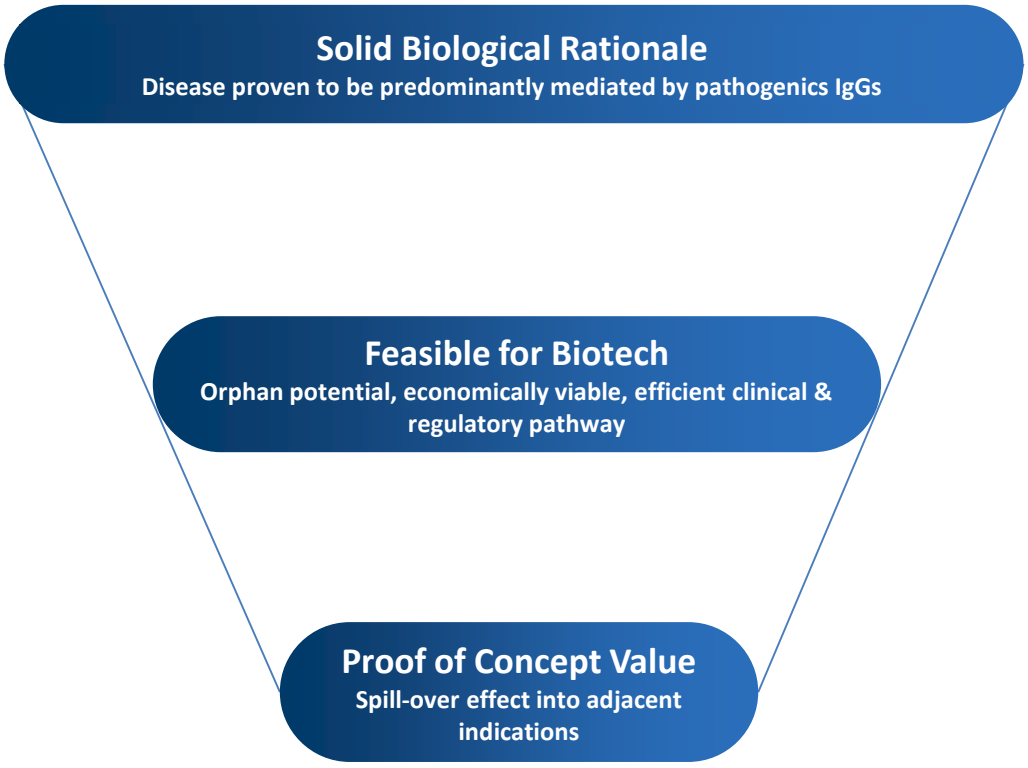
ARGX-113: A pipeline-in-a-product opportunity

ARGX-113: A pipeline-in-product opportunity

Prioritizing IgG autoantibody mediated diseases

Landscape of IgG severe auto-immune diseases (selection)

Immune Thrombocytopenia
Scleroderma
Lupus
Epidermolysis Bullosa Acquisita
Multiple Sclerosis
Anca Vasculitis
Myasthenia Gravis
Rheumatoid Arthritis
Pemphigus
Bullous Pemphigoid

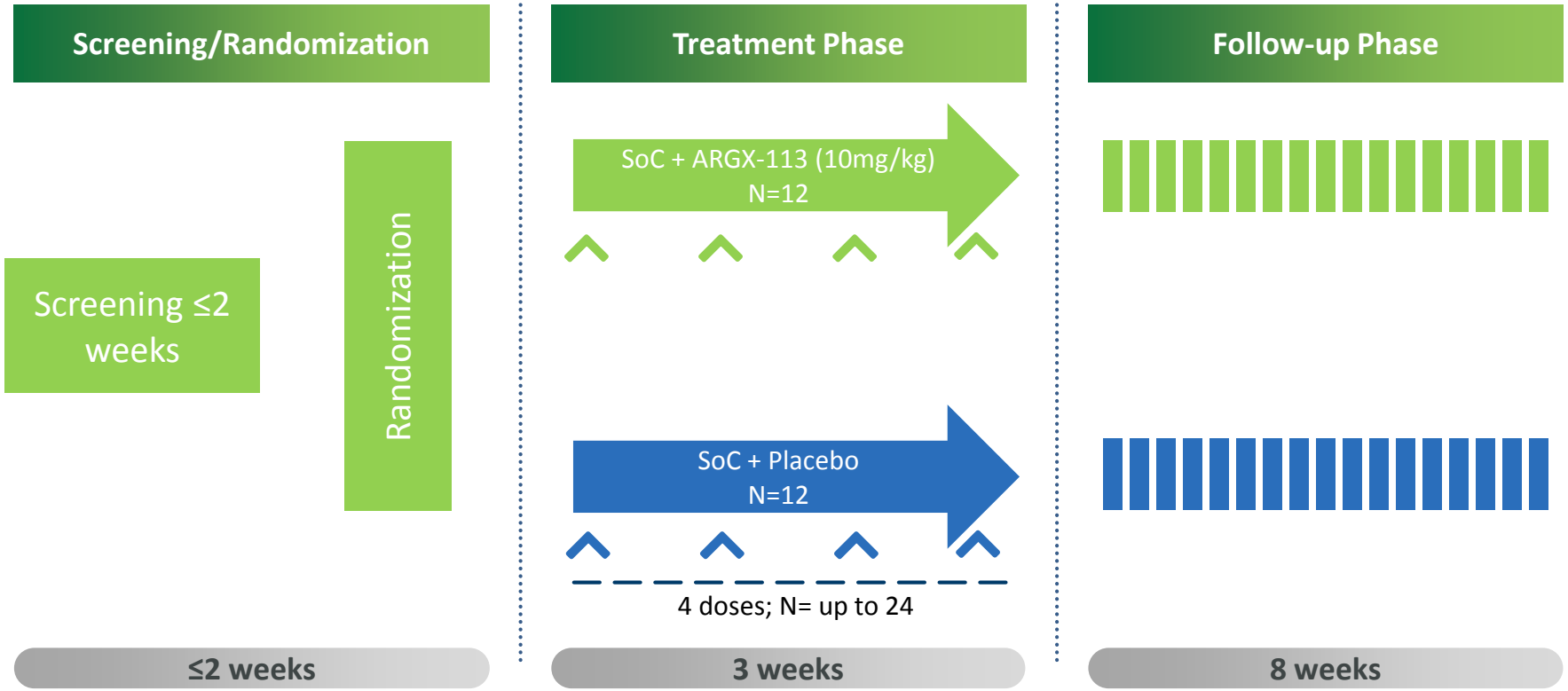


Myasthenia Gravis	Immune Thrombocytopenia	Pemphigus Vulgaris
Beachhead neuromuscular diseases	Beachhead Heme disorders	Beachhead blistering diseases

argenx data

ARGX-113 in MG: Phase 2 trial design

Study 100% recruited Oct 2017

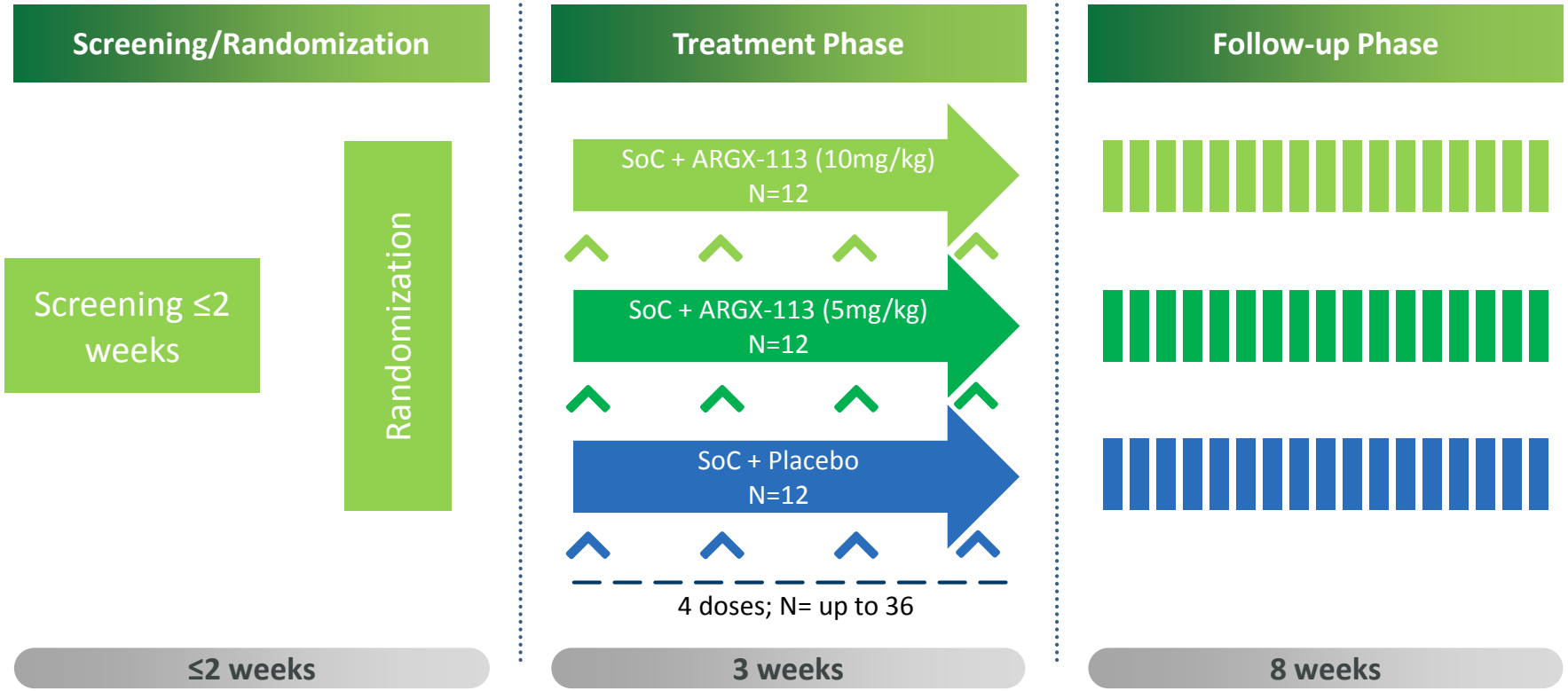


- Population: MG patients with generalized muscle weakness with total MG-ADL score ≥ 5*
- Primary Objectives: Evaluate safety and tolerability
- Secondary Objectives: (i) Evaluate efficacy, impact on quality of life and immunogenicity
  (ii) Assess pharmacokinetics (PK) and pharmacodynamics (PD) markers

argenx data, *> 50% of this score attributed to non-ocular items.

ARGX-113 in ITP: Phase 2 trial design

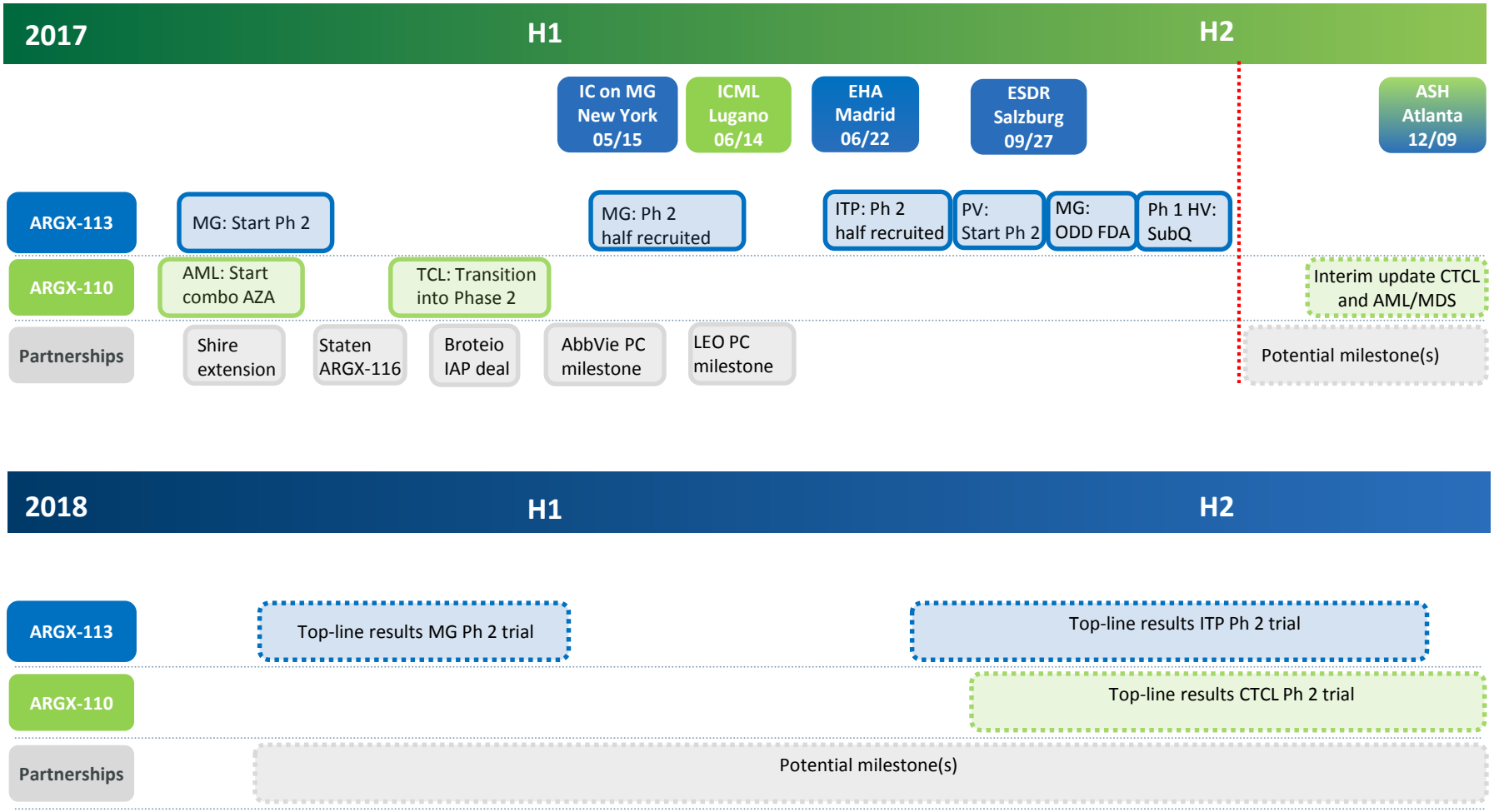
Study
50% recruited
Sept 2017



- Population: ITP patients with platelet levels < 30 X 10^9/L
- Primary Objectives: Evaluate safety and tolerability
- Secondary Objectives: (i) Evaluation of efficacy based on platelet counts, use of rescue treatment & bleeding events
  (ii) Assess pharmacokinetics (PK) and pharmacodynamics (PD) effect
  (iii) Evaluate immunogenicity

argenx data

Key Upcoming Milestones & Communications



argenx

Please Join argenx for a Lunch and Discussion during the ASH Annual Meeting

Advancing ARGX-110 to clinical proof-of-concept in acute myeloid leukemia (AML) & cutaneous t-cell lymphoma (CTCL)

Monday, December 11, 2017

12:00 PM – 1:30 PM

Omni Atlanta Hotel

South Tower Atrium Terrace
Birch Room
100 CNN Center NW
Atlanta, GA 30303

Next to Convention Center

Please RSVP by Monday, December 4

Rachel Frank
rachelf@sternir.com
212.362.1200

Agenda

Overview of AML
Gail Roboz, MD
Weil Cornell Medicine, New York

CD70: Novel AML Target
Hans de Haard, PhD, argenx

Phase 1/2 AML Trial: Proof-of-Biology Data
Nicolas Leupin, MD, argenx

Phase 1/2 CTCL Trial: Data Update
Nicolas Leupin, MD, argenx

Q&A

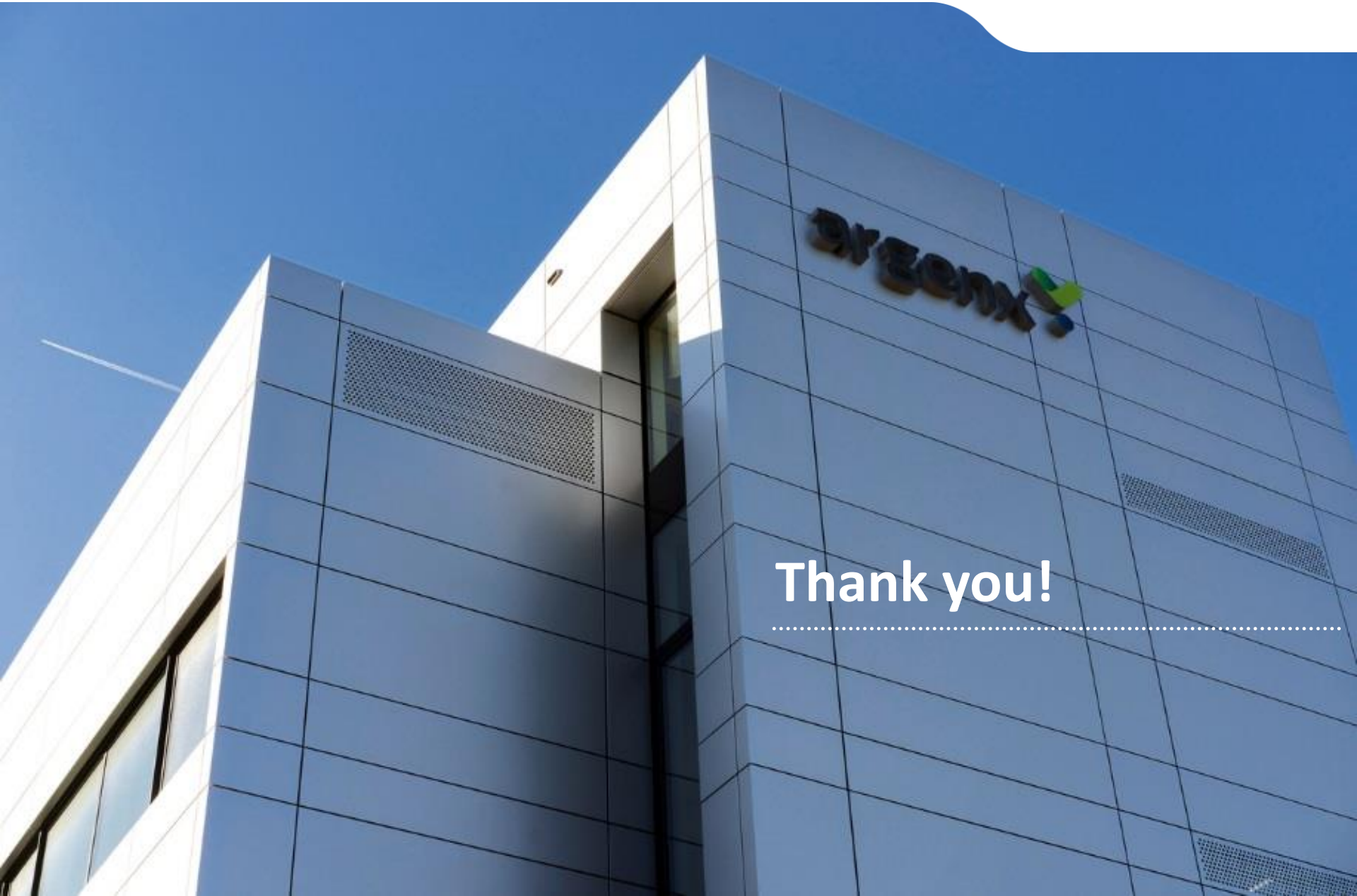
Guest Speaker

Gail Roboz, MD

Professor of Medicine and Director of Clinical and Translational Leukemia Program

Weill Medical College of Cornell University and New York Presbyterian Hospital

Following the presentation, please join us and our colleagues from the University of Bern for an informal mix and mingle.



Thank you!