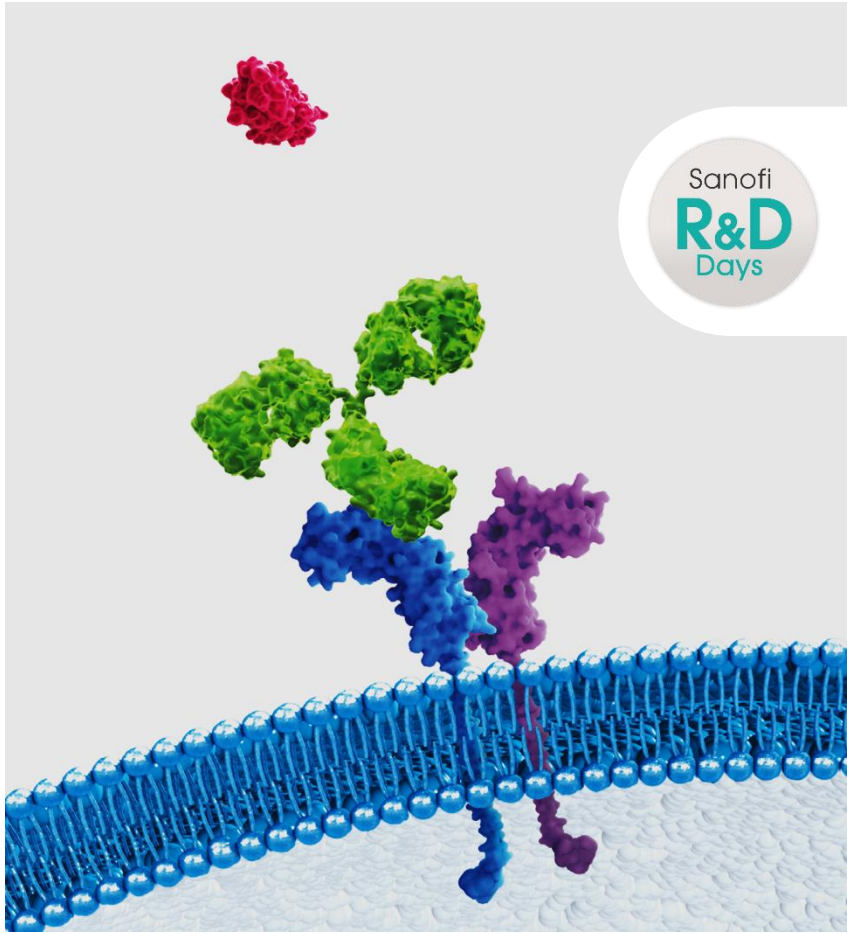

Dupixent® IR event

It takes two for Type 2 inflammation

June 11, 2020

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Forward looking statements

This presentation contains forward-looking statements as defined in the Private Securities Litigation Reform Act of 1995, as amended. Forward-looking statements are statements that are not historical facts. These statements include projections and estimates and their underlying assumptions, statements regarding plans, objectives, intentions and expectations with respect to future financial results, events, operations, services, product development and potential, and statements regarding future performance. Forward-looking statements are generally identified by the words “expects”, “anticipates”, “believes”, “intends”, “estimates”, “plans” and similar expressions. Although Sanofi’s management believes that the expectations reflected in such forward-looking statements are reasonable, investors are cautioned that forward-looking information and statements are subject to various risks and uncertainties, many of which are difficult to predict and generally beyond the control of Sanofi, that could cause actual results and developments to differ materially from those expressed in, or implied or projected by, the forward-looking information and statements. These risks and uncertainties include among other things, the uncertainties inherent in research and development, future clinical data and analysis, including post marketing, decisions by regulatory authorities, such as the FDA or the EMA, regarding whether and when to approve any drug, device or biological application that may be filed for any such product candidates as well as their decisions regarding labelling and other matters that could affect the availability or commercial potential of such product candidates, the fact that product candidates if approved may not be commercially successful, the future approval and commercial success of therapeutic alternatives, Sanofi’s ability to benefit from external growth opportunities, to complete related transactions and/or obtain regulatory clearances, risks associated with intellectual property and any related pending or future litigation and the ultimate outcome of such litigation, trends in exchange rates and prevailing interest rates, volatile economic and market conditions, cost containment initiatives and subsequent changes thereto, and the impact that COVID-19 will have on us, our customers, suppliers, vendors, and other business partners, and the financial condition of any one of them, as well as on our employees and on the global economy as a whole. Any material effect of COVID-19 on any of the foregoing could also adversely impact us. This situation is changing rapidly and additional impacts may arise of which we are not currently aware and may exacerbate other previously identified risks. The risks and uncertainties also include the uncertainties discussed or identified in the public filings with the SEC and the AMF made by Sanofi, including those listed under “Risk Factors” and “Cautionary Statement Regarding Forward-Looking Statements” in Sanofi’s annual report on Form 20-F for the year ended December 31, 2019. Other than as required by applicable law, Sanofi does not undertake any obligation to update or revise any forward-looking information or statements.

Agenda

Success in AD and Asthma	**Brian Foard**	Global Head of Dupixent® Franchise	
Deep-dive on Type 2 inflammation	**Frank Nestle**	Global Head of Research, Immunology & Inflammation	
Line extensions in Type 2 diseases	**Naimish Patel**	Global Head of Development, Immunology & Inflammation	
Conclusion	**John Reed**	Global Head of Research and Development	
Q&A (also joining)	**Paul Hudson**	Chief Executive Officer	
	Jean-Baptiste de Chatillon	Chief Financial Officer	
	Bill Sibold	Global Head of Specialty Care	

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AD: Atopic dermatitis
Dupixent® is developed and commercialized in collaboration with Regeneron

Success in Atopic dermatitis and Asthma

Brian Foard

Global Head of Dupixent® Franchise

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Dupixent®: First biologic approved in AD pediatrics in US

Dupixent® label in AD pediatrics[1]

Dupixent® is indicated for the treatment of patients aged 6 years and older

- With moderate-to-severe atopic dermatitis
- Whose disease is not adequately controlled with topical prescription therapies or when those therapies are not advisable

Dupixent® can be used with or without topical corticosteroids

U.S. AD population by age group[2]
('000, approximate)

	Adults	12-17Y	6-11Y	<6Y[4]
Prevalence	8,200	2,500	2,500	2,400
Moderate-to-severe	2,600	800	700	700
Biologics eligible	1,700	400	90	75
DUPIXENT® (dupilumab)	**74[3]**	**8[3]**	**Approved**	**2022e**
Share of Biologics eligible	*4.4%*	*2.0%*		

AD: Atopic dermatitis
(1) Dupixent® U.S. Prescribing Information (2) Truven Payer Claims Data; IQVIA Sanofi Custom SOB Report; Data on file (3) Reflects the number of patients currently on treatment (4) Estimated regulatory submission timing and data has not been reviewed by any regulatory authority
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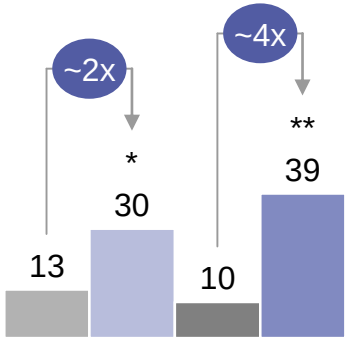
Safety and efficacy consistent with what has been seen in adults and adolescents

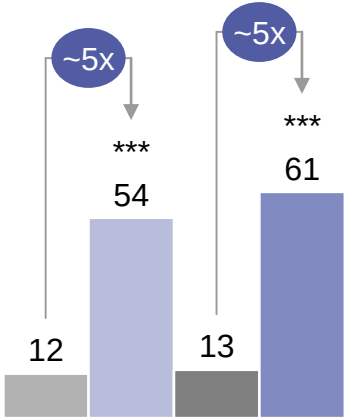
IGA score of 0 or 1

% of patients (6-11 years with severe AD)



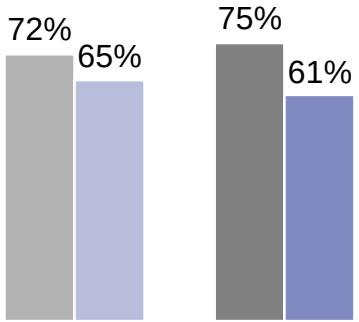
≥ 4-point improvement in peak pruritus NRS

% of patients (6-11 years with severe AD)



Overall rates of adverse events(1)

% of patients (6-11 years with severe AD)



Placebo + TCS 15-30kg (n=61)

Dupixent® 300mg q4w + TCS 15-30kg (n=61)

Placebo + TCS 30-60kg (n=62)

Dupixent® 200mg q2w + TCS 30-60kg (n=59)

~5x as many children achieved itch reduction with Dupixent® + TCS vs. TCS alone

AD: Atopic dermatitis; TCS: Topical corticosteroids; IGA: Investigator global assessment; NRS: Numeric rating scale
*p<0.05; **p<0.01; ***p<0.001
(1) Adverse events that were more common in the Dupixent® arm included injection site reactions and conjunctivitis. Detailed adverse events rates are in the appendix.
Source : 2020-05-26 Sanofi press release
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Safety data support further expansion into pediatrics

Robust safety dataset[1]

Clinical trials

- **10,000+** patients
  Studied across 50+ clinical programs
- **148-week** long-term safety data
  In adults (>18 years)
- **52-week** long-term safety data
  In adolescents (12-17 years)
- **52-week** safety data
  In pediatric population (6-11 years)

Clinical practice

- **150,000+** patients
  Treated globally since launch

Expanding to pediatrics

	Target submission
AD 6-11 years *Breakthrough designation*	✓ [2]
AD <6 years *Breakthrough designation*	2022e
Asthma 6-11 years	2021e
Asthma <6 years	Planned
EoE <12 years	Planned

No black box warning and no evidence of immunosuppression[3]

AD: Atopic dermatitis; EoE: Eosinophilic esophagitis
(1) Majority of data to date in adults and adolescents (2) FDA approved (3) Meta-analyses show Dupixent® associated with decreased infections; in approved indications
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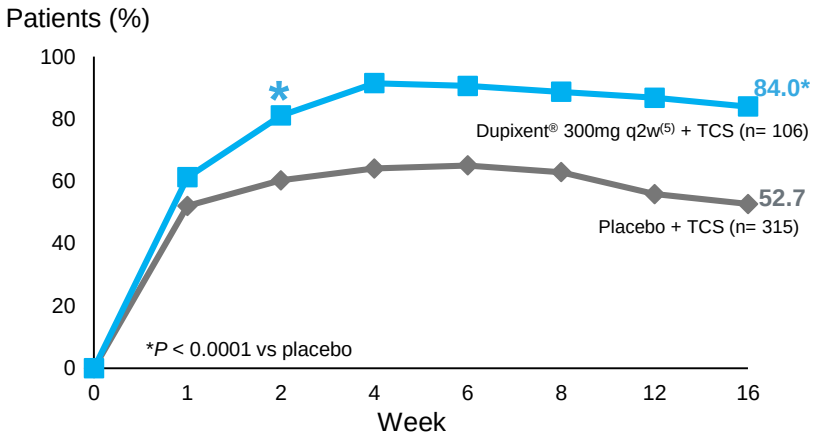
Atopic dermatitis: Rapid and sustained efficacy

Rapid efficacy across multiple measures

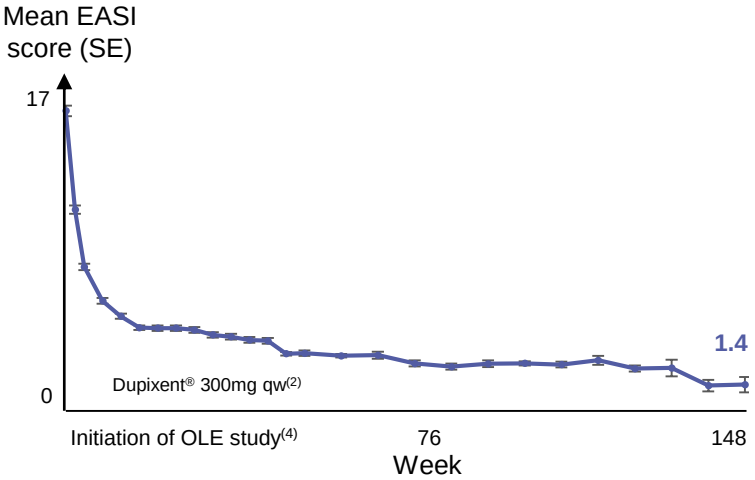
Proportion of patients achieving EASI-50 or peak pruritus NRS ≥ 3-point improvement or DLQI ≥ 4-point improvement with Dupixent® in CHRONOS studies[2,3]



- >80% of patients saw improvement in 1 or more disease measures (lesions, itch, QoL) after first dose

Sustained efficacy and safety[1]

Long-term extension study



- Efficacy maintained over 3 years
- 3 year safety profile shows no change from previous studies

EASI: Eczema area severity index; EASI-50: at least 50% improvement of EASI score; SE: Standard error, OLE: Open label extension; NRS: Numerical rating scale; DLQI: Dermatology quality of life index; TCS: Topical corticosteroids; QoL: Quality of life; q2w: once every other week; qw: once weekly

(1) LIBERTY AD OLE, Thaci et al; Favorable Safety and Sustained Efficacy with Long Term Dupilumab Treatment in Adults with Moderate-to-Severe Atopic Dermatitis; an analysis up to 3 years. Poster presented at Maui Dermatology Conference January 25-29, 2020; analysis based on the secondary endpoint (2) De Bruin-Weller et all, Oral presentation EADV congress 2018 (3) Based on post hoc analysis (4) Mean EASI score of baseline study was 33.37 (5) Approved recommended dose level for Dupixent® in adult patients with AD after initial dose is 300mg once every other week (q2w) .Dupixent® is developed and commercialized in collaboration with Regeneron

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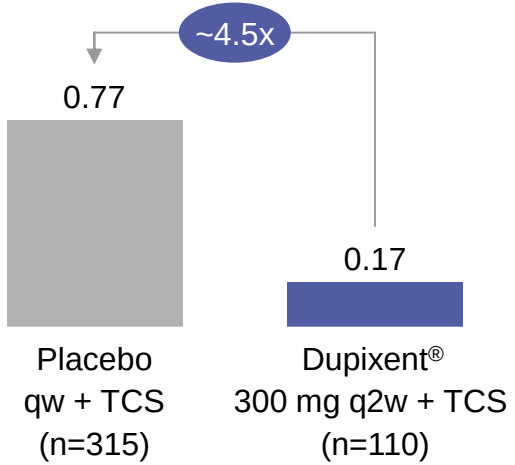
Atopic dermatitis: Dupixent® greatly reduced flares in adults with moderate-to-severe AD

Flares are defined as worsening of the disease requiring escalation / intensification of AD treatment

Flare prevention is a primary goal of long-term AD disease control

- High costs & burden (emergency room often required)
- No specific treatment approved (systemic steroids used)

Dupixent® impact on annualized flare rate of adults with moderate-to-severe AD(1)



Relative risk reduction = 0.22, 95% CI: 0.13, 0.39
Nominal P-value < 0.0001(2)

84% of patients who have experienced >1 flare before treatment and received Dupixent® were **flare-free** during the treatment period

Patients receiving TCS alone experienced ~4.5x more flares vs. patients treated with Dupixent® + TCS

AD: Atopic dermatitis; TCS: Topical corticosteroids; qw: once weekly; q2w: once every other week
(1) LIBERTY AD CHRONOS (AD-1225) : randomized, double-blind, placebo controlled, phase 3 trial; all patients received concomitant medium potency topical corticosteroids
Source: Dupilumab prevents flares in adults with moderate-to-severe atopic dermatitis in a 52-week randomized controlled phase 3 trial, Joseph F. Merola et al.
(2) Flares at 52 weeks is a pre-specified, secondary endpoint
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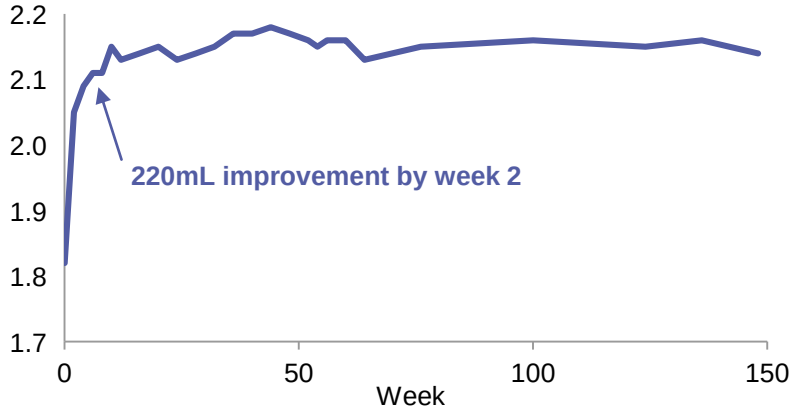
Asthma: New data show rapid and sustained efficacy

Rapid & sustained improvement in FEV1

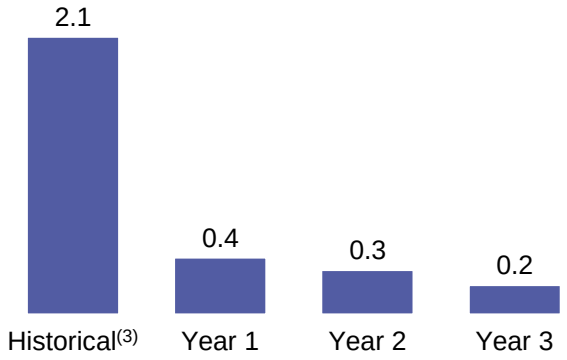
Average FEV1 (L)[1]
(n=182)



29% Type 2 patients achieve a FEV1 in normal range

Progressively declining exacerbation rate

Unadjusted annualized exacerbation rate across 3 years[1]
(n=182)



>60% Type 2 patients had no exacerbations for 3 years

2,000+ patients completed OLE treatment period with a safety profile consistent with shorter-duration parent studies

OLE: Open label extension; FEV1: Forced exhalation volume in one second
(1) Population treated with dupilumab 300 mg Q2W across QUEST & TRAVERSE (with 96w data on TRAVERSE) (2) Type 2 patients refer to the patients with Type 2 inflammatory phenotype (blood eosinophil >= 0.15 Giga/L or FeNo >= 25 ppb) at the baseline of parent study (3) Historical refers to the mean event count of severe exacerbations in the 1 year prior to QUEST
Note: data to be presented in September
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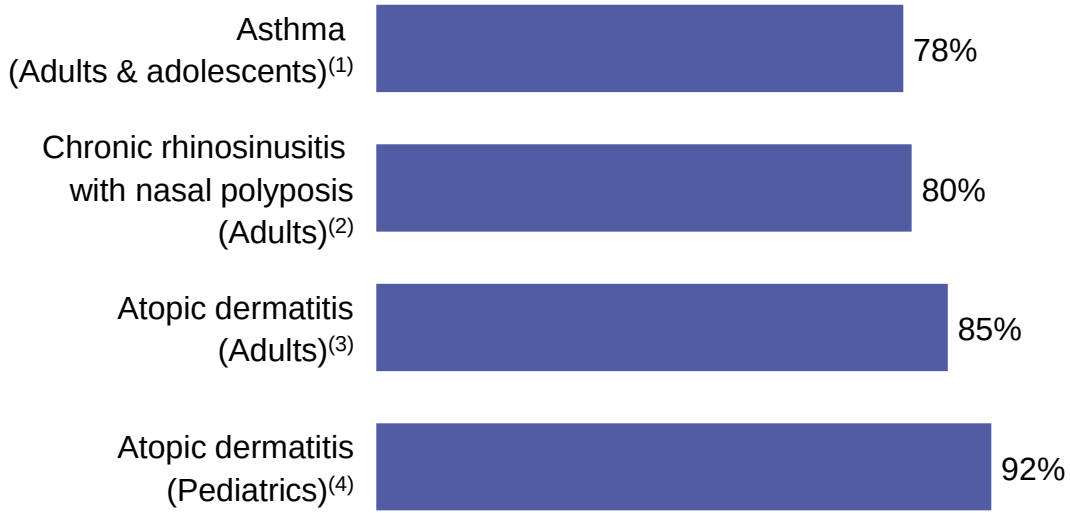
~80-90% of patients suffer from multiple Type 2 diseases

% of patients with concurrent Type 2 disease across studies (n=5,378)[5]



Dupixent® aims to maximize patient benefit by addressing all concurrent Type 2 diseases

(1) DRI and QUEST studies, history + current (2) SINUS-24 and SINUS 52 studies, history + current (3) SOLO and 1021 studies, history + current
(4) AD studies for 6-11 yo, >=1 concurrent ; Dupixent® has not been approved for pediatric AD (6-11) patients outside the U.S. (5) List of Type 2 diseases includes atopic dermatitis, asthma, chronic rhinosinusitis with nasal polyposis, eosinophilic esophagitis, food allergy, allergic rhinitis, allergic conjunctivitis, hives, and other allergies
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Deep-dive on Type 2 inflammation

Frank Nestle

Global Head of Research, Immunology & Inflammation

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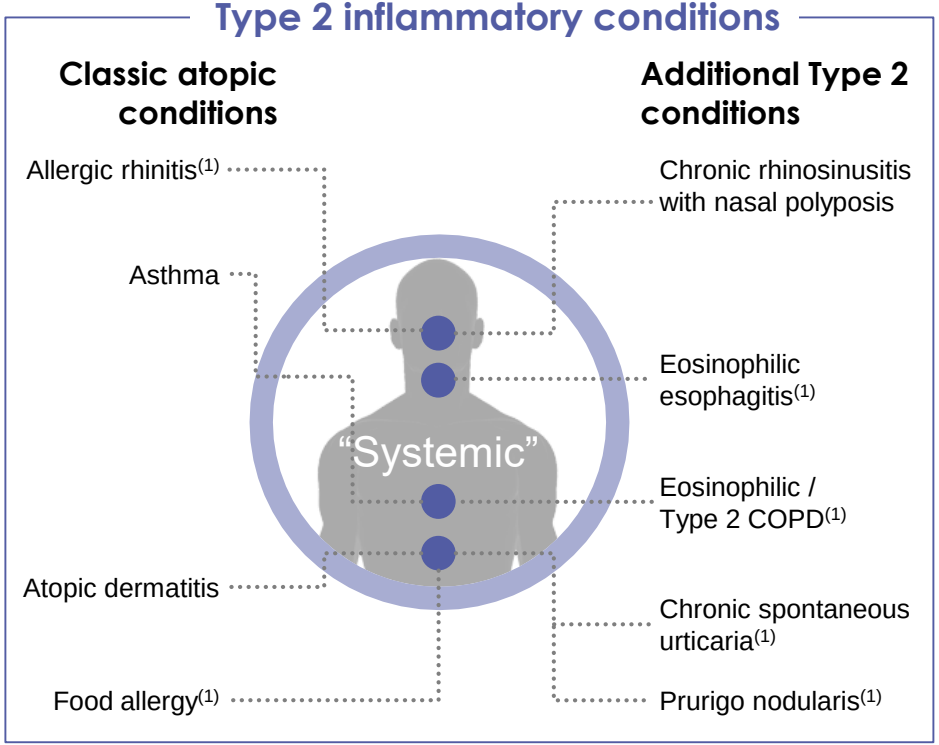
Type 2 inflammation: Connected pathology of systemic, chronic diseases

- Type 2 immunity evolved as **protection against venoms, parasites and dangerous environmental exposures**
- **When maladapted** and reactive to harmless agents, Type 2 immunity drives **atopic diseases** and **other pathologies with Type 2 hallmarks**
- **Type 2 hallmarks** include increased Type 2 cytokines and chemokines (e.g. TARC), Eosinophils, Basophils, Mast cells, ILC2s, IgE and barrier disruption
- Type 2 inflammation **occurs systemically** but can present **many different local/tissue manifestations**
  - Mucus production
  - Airway hypersensitivity
  - Itch
  - Dysphagia



COPD: Chronic obstructive pulmonary disease; TARC: Thymus and activation-regulated chemokine; ILC2s: Type 2 innate lymphoid cells
(1) Use of Dupixent® in these indications has not been fully evaluated by any regulatory authority
Source: Gandhi NA, et al. Nat Rev Drug Discov. 2016;15(1):35-50; Carr S, et al. Allergy Asthma Clin Immunol. 2011;7(suppl 1):S8; Steinke JW, Wilson JM. J Asthma Allergy. 2016;9:37-43
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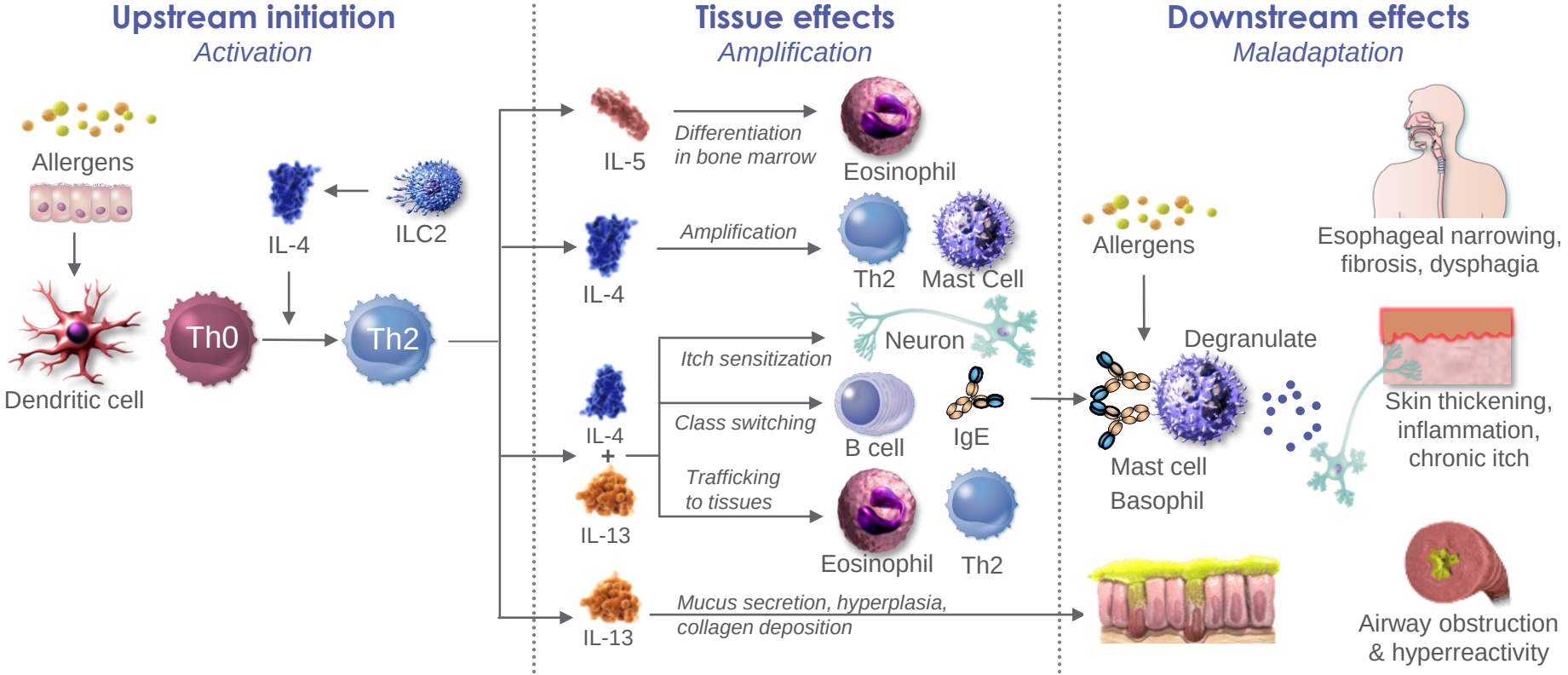
IL-4 regulates multiple phases of Type 2 effector biology



Th cells: T helper cells; ILC: Innate lymphoid cells ; IgE: Immunoglobulin E
Source: https://www.type2inflammation.com/science-cytokines
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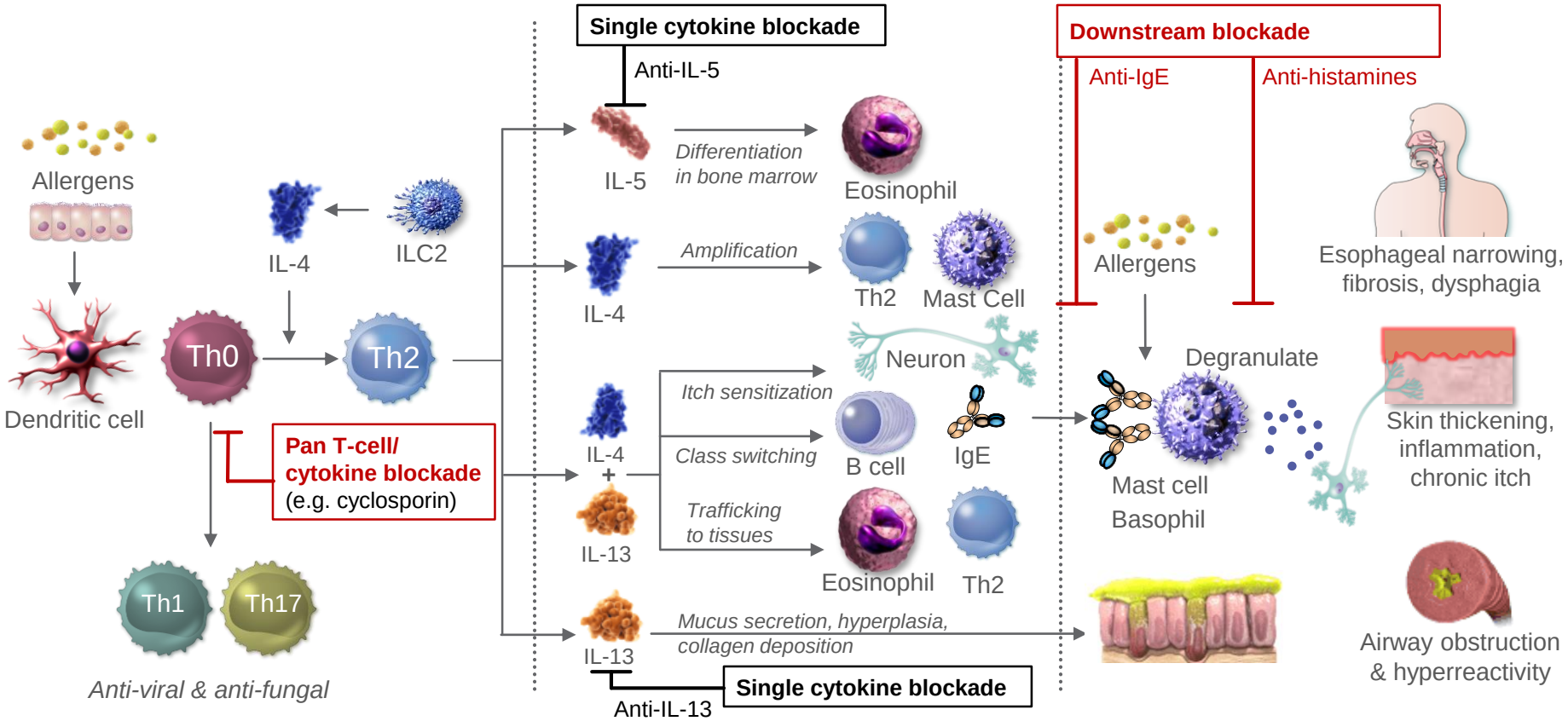
Pharmacological modulation of Type 2 immunity



Th cells : T helper cells; ILC: Innate lymphoid cells; IgE: Immunoglobulin E
Source: https://www.type2inflammation.com/science-cytokines
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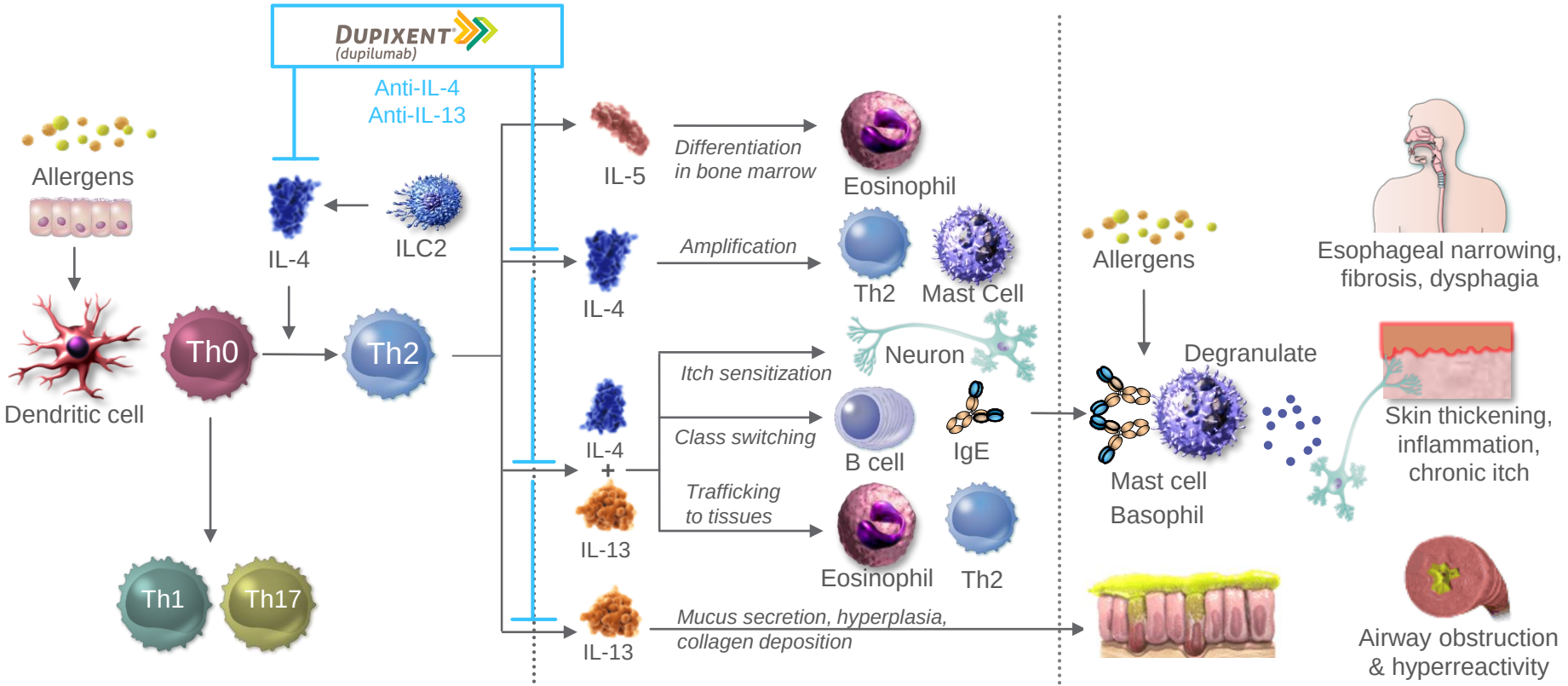
Dupixent® selectively inhibits Type 2 effector biology through blockade of IL-4 and IL-13



Th cells : T helper cells; ILC: Innate lymphoid cells; IgE: Immunoglobulin E
Source: https://www.type2inflammation.com/science-cytokines
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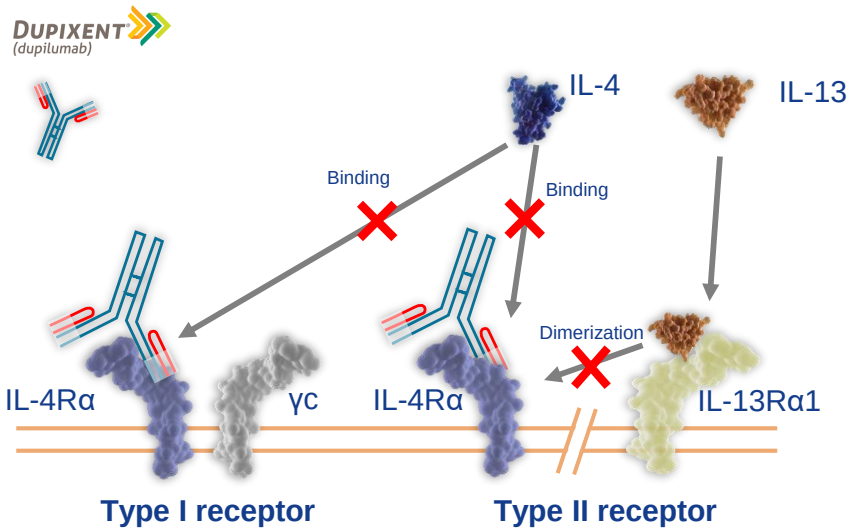
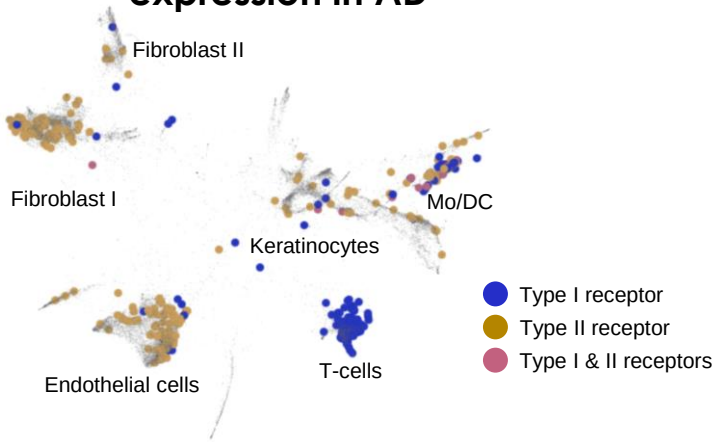
Distinct patterns of IL-4 & IL-13 receptors across cell types

IL-4/IL-13 receptor types, cytokine binding and mechanism of Dupixent® (1)



Single-cell view of IL-4/IL-13 receptor expression in AD(2)



	T-cells	Non-Immune cells	Mo/DC
Most abundant receptor(s)	Type I	Type II	Type I & II
Ligand(s)	IL-4	IL-4 & IL-13	IL-4 & IL-13

Targeting both receptors is important to inhibit Type 2 signaling

γc: common gamma chain; R: Receptor; AD: Atopic dermatitis; Mo/DC: Monocytes/Dendritic cells

(1) Hamilton et al EAACI Digital Congress 2020 (2) Internal analysis of data from https://doi.org/10.1016/j.jaci.2020.01.042

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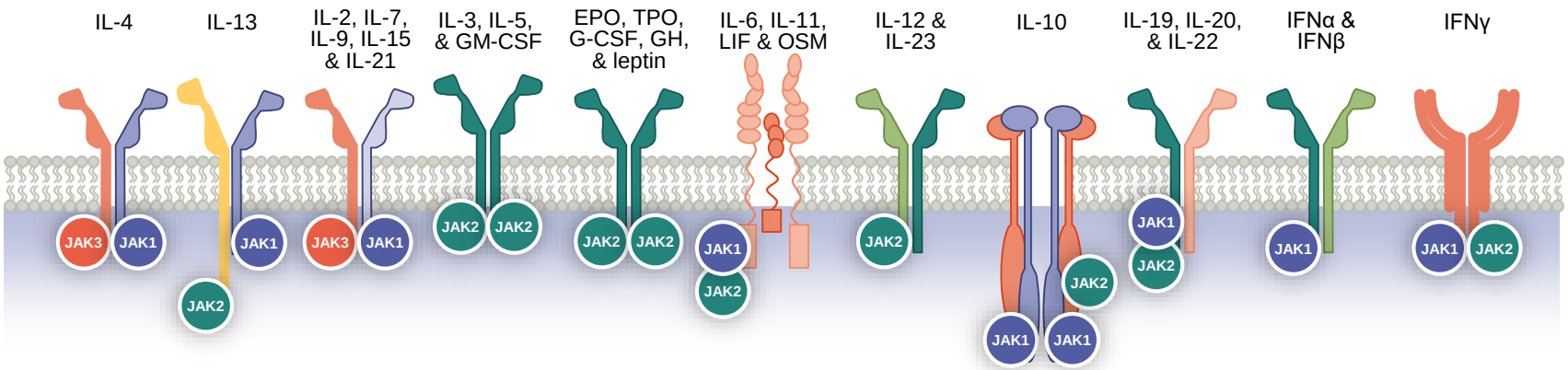
Dupixent®: Mechanism of action is highly specific



	IL-4	IL-13	IL-2, IL-7, IL-9, IL-15 & IL-21	IL-3, IL-5, & GM-CSF	EPO, TPO, G-CSF, GH, & leptin	IL-6, IL-11, LIF & OSM	IL-12 & IL-23	IL-10	IL-19, IL-20, & IL-22	IFNα & IFNβ	IFNγ
DUPIXENT (dupilumab)	×	×	-	-	-	-	-	-	-	-	-
JAK1	×	×	×	-	-	×	-	×	×	×	×
JAK2	-	×	-	×	×	×	×	×	×	-	×
JAK3	×	-	×	-	-	-	-	-	-	-	-

× Cytokine blockade

Source: Schwartz DM. et al. Nat Rev Drug Discov. 2017; 16(12):843-862

This slide is solely intended to describe the targeting approaches of various therapeutic options in this space. The clinical significance of these datapoints has not been definitively established.

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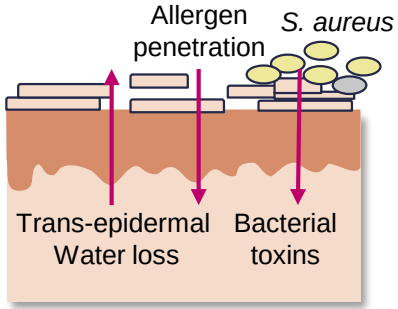
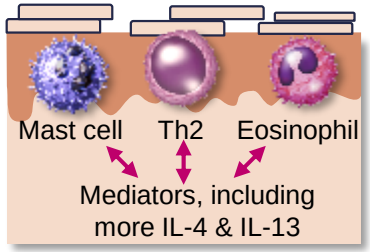
Dupixent® helps restore barrier function and microbiome

Atopic dermatitis skin



Barrier loss and overgrowth of disease-associated bacteria

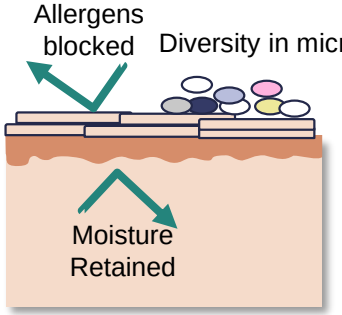


Inflammatory pathogenesis

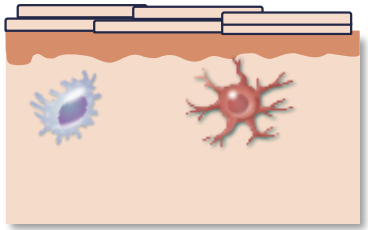
DUPIXENT® (dupilumab)

DUPIXENT® (dupilumab)

Healthy skin function



Healthy tissue homeostasis and commensal microbiome



Protective immunosurveillance

Th: T helper; S. aureus: Staphylococcus aureus

Sources: Callewaert C et al. J Invest Dermatol, 2020 140(1), p191-202; Guttman-Yassky E et al. Journal of Allergy and Clinical Immunology, Volume 143, Issue 1, (2019), Pages 155-172

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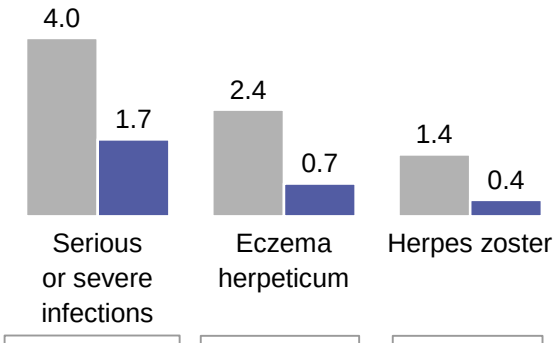
Fewer infections observed with Dupixent® vs placebo

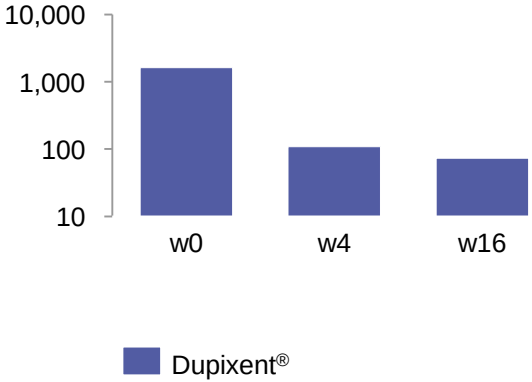
Atopic dermatitis[1]

Patients with ≥ 1 event (nP/100Py)



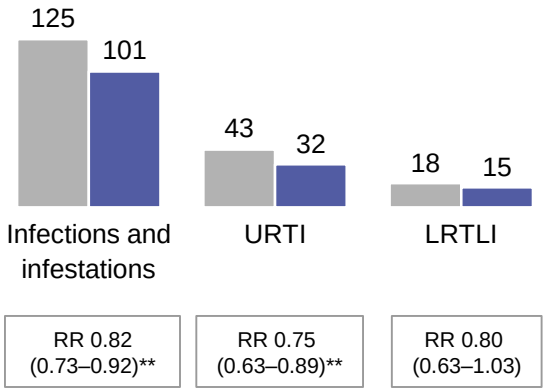
S. aureus infection[2]

Absolute abundance of S. aureus over time in lesional skin (qPCR) (rCFU/area) (median)



Asthma[3]

Annualized infection rates at patient level



nP: number of patients; Py: patient-years; URTI: upper respiratory tract infections; qPCR: quantitative polymerase chain reaction; rCFU: relative colony forming units; LRTLI: lower respiratory tract and lung infections; RR: risk ratio

*p<0.05; **p<0.01; ***p<0.001; values on graphs are risk ratio (RR) vs placebo (95% CI)

(1) Eichenfield L.F., American Journal of Clinical Dermatology 20, 443–456 (2019) (2) AD-1307 CSR Table 56 (3) Geng et al., EAACI Digital Congress 2020

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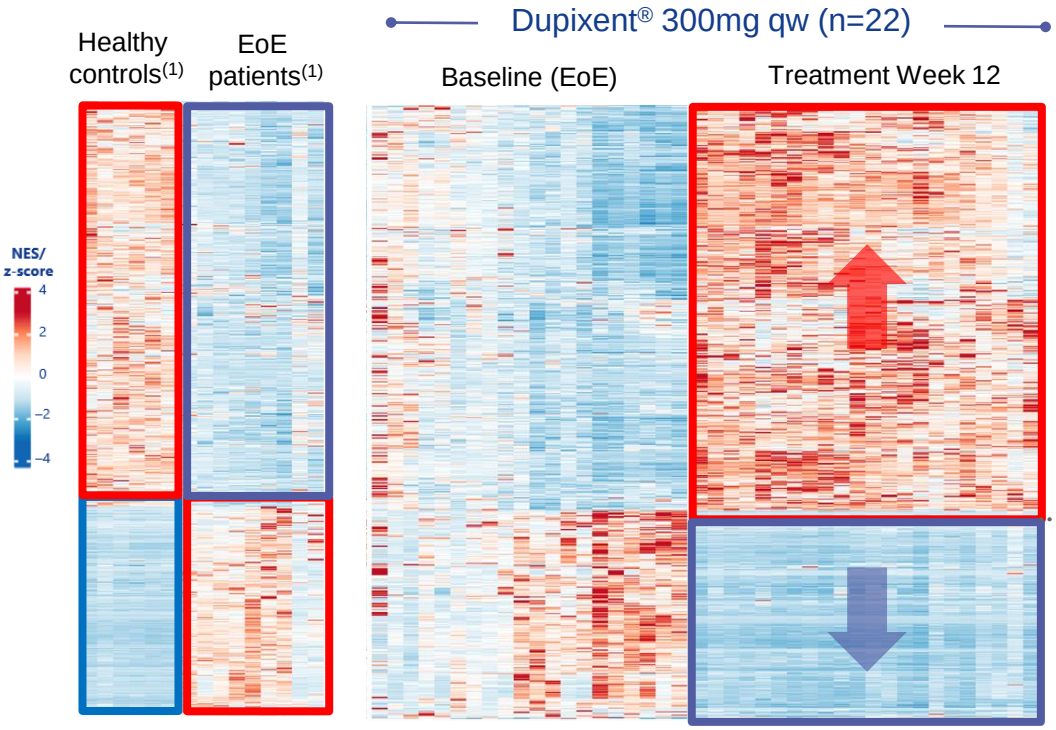
Dupixent® normalizes altered gene expression in EoE



Established EoE signature genes

Down in EoE
Up by Dupixent®

FLG
DSG1
SPINK7
CRISP3

- Improved barrier

Up in EoE
Down by Dupixent®

CCL26
CCR3
ALOX15
POSTN

- Reduced eosinophil recruitment
- Reduced Type 2 inflammation
- Reduced tissue remodeling

EoE: Eosinophilic esophagitis; NES: Normalized enrichment score; qw: once weekly
All genes included met significance thresholds: fold change ≥2, q<0.05
(1) Sherrill JD, et al. Genes Immun. 2014;15:361-9
Source: Hamilton et al EAACI Digital Congress 2020
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Dual pharmacology yields meaningful efficacy with a favorable safety profile

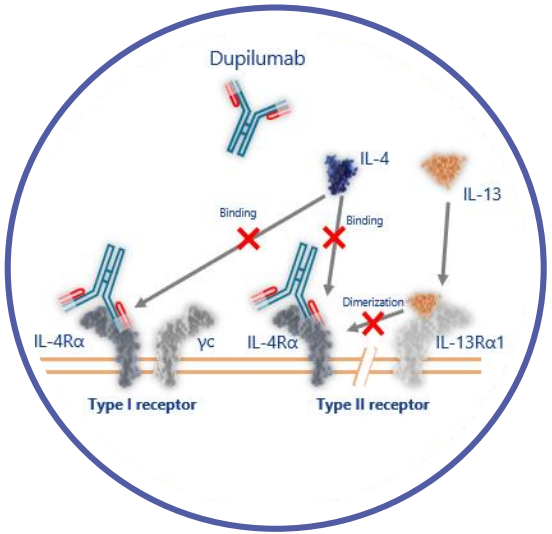
Efficacy

Systemic inhibition

- Upstream initiation
  IL-4 inhibition targets initiation and propagation of inflammation
- Tissue amplification
  - IL-13 inhibition prevents inflammatory and structural tissue changes
  - IL-4 inhibition prevents tissue Th2 and mast cell amplification

It takes two!

IL-4 & IL-13



Safety

Normalized immune system

- Restoring barrier function
- Rebalancing microbiome
- Limiting side-effects
- Reduced risk of bacterial and viral infections

Th: T helper; γc: common gamma chain; R: Receptor
Source: Gandhi NA, et al. Nat Rev Drug Discov. 2016; Le Floc'h A, et al. Allergy. 2019. doi: 10.1111/all.14151; Eichenfield L.F., American Journal of Clinical Dermatology 20, 443–456(2019)
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Line extensions in Type 2 diseases

Naimish Patel

Global Head of Development, Immunology & Inflammation

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Using precision medicine to prioritize Type 2 indications

Real-world evidence score + **ANDIE score** + **Case study score** + **Sanofi clinical consensus** + **KOL opinion**

Prioritized list of indications based on real-world evidence, vetted with KOL input

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Note: KOL: Key opinion leader; ANDIE: A network-based disease indication engine
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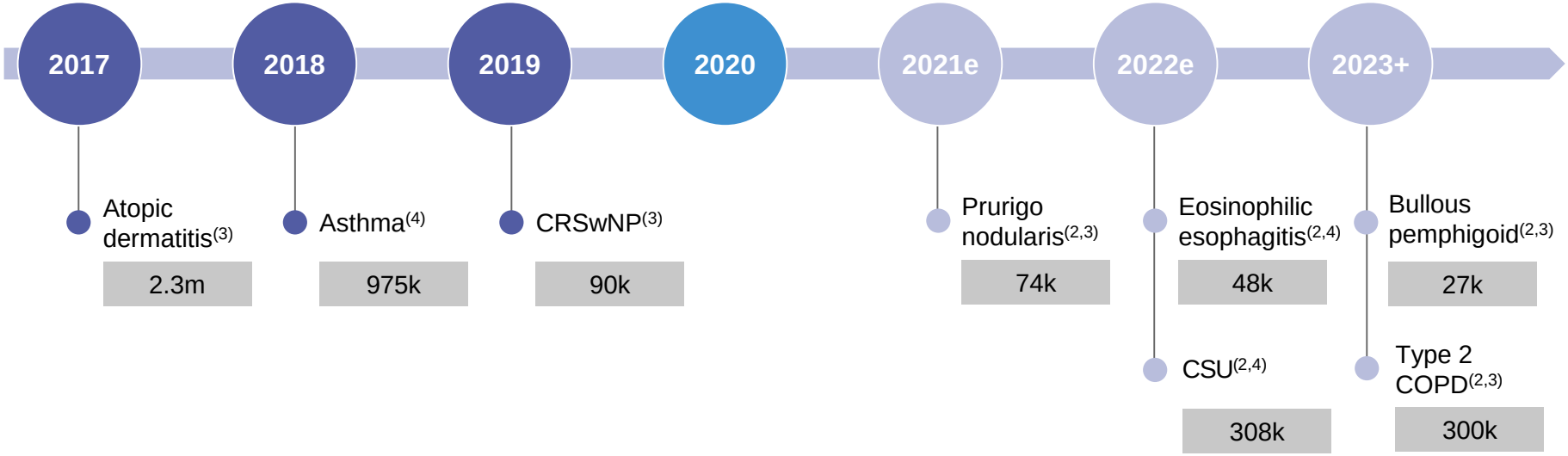
Prioritized Type 2 indications for Dupixent®



Source: Epidemiology data primarily from Sanofi real-world evidence platform
COPD: Chronic obstructive pulmonary disease; CSU: Chronic spontaneous urticaria; CRSwNP: Chronic rhinosinusitis with nasal polyposis
(1) Approved by FDA (2) Investigational program not yet reviewed by any Regulatory Authority (3) Initial launch in adults (4) Initial launch in 12 years and older
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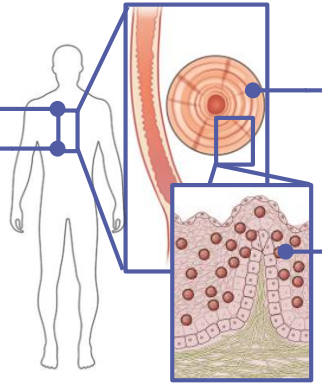
EoE: Type 2 inflammation with limited treatment options



Symptoms

- Dysphagia[1]
- Food impaction[2]
- Regurgitation
- Chest pain

Endoscopic signs

- Rings
- Furrows
- Strictures
- Edema
- Exudates

Histologic signs

- Eosinophilic infiltration[3]

Long-term disease complications

- Progressive symptoms
- Increasing esophageal constriction
- Potentially irreversible damage to esophageal tissue

- **Chronic inflammatory disease**
  - Triggered by an abnormal Type 2 inflammatory response to allergens
  - Characterized by eosinophilic inflammation of the esophagus and remodeling
- **Treatment options limited** to diet changes, proton-pump inhibitors, corticosteroids and surgery (dilation)
- **~159k treated patients in the U.S. (12+)**
  - ~48k biologics eligible[4]
  - Rising incidence

Type 2 hallmark: Type 2 gene signature and eosinophilic infiltration into the esophagus

PPI: Proton pump inhibitors; EoE: Eosinophilic esophagitis
(1) Defined as difficulty swallowing (2) Defined as esophageal obstruction by a foreign body (eg food) that may require medical intervention (3) ≥15 eosinophils/ high-power field (4) Severe: uncontrolled on high dose PPI and topical steroid slurry and elimination diet / trigger avoidance
Images adapted from Dellon ES, Hirano I. Gastroenterology. 2018;154:319–332. Lucendo AJ, et al. United European Gastroenterol J. 2017;5:335–358
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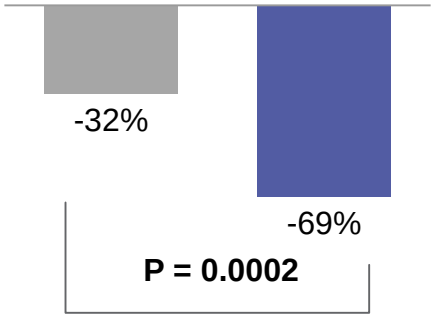
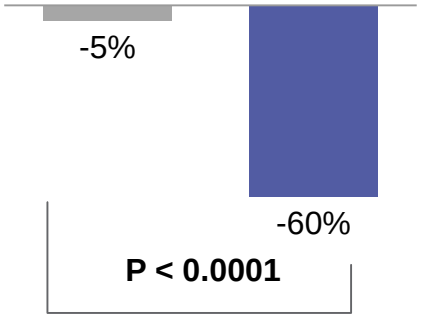
EoE: Highly significant effect on Primary and Secondary endpoints demonstrated in Phase 3 Part A

Co-primary endpoints

Disease symptoms reduction (%) measured by DSQ scale

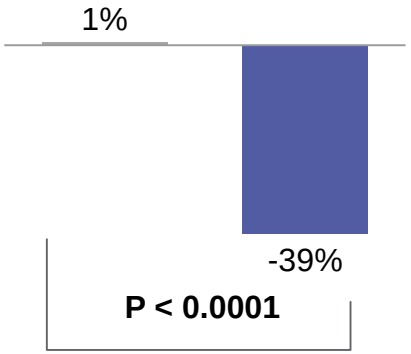


Esophageal eosinophil count reduction (%)



Secondary endpoint

Reduction in abnormal endoscopic finding (%) measured by EoE-EREFS



Placebo (n=39) Dupixent® 300mg qw (n=42)

EoE: Eosinophilic esophagitis; qw: once weekly; DSQ: Dysphagia Symptom Questionnaire; EOE-EREFS: EoE Endoscopic Reference Score
Dupixent® in EoE is investigational and has not been fully reviewed by regulatory authorities
Safety: Adverse event rate: 86% for Dupixent® (vs. 82% for placebo)
Sources: Sanofi press release of May 22, 2020
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COPD: Potential to treat Type 2 subpopulation

Large unmet need for new treatment options in COPD
- Many patients still experience severe exacerbations
- No approved biologics to date

Evidence of Type 2 inflammation in subset of patients
- Type 2 gene expression in epithelium
- Blood or sputum eosinophilia
- Increased exacerbation risk
- Lower FEV1, symptoms, increased airways disease
- More responsiveness to ICS

Rationale for Dupixent®
- Lung function significantly improved in patients with CRSwNP and COPD
- Effective depletion of eosinophils in tissues

Epidemiology

U.S. population
(patients in '000, approximate)

Prevalence	10,052
Treated	8,367
GOLD D	1,581
Type 2 population(1)	300

Second pivotal study triggered based on surpassing stringent 'Go/No-Go' assessment in COPD; not included in Dupixent® >€10bn ambition

Type 2 hallmark: Airways of Type 2 sub-population with Type 2 gene signature and blood/sputum eosinophilia

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COPD: Chronic obstructive pulmonary disease; FEV1: Forced expiratory volume in 1 second; CRSwNP: Chronic rhinosinusitis with nasal polyposis; ICS: Inhaled corticosteroids
(1) COPD with Type 2 phenotype uncontrolled with current SoC (patients that are on triple therapy among GOLD D - Global Initiative for Chronic Obstructive Lung Disease Group D (high risk, more symptoms))
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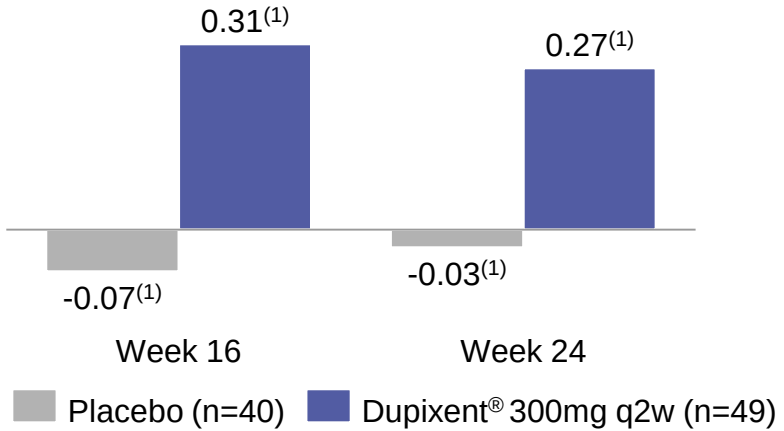
COPD: Significant improvement in lung functions within CRSwNP subset

COPD patient subset in SINUS trial with ≥1 of these criteria

- Forced expiratory volume in 1 second (FEV1), forced vital capacity (FVC) <0.70 and smoking history
- History of COPD
- Asthma and >10 pack-years smoking history
- Long-acting beta2-agonist(s) long-acting muscarinic antagonist (+/- inhaled corticosteroid) regimen and >10 pack-years smoking history

COPD patient subset demonstrated significant improvement in lung function

LS mean change from baseline in pre-BD FEV1, (L) in patients with clinical features of COPD



Results to be confirmed in Phase 3 trial; it is an investigational use and safety & efficacy have not been evaluated by regulatory authority
ICS: Inhaled corticosteroid; FEV1: Forced expiratory volume in 1 second; FVC: Forced vital capacity; LS: least squares; pre-BD: pre-bronchodilator; L: liters; CRSwNP: Chronic rhinosinusitis with nasal polyposis; COPD: Chronic obstructive pulmonary disease; q2w: once every other week
Source: J.F. Maspero et al, Am J Respir Crit Care Med 2020;201:A4544
(1) Standard error of (0.08)
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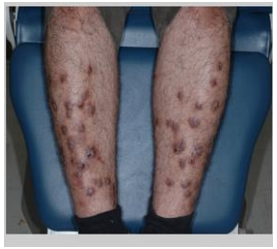
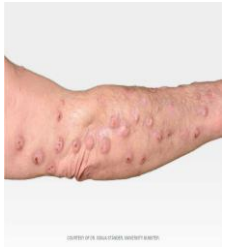
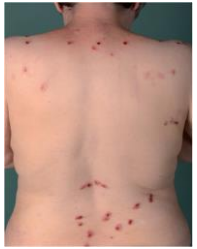
PN: Pruritic skin disease driven by Type 2 inflammation

High unmet need

- Intensely pruritic, chronic recalcitrant skin disease characterized by multiple lichenified papules and nodules
- Considerable impact on patients' quality of life due to the pruritus and sleep disturbance and depression
- No approved therapy; treated by TCS and antihistamines, if refractory phototherapy, cryotherapy, OCS or immunosuppressive prescription

Epidemiology

U.S. population (patients in '000, approximate)	
Prevalence	298
Biologics eligible(1)	74

Type 2 hallmark: Type 2 cytokines (IL-4 / IL-13) implicated in the pathogenesis and eosinophilia infiltration in lesions

PN: Prurigo nodularis; TCS: Topical corticosteroids; OCS: Oral corticosteroids
(1) Patients inadequately controlled by topical corticosteroids
Sources: Zeidler et al. Acta Derm Venereol 2018; 98: 173–179, Vaidya et al. Acta Dermatovenerol Croat 2008;16(1):38-44, Steinke et al. JAAD 2018; 79: 459-63
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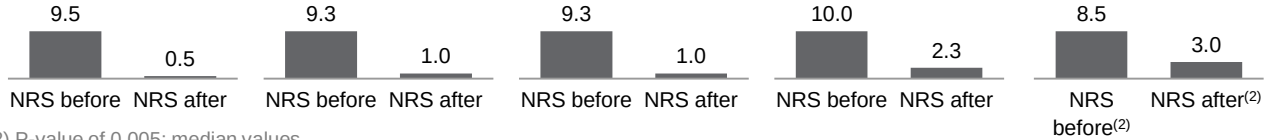
PN: Promising results seen from case series

	Calugareanu et al, 2019[1]	Beck et al, 2019	Mollanzaar 2019	Almustafa et al, 2019	Calugareanu et al, 2019[2]	Holm et al, 2020
Patients	1	3	4	3	16	3
Mean duration of pruritus (years)	9	8.6	NA	3.1	6[1]	13.1
Previous treatment *(non exhaustive)*	• TCS • Cryotherapy • Hydroxyzine Hydrochloride • MTX • Thalidomide • Cylcosporine	• TCS • Cryotherapy • Hydroxyzine Hydrochloride • Phototherapy • Doxepin • Dronabinol	• TCS • Topical calcineurin inhibitors • Phototherapy • Mirtazapine	• Phototherapy • TCS • Topical calcineurin inhibitors • Antihistamines	• TCS • Phototherapy • Methotrexate • Cyclosporine • Thalidomide	• UVB • Tacrolimus • PTX • Antihistamine • CS • Azathioprin • Cannabidiol
Treatment duration	8 months	3 months	3 months	6 months	12 months	4-7 months
Results post treatment (average)	After 3m: PN nodules steadily flattened					Itch markedly decreased, improved sleep



(1) Median (2) P-value of 0.005; median values
PN: Prurigo nodularis; NRS: Numerical rating scale; TCS: Topical corticosteroids; MTX methotrexate; UVB: ultra violet B; PTX: pentoxifylline; CS: cyclosporine
Note: The use of Dupixent® to treat PN is investigational and has not been reviewed by any regulatory agency
No side effects reported, except herpes labialis at w8 for 1 patient in Beck et al. 2019, dry eyes for 1 patient in Holm et al, 2020
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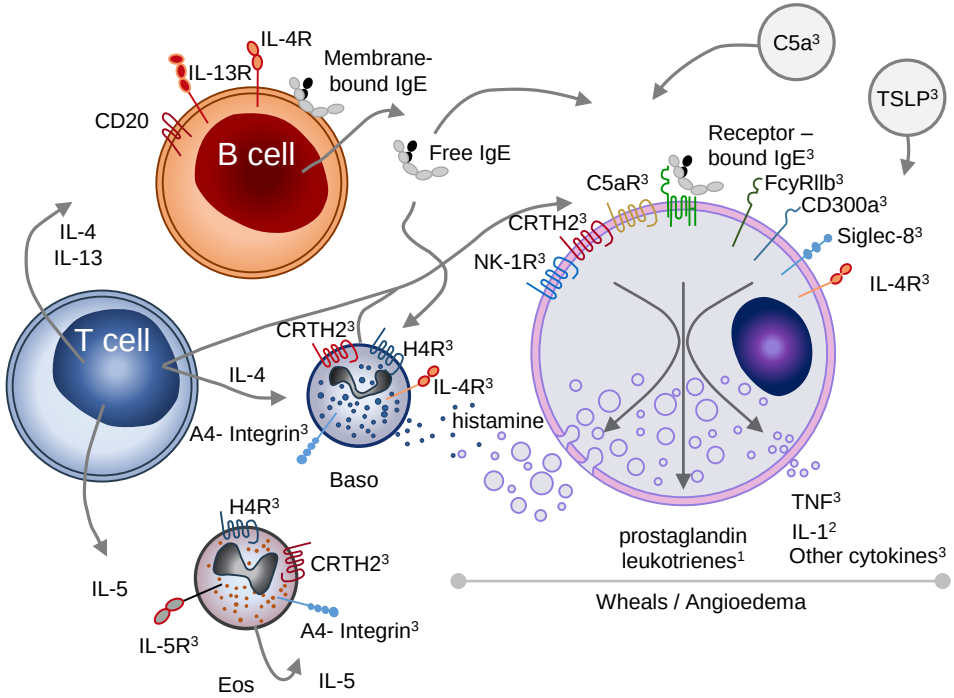
CSU: Dupixent® potential in mast cell related disease



- Highly pruritic lesions with significant impact on patients' quality of life
- U.S. prevalence of 1.5M patients, among which 308k biologics eligible(1)
- Existing treatments: antihistamines, omalizumab for patients not responding to antihistamines; but 40-50% not responding to omalizumab(2)
- Degranulated mast cells releasing histamines, proteases, cytokines (incl. IL-4 and IL-13), evidence of autoimmune IgG and IgE in ~40% of CSU patients with autoantibodies to FcER1 and IgE
- Rationale for Dupixent®: reduction of mast cell activation, by decreasing levels of IgE and directly inhibiting IL-4 (vs omalizumab targeting only IgE)

Type 2 hallmark: Eosinophilic infiltration in lesions, IgE and IL-4R implicated in mast cell hyper-reactivity

CSU: Chronic spontaneous urticaria; IgE: Immunoglobulin E; IgG: Immunoglobulin G; FcER1: high-affinity IgE receptor
(1) Patients uncontrolled on anti-histamines and other SoC (excluding biologics)
(2) Kaplan et al. J Allergy Clin Immunol, Volume 137, Number 2 (2015)
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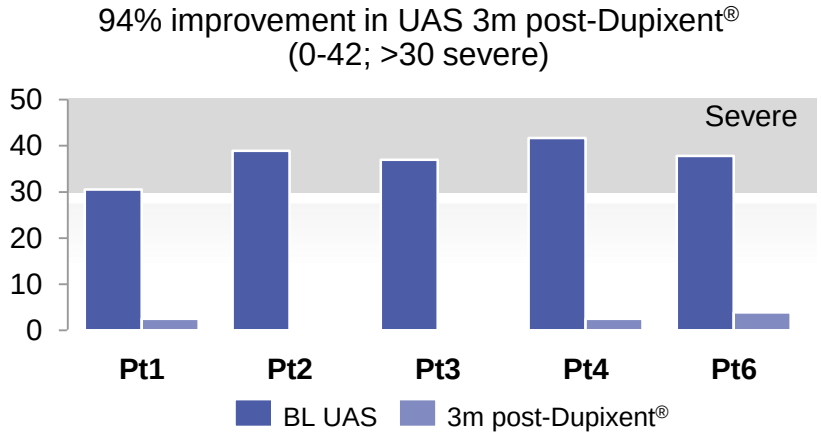
CSU: Clinical improvement observed in case series

Patient characteristics

- Severe CSU: baseline Urticaria Activity Score (UAS) between 31 to 42 (score range 0-42; score of ≥ 28 corresponds to severely active disease)
- Fail with antihistamines and omalizumab (treated for 4 to 12 months at 300 and 600mg dosed monthly)
- Medical historic of AD, asthma, juvenile idiopathic arthritis, autoimmune hypothyroid

Results



Note: Pt2 reported no urticaria after Dupixent®, but no formal score taken; Pt5 did not have baseline value

CSU: Chronic spontaneous urticaria; UAS: Urticaria activity score; AD: Atopic Dermatitis; Pt: Patient; BL: baseline; m: month
Use in CSU is investigational and safety/efficacy has not been evaluated by regulatory authority
Sources: Lee and Simpson. JACI in Practice. 2018
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Conclusion

John Reed

Global Head of Research & Development

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Reasons to believe in line extensions

	Epidemiology (U.S biologics eligible)	Type 2 hallmarks	Supporting evidence
Eosinophilic esophagitis	48k[2]	Type 2 gene signature and eosinophilic infiltration into the esophagus	P3 Part A: 69% reduction in dysphagia symptoms with Dupixent®, compared to 32% for placebo
COPD Type 2	300k[3]	Airways of Type 2 sub-population with Type 2 gene signature and blood/sputum eosinophilia	Evidence in COPD subset with CRSwNP (n=89): +0.22 L mean in pre-BD FEV 25 days post treatment
Prurigo nodularis	74k[4]	Type 2 cytokines (IL-4 / IL-13) implicated in the pathogenesis and eosinophilic infiltration in lesions	Promising results seen on itch and lesions in 6 case reports (n=30): from NRS baseline 8.5-10 to 0.5-3
Chronic spontaneous urticaria	308k[5]	Eosinophilic infiltration in lesions, IgE and IL-4R implicated in mast cell hyper-reactivity	94% improvement in Urticaria Activity Score in case series (n=6)
Bullous pemphigoid	27k[6]	High blood eosinophils and high IgE levels	Evidence in case series (n=3): free of itch between 1w and 3m and free of blisters 3-5m post treatment

NRS: Numerical rating scale; IgE: Immunoglobulin E; CRSwNP: Chronic rhinosinusitis with nasal polyposis; m: months: w: week; ICS: Inhaled corticosteroids
(1) Numerical Rating Score (2) Severe: uncontrolled on high dose PPI and topical steroid slurry and elimination diet / trigger avoidance (3) COPD with Type 2 phenotype uncontrolled with current SoC (patients that are on triple therapy among GOLD D) (4) Patients inadequately controlled by topical corticosteroids (5) Patients uncontrolled on anti-histamines and other SoC (excluding biologics) (6) 18 yo and plus; Patients on chronic OCS
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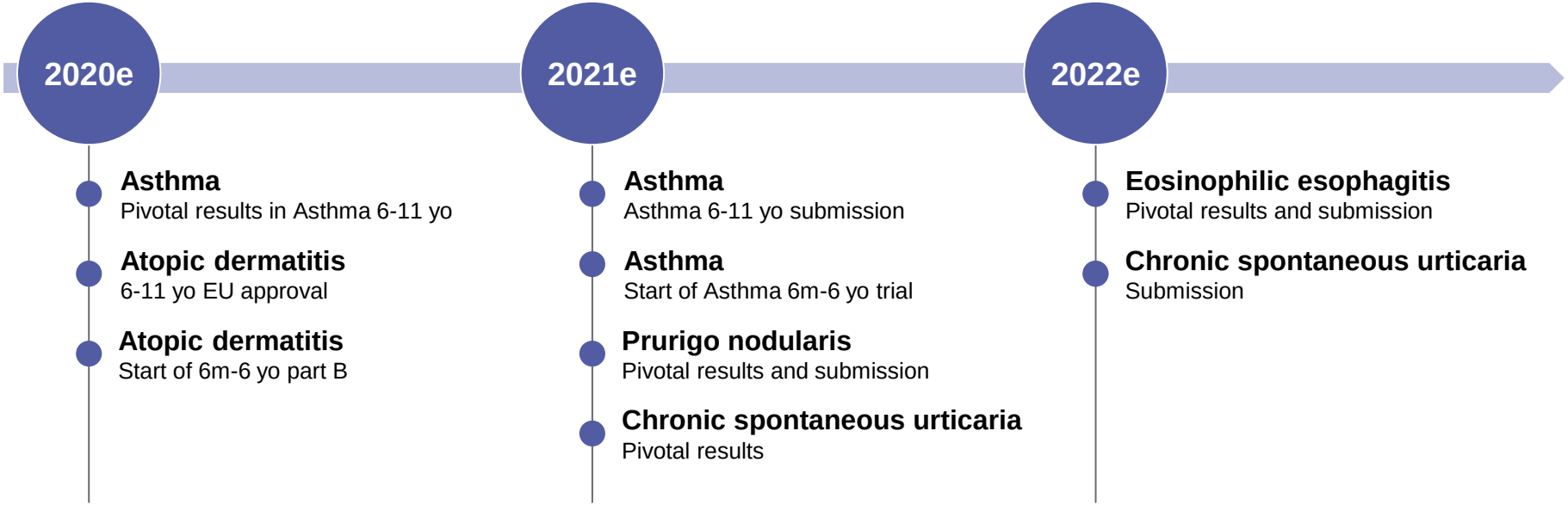
What you can expect from Dupixent® in the near term



Next wave of Type 2 inflammatory indications to be announced

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yo: years old
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Conclusion

Maximize patient benefits across several type 2 inflammatory diseases

- Type 2 inflammation is a maladapted immune response which leads to systemic barrier dysfunction, driving multiple chronic diseases occurring at different stages of life
- IL-4 and IL-13 play a critical role in Type 2 inflammation
- By targeting IL-4 and IL-13 only, Dupixent® enables systemic inhibition with great specificity, which we believe contributes to its efficacy and safety profile
- First pediatric approval for Dupixent® in the U.S. and long-term extension study in adult AD reconfirm strong safety and efficacy profile
- Strong confidence in Dupixent® line extensions across Type 2 diseases based on clinical data and real-world evidence

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AD: Atopic dermatitis
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Dupixent®: A leader in Type 2 inflammatory diseases

✓ Evidence generated from positive P3 trial

✗ No supporting evidence from P3 trial

		Efficacy[1]					Safety	
		AD	Asthma	CRSwNP	EoE[2]	Ped. data (<12 yo)	Anti-tumor immune response	Host defense against pathogens
DUPIXENT (dupilumab)	IL-4 & IL-13	✓	✓	✓	✓	✓	✓	✓
Targeted therapy	IL-13	✓	✗	✗	✗	✗	✓	✓
	IL-5	✗	✓	✓	✗	✓	✓	✓
	Anti-IgE	✗	✓	✓	✗	✓	✓	✓
	TSLP	✗	✓[3]	✗	✗	✗	✓	✓
Broad immuno-suppressant	JAK inhibitors	✓	✗	✗	✗	✗	✗	✗

AD: Atopic dermatitis; P3: Phase 3; CRSwNP: Chronic rhinosinusitis with nasal polyposis; EoE: Eosinophilic esophagitis
This slide is intended to show the breadth and scope of Dupixent data across various Type 2 inflammatory diseases and is not intended to suggest any comparison of safety or efficacy between the various therapeutic approaches in any given disease
(1) Type 2 indications where data from a pivotal trial have been released (2) Dupixent® in EoE is investigational and has not been fully reviewed by regulatory authorities
(3) Based on Phase 2b results
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Q&A session

John Reed
EVP, Global Head of R&D

Bill Sibold
EVP, Global Head of Specialty Care

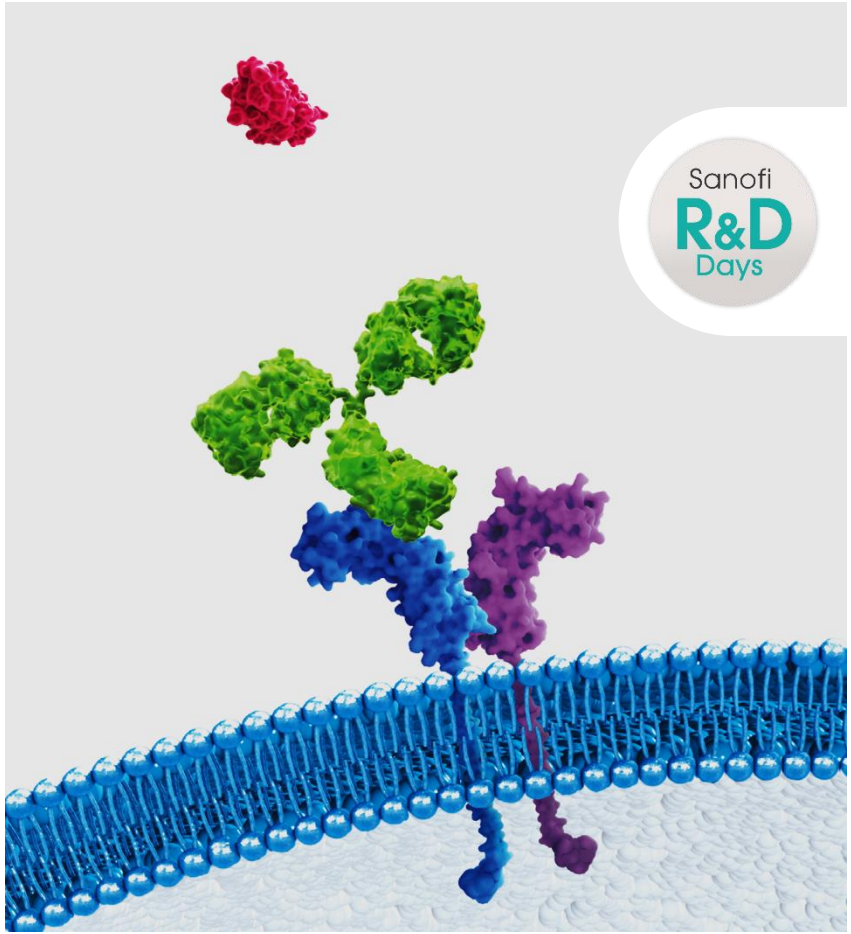
Brian Foard
Global Head of Dupixent® Franchise

Naimish Patel
Global Head of Development, Immunology & Inflammation

Paul Hudson
Chief Executive Officer

Jean-Baptiste de Chatillon
EVP, Chief Financial Officer

Frank Nestle
Global Head of Research, Immunology & Inflammation



Dupixent® IR event

Appendices

June 11, 2020

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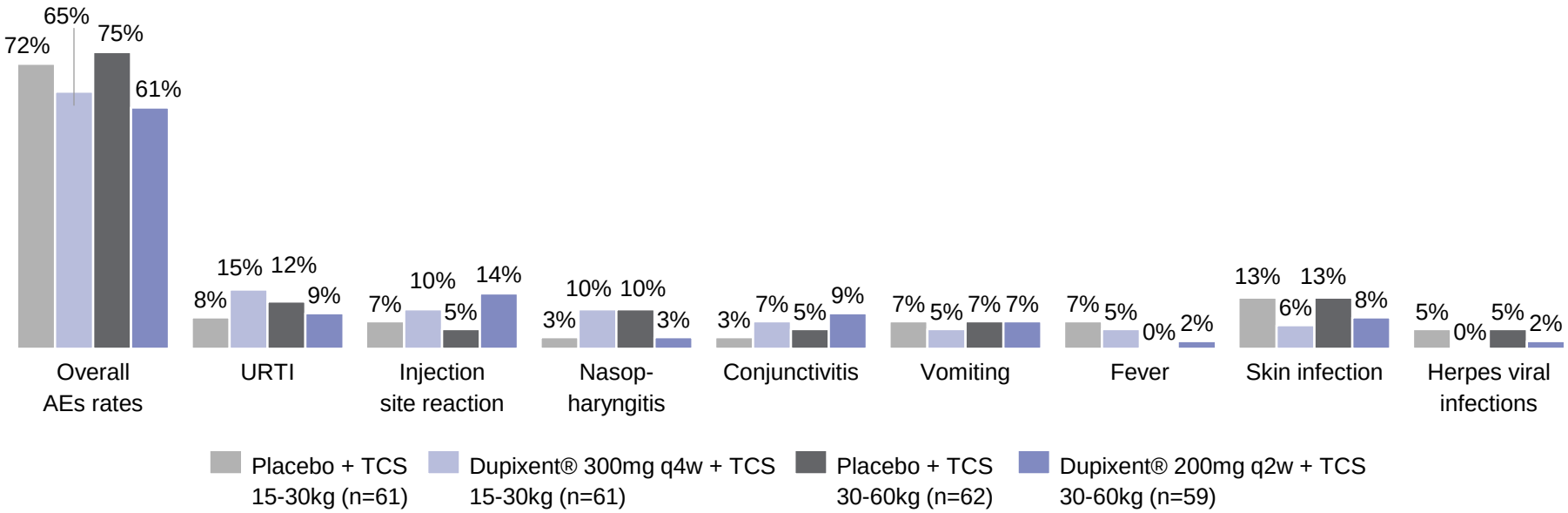
Dupixent® safety profile in pediatrics consistent with what has been seen in adults and adolescents

Rates of adverse events

% of patients (6-11 years with severe AD)



AD: Atopic dermatitis; AE: Adverse event; TCS: Topical corticosteroids; URTI: Upper respiratory tract infection
Source : 2020-05-26 Sanofi press release
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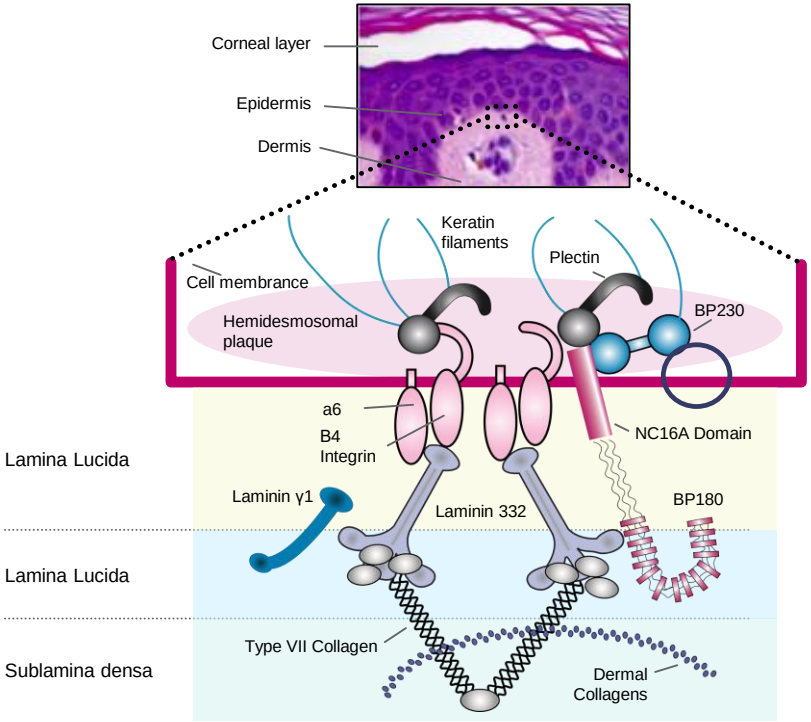
BP: Type 2 inflammation with limited treatment options



- Bullous pemphigoid is an autoimmune subepithelial blistering skin disease, predominately affecting the elderly (>age 60)
- ~52k patients in the U.S., with 27k biologics eligible(1)
- No FDA approved therapy
- Autoantibodies IgG4 and IgE predominant and driving Type 2 inflammation:
  - Increase of blood and tissue eosinophils and increase in IgE
  - Increase circulating / blister fluid levels of Type 2 cytokines, e.g. IL-4, IL-13, CCL17 (TARC), CCL18 (PARC)

Type 2 hallmark: High blood eosinophils and high IgE levels

BP: Bullous pemphigoid; IgE: Immunoglobulin E: IgG: Immunoglobulin G; OCS: Oral corticosteroids; TARC: Thymus and activation-regulated chemokine; PARC: Pulmonary activation-regulated chemokine
(1) Patients on chronic OCS
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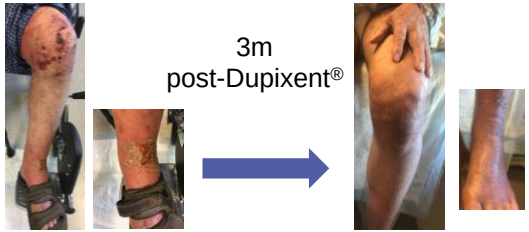
BP: Dupixent® efficacy observed in case series

	Patient 1 (1)	Patient 2 (2)	Patient 3 (2)
Patients characteristics	• Male >80 yo with biopsy proven BP not tapered down with high dose oral prednisone	• Female of 77 yo, with eosinophils at the sub-epi bullae • Failed treatment: mycophenylate, doxycycline, niacinamide	• Male of 77 yo, with eosinophils at the sub-epi zone • Failed treatment: Doxycycline
Treatment duration	12 months	> 5 months	> 3.5 months
Results post treatment	• Itch improvement in 1 week • Resolution of all blisters in 3 months • Patient still clear after 12 months on Dupixent® monotherapy	• Itch free in 3 months of Dupixent® treatment: 1-2/10 (itch rating) 3 months post treatment and 0/10 5 months post treatment • Blister free between 5 months	• Itch free in 2.5 months of Dupixent® treatment • Blister free in 3.5 months



BP: Bullous pemphigoid; yo: years old

(1) Kaye, Alex, et al. JAMA dermatology 154.10 (2018): 1225-1226 (2) Abdat, Rosmarin and al. Journal of the American Academy of Dermatology, March 2020

Results subject to verification in phase 3 study. Use has not been evaluated by any regulatory Authority

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