Ruxolitinib Cream: Phase 3 Data in Atopic Dermatitis

APRIL 6, 2020
SPEAKERS ON TODAY’S WEBCAST

- Hervé Hoppenot
  Chief Executive Officer, Incyte

- Lawrence Eichenfield, M.D.
  Professor of Dermatology and Pediatrics; Vice Chair, Department of Dermatology;
  Chief, Pediatric and Adolescent Dermatology
  UC San Diego School of Medicine and Rady Children's Hospital, San Diego

- Jim Lee, MD, PhD
  Group Vice President, Inflammation and Autoimmunity, Incyte
FORWARD-LOOKING STATEMENTS

Except for the historical information set forth herein, the matters set forth in this presentation contain predictions, estimates and other forward-looking statements, including without limitation statements regarding: expectations regarding the impact of COVID-19 on our business, including our commercial, supply and clinical and regulatory operations; our expectations regarding our contingency plans for our clinical trials; our expectations regarding FDA review and approval for pemigatinib, capmatinib and tafasitamab; our expectations regarding the NDA submission of ruxolitinib cream for atopic dermatitis; our expectations regarding the clinical trial of ruxolitinib for COVID-19; our expectations regarding an expanded access program for ruxolitinib for COVID-19 and the adequacy of our supply of ruxolitinib; and our expectations regarding the timelines of events for ruxolitinib for atopic dermatitis and for vitiligo, including timing of data results, commercial strategy and launch plans.

These forward-looking statements are based on our current expectations and are subject to risks and uncertainties that may cause actual results to differ materially, including unanticipated developments in and risks related to: unanticipated delays, including delays as a result of the COVID-19 outbreak and the measures taken to limit the outbreak; further research and development and the results of clinical trials possibly being unsuccessful or insufficient to meet applicable regulatory standards or warrant continued development; the ability to enroll sufficient numbers of subjects in clinical trials; determinations made by the FDA; our dependence on relationships with and changes in the plans and expenditures of our collaboration partners; the efficacy or safety of our and our collaboration partners’ product candidates; market competition; sales, marketing, manufacturing and distribution requirements, including our ability to successfully commercialize and build commercial infrastructure for any new products that become approved; and other risks detailed from time to time in our reports filed with the U.S. Securities and Exchange Commission, including our annual report on Form 10-K for the year ended December 31, 2019. We disclaim any intent or obligation to update these forward-looking statements.
BUSINESS UPDATE IN THE TIME OF COVID-19

PRIORITY IS TO ENSURE PATIENTS MAINTAIN ACCESS TO MEDICINES

- **Commercial**
  - No impact to date; Incyte to report Q1 2020 in early May

- **Supply**
  - No supply chain issues to date; manufacturing proceeding uninterrupted

- **Clinical & Regulatory**
  - Limited impact to date on key goals for the year
    - No impact to date on FDA timelines for pemigatinib, tafasitamab\(^1\) or capmatinib\(^2\)
    - No impact to date on expected NDA submission for ruxolitinib cream by end of 2020
    - Future impact may depend on disease state & severity, subjects, sites and geography
  - Priorities are to:
    - Ensure continuity of care for study subjects (eg. supplying drug directly to patients)
    - Maintain the integrity of the studies (eg. adopting and providing remote / tele-monitoring tools)
  - Plans to initiate Phase 3 and EAP trials for ruxolitinib as treatment for COVID-19 associated cytokine storm

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1. Development and U.S. commercialization of tafasitamab in collaboration with MorphoSys
2. Worldwide rights to capmatinib licensed to Novartis
TWO KEY RUXOLITINIB CREAM DEVELOPMENT PROGRAMS

**Atopic dermatitis**

**Phase 3** primary endpoints met; long-term extension ongoing

NDA expected late 2020

FDA decision expected late 2021

**Vitiligo**

**Phase 3** recruitment ongoing

Results & data expected 2021
ATOPIC DERMATITIS

LAWRENCE EICHENFIELD, M.D.
Atopic Dermatitis: Impact, Therapeutic Landscape and Clinical Need

Lawrence F. Eichenfield, M.D.
Professor of Dermatology and Pediatrics
Rady Children’s Hospital, San Diego
University of California, San Diego
The “Short Story” of Atopic Dermatitis

- High prevalence
  - 10 to 20% in children; 2 to 10% in adults²
- Variable course and severity
- Significant disease burden, comorbidities¹⁻³,⁵
- Historically, limited treatments beyond topical steroids
- High unmet need for long-term inflammatory control

Atopic Dermatitis: What Is It?

• Atopic dermatitis (AD) is the term for the most common type of eczema
• It is an inflammatory skin disease, often starting in childhood, with a chronic, intermittent or persistent course
• It manifests as eczematous rashes, itch, bacterial colonization and secondary infections
• Disease impact is multiplied by associations with allergies (food/environmental), asthma, hayfever, neuropsychiatric effects
Eczema is a Worldwide Issue

- Rates in industrialized countries:
  - 8-15% of children in the first few years of life
  - Rural, non-industrialized regions: 4 to 5%
- Rates “flip” with “westernization” or emigration to industrialized areas
- In teens and adults: May persist; Or new onset disease
  - 5-7% of adults in the US estimated to have AD

Silverberg J. EADV Congress 2018


Silverberg J. Dermatologic Clinics 2017 35(3):283-289
Most atopic dermatitis categorizes as mild to moderate.
Staph and Clinical Infection in AD

- Impetiginized AD
- Pustules
- Abcesses
- Rare: Cellulitis, sepsis, osteomyelitis, others
- Colonization, without infection
- Herpes infections
  - (eczema herpeticum)
Medical Consequences

- Chronic rashes
- Itch: Drives disease manifestations
- Infections (Bacterial, Viral)
- Sleep disturbance
- Atopic and non-atopic comorbidities
Atopic Dermatitis: Associated with Other Conditions!!

- Asthma
- Allergic Rhinitis and Conjunctivitis (Hayfever)
- Food Allergy
- Contact Allergy ("Occupational dermatitis")

Tremendous “cost multipliers” in disease impact
Comorbid Allergies: Prevalence

Infants

• US population-based study of >1000 infants with AD\(^1\)
  • Approximately 11% developed asthma
  • 37% had ≥1 atopic comorbidities
  • Development of allergic rhinitis and food allergy correlated with baseline severity of AD

Children

• Cross-sectional study of 2270 children with AD\(^2\)
  • Nearly 80% reported another form of allergy (asthma, AR, animal allergies, food allergies, drug allergies)
  • 33% also had symptoms of asthma or AR
  • 38% also had both asthma and AR

Adults

• Retrospective cohort study of >135,000 adults with AD
  • Patients with AD have a 33% greater risk for developing other atopic diseases compared with patients without AD\(^3\)
  • Overall, 63–76% of adult patients with AD, regardless of severity, suffer from at least one other atopic comorbidity\(^4,5\)

Atopic comorbidities in adults with moderate to severe AD (N=380)\(^6\)

Comorbidities and Atopic Dermatitis

• Mental Health Issues appear more common
• Anxiety and Depression with More Severe Disease
  • Approximately 1 in 5 adults: meet “diagnostic criteria” for major depression
• Attention Deficit Hyperactivity Disorder (ADHD) in younger children
• SLEEP DISTURBANCE and FATIGUE!

The Impact of Atopic Dermatitis

AD symptoms (esp. pruritus)
Frequent and intense itch

AD Eczematous Rashes/Flares
Signs and symptoms

School/Work productivity
Absence, presenteeism

Sleep disturbances
Difficulty falling asleep
Frequent awakenings

Atopic comorbidities
Asthma, nasal rhinitis, food allergies, allergic conjunctivitis

Other comorbidities
Bacterial, viral, fungal infections
Mental health disorders
Other immune-mediated diseases
*Cardiovascular disease
*Limited and conflicting evidence

Impaired Quality of Life
Daily activities, social functioning, and life-course decisions

Pathogenesis of AD: Barrier, Inflammation

Environmental factors/allergens

Itch

Scratch

Lichenification

Induced barrier defects:
Differentiation (filaggrin, ceramides, AMPs)

Barrier disruption

Barrier defects

IL-25, IL-33, TSLP

Barrier disruption

Th2

IL-31

IL-4, IL-13

IL-25, IL-33, TSLP

Type 2 immune response

Th2

IL-4, IL-13

ILC2

IL-13 production

Intensification of Type 2 effects

Th2

IL-31

IL-4, IL-13

IFN-γ

IL-31

CCL17 (TARC)

DC

Th1

Th2

Nonlesional skin

Acute lesional stage

Chronic lesional stage

Barrier disruption

Barrier defects

ILC2

IL-13 production

Th2

IL-4, IL-13

DC

Th1

IL-31

Th2

IL-4, IL-13

DC

Th1

IL-31

Th2

IL-4, IL-13

DC

Th1

IL-31

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IL-4, IL-13

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IL-4, IL-13

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IL-4, IL-13

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Th1

IL-31

Th2

IL-4, IL-13

DC

Th1

IL-31

Th2

IL-4, IL-13

DC

Th1

IL-31

Th2
WHAT THERAPIES ARE USED, AND WHAT CLINICAL NEEDS ARE THERE?
First Interventions: “Good Skin Care”

Bathing and moisturizing
Topical Corticosteroids

Traditional mainstay of therapy for Atopic Dermatitis
• Anti-inflammatory
• Used for acute flare management
• Intermittently for maintenance therapy

SIDE EFFECTS, CONCERNS, PHOBIAS:
• SYSTEMIC ABSORPTION
  • Concern with higher potency agents
• Local effects
  • Skin atrophy
  • Striae (stretch marks): irreversible complication
Topical Calcineurin Inhibitors

Anti-inflammatory medicines
Non-steroids

• Second-line therapy
  • Tacrolimus
  • Pimecrolimus

• Fair to good efficacy: mild, moderate, severe AD

• Stinging and burning may occur
Crisaborole 2% ointment: Topical PDE-4 Inhibitor

• Approved for patients ≥3 months old with mild-to-moderate atopic dermatitis
• Approved dosing: apply a thin layer twice daily to affected areas
• Appears Safe; Limited Efficacy
• Stinging and burning in subset of patients

Systemic Therapy: Moderate to Severe AD

• ‘Traditional Systemics:’
  • MTX most commonly used in US, but uncommonly used. Not approved
• Evolving area of Clinical Work..
• Dupilumab is first up of new agents

- Phototherapy
- Cyclosporine
- Azathioprine
- Methotrexate
- Mycophenolate mofetil
- Systemic steroids (Approved, not advised)
- Dupilumab (FDA Approved 12+)

https://www.accessdata.fda.gov/drugsatfda_docs/label/2017/761055lbl.pdf
Evolving Systemic Agents

Systemic Agents
• Oral JAK Inhibitors
• IL-13 Blockers (Lebrikizumab; Tralokinumab)
• Nemolizumab (IL31 blocker)

Multiple Others!
Integrating the New with the Standard

• Mild to Moderate Disease: MOST ECZEMA!
• **TOPICALS** WILL STILL HANDLE MOST DISEASE!
• There IS GREAT NEED for a more potent, well tolerated non-steroid topical agent that is anti-inflammatory and effectively decreases itch!
• The market is still relatively untouched! Much work to be done to establish long term disease control!
TRUE-AD1 & TRUE-AD2
PHASE 3 DATA

JIM LEE, MD, PHD
Efficacy and Safety of Ruxolitinib Cream for the Treatment of Atopic Dermatitis: Results From Two Phase 3, Randomized, Double-Blind Studies

Kim Papp, MD, PhD, 1 Jacek C. Szepietowski, MD, PhD, 2 Leon Kircik, MD, 3 Darryl Toth, MD, 4 Michael E. Kuligowski, MD, PhD, MBA, 5 May Venturanza, MD, 5 Kang Sun, PhD, 5 Eric Simpson, MD 6

1K. Papp Clinical Research and Probity Medical Research, Waterloo, ON, Canada; 2Department of Dermatology, Venereology and Allergology, Wroclaw Medical University, Wroclaw, Poland; 3Icahn School of Medicine at Mount Sinai, New York, NY, USA; 4XLR8 Medical Research and Probity Medical Research, Windsor, ON, Canada; 5Incyte Corporation, Wilmington, DE, USA; 6Oregon Health and Science University, Portland, OR, USA
Atopic dermatitis (AD) is a chronic, inflammatory skin disease that greatly impacts patients’ quality of life. JAKs modulate inflammatory cytokines involved in the pathogenesis of AD and may also directly modulate itch. Ruxolitinib (RUX) is a potent, selective inhibitor of JAK1 and JAK2.

In a phase 2 study (NCT03011892), RUX cream provided dose-dependent efficacy in patients with AD, with no notable adverse events.

Objective: To report efficacy and safety of RUX cream in patients with AD in two phase 3 studies (TRuE-AD1 [NCT03745638] and TRuE-AD2 [NCT03745651]).

IgE, immunoglobulin E; IL, interleukin; JAK, Janus kinase; STAT, signal transducer and activator of transcription; TYK2, tyrosine kinase 2.

STUDY DESIGN

Vehicle-controlled (VC) period (8 continuous weeks)

- **0.75% RUX BID** (n=~240 in each study)
- **1.5% RUX BID** (n=~240 in each study)
- **Vehicle** (n=~120 in each study)

Long-term safety (treat as needed for 44 weeks)

- **Patients initially randomized to RUX remain on their regimen**
- **Patients on vehicle randomized 1:1 to 0.75% RUX or 1.5% RUX**

Visits every 4 weeks

- **Recurrence of lesions**
- **Clearance of lesions**

Day 1  |  Week 8  |  Week 52

**Patients Randomized 2:2:1**

*BID, twice daily; BSA, body surface area.
* Patients will self-evaluate recurrence of lesions between study visits and will treat lesions with active AD (≤20% BSA). If lesions clear between study visits, patients will stop treatment 3 days after lesion disappearance. If new lesions are extensive or appear in new areas, patients will contact the investigator to determine if an additional visit is needed.
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STUDY ENDPOINTS

- **Primary Endpoint**
  - Proportion of patients achieving IGA-TS (score of 0/1 with ≥2-grade improvement from baseline) at Week 8

- **Main Secondary Endpoints**
  - Proportion of patients achieving ≥75% improvement in EASI score vs baseline (EASI-75)
  - Proportion of patients with a ≥4-point improvement in itch NRS score from baseline to Week 8
ELIGIBILITY CRITERIA

- **Key Inclusion Criteria**
  - Patients aged ≥12 years with AD ≥2 years
  - IGA score of 2 or 3
  - 3%–20% affected BSA

- **Key Exclusion Criteria**
  - Unstable course of AD
  - Other types of eczema
  - Immunocompromised status
  - Any serious illness/medical condition that could interfere with study conduct, interpretation of data, or patients’ well-being
  - Use of AD systemic therapies during the washout period and during the study
  - Use of AD topical therapies (except bland emollients) during the washout period and during the study
PATIENT DEMOGRAPHICS

Distribution of baseline demographics was similar across treatment groups

<table>
<thead>
<tr>
<th></th>
<th>TRuE-AD1</th>
<th>TRuE-AD2</th>
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<tbody>
<tr>
<td></td>
<td>Vehicle (n=126)</td>
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<td>0.75% RUX (n=252)</td>
<td>0.75% RUX (n=248)</td>
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<td>1.5% RUX (n=253)</td>
<td>1.5% RUX (n=246)</td>
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<tr>
<td>Age, median (range), y</td>
<td>Age, median (range), y</td>
<td>Age, median (range), y</td>
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<tr>
<td>12–17, n (%)</td>
<td>31.5 (12–82)</td>
<td>37.5 (12–82)</td>
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<td>23 (18.3)</td>
<td>22 (17.7)</td>
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<td>103 (81.7)</td>
<td>102 (82.3)</td>
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<td>≥18, n (%)</td>
<td>34.0 (12–85)</td>
<td>33.0 (12–81)</td>
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<td>53 (21.0)</td>
<td>55 (22.2)</td>
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<td>199 (79.0)</td>
<td>193 (77.8)</td>
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<td>30.0 (12–77)</td>
<td>32.0 (12–85)</td>
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<td>47 (18.6)</td>
<td>45 (18.3)</td>
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<td>206 (81.4)</td>
<td>201 (81.7)</td>
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<td>Female, n (%)</td>
<td>79 (62.7)</td>
<td>80 (64.5)</td>
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<td>154 (61.1)</td>
<td>150 (60.5)</td>
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<td>158 (62.5)</td>
<td>150 (61.0)</td>
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<td>Race, n (%)*</td>
<td>White</td>
<td>White</td>
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<td>85 (67.5)</td>
<td>84 (67.7)</td>
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<td>171 (67.9)</td>
<td>174 (70.2)</td>
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<td>175 (69.2)</td>
<td>178 (72.4)</td>
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<td>29 (23.0)</td>
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<td>56 (22.1)</td>
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<td>Other</td>
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<td>12 (9.5)</td>
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<td>26 (10.3)</td>
<td>11 (4.4)</td>
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<td>21 (8.3)</td>
<td>11 (4.5)</td>
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<td>Region, n (%)</td>
<td>North America</td>
<td>Europe</td>
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<td>88 (69.8)</td>
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<td>176 (69.8)</td>
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<td>38 (30.2)</td>
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<td>77 (30.4)</td>
<td>81 (32.9)</td>
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* Data missing from 1 patient in the 1.5% RUX group in TRuE-AD1.
### PATIENT CLINICAL CHARACTERISTICS

Distribution of baseline clinical characteristics was similar across treatment groups

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<thead>
<tr>
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<td>BSA, mean ± SD, %</td>
<td>9.2±5.1</td>
<td>9.9±5.4</td>
<td>9.3±5.2</td>
<td>10.1±5.8</td>
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<td>10.1±5.3</td>
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<td>9.9±5.4</td>
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<td>Baseline EASI, mean ± SD</td>
<td>7.4±4.3</td>
<td>8.2±4.8</td>
<td>7.9±4.6</td>
<td>8.2±5.2</td>
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<td>8.1±5.0</td>
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<td>7.8±4.9</td>
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<td>Baseline IGA, n (%)</td>
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<td>31 (24.6)</td>
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<td>95 (75.4)</td>
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<td>Itch NRS score, mean ± SD</td>
<td>5.1±2.5</td>
<td>5.1±2.3</td>
<td>5.2±2.5</td>
<td>5.1±2.4</td>
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<td>5.2±2.5</td>
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<td>4.9±2.5</td>
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<td>Itch NRS score ≥4, n (%)</td>
<td>78 (61.9)</td>
<td>156 (61.9)</td>
<td>161 (63.6)</td>
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<td>Duration of disease, median (range), y</td>
<td>17.9 (1.9–79.1)</td>
<td>14.1 (1.0–68.8)</td>
<td>16.0 (0–69.2)</td>
<td>15.9 (0.8–70.7)</td>
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<td>16.6 (0–68.8)</td>
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<td>Facial involvement, n (%)</td>
<td>52 (41.3)</td>
<td>112 (44.4)</td>
<td>118 (46.6)</td>
<td>41 (33.1)</td>
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SAFETY

- RUX cream was well tolerated and not associated with clinically significant application site reactions
- All treatment-related TEAEs were mild or moderate in severity
- No TEAEs suggestive of a relationship to systemic exposure were observed

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<td>Patients with TEAE, n (%)</td>
<td>44 (34.9)</td>
<td>40 (32.3)</td>
</tr>
<tr>
<td></td>
<td>74 (29.4)</td>
<td>73 (29.4)</td>
</tr>
<tr>
<td></td>
<td>73 (28.9)</td>
<td>58 (23.6)</td>
</tr>
<tr>
<td>Treatment-related TEAE, n (%)</td>
<td>16 (12.7)</td>
<td>12 (9.7)</td>
</tr>
<tr>
<td></td>
<td>15 (6.0)</td>
<td>8 (3.2)</td>
</tr>
<tr>
<td></td>
<td>14 (5.5)</td>
<td>11 (4.5)</td>
</tr>
<tr>
<td>Most common treatment-related TEAEs, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Application site burning</td>
<td>2 (1.6)</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>2 (0.8)</td>
</tr>
<tr>
<td></td>
<td>8 (6.5)</td>
<td>2 (0.8)</td>
</tr>
<tr>
<td></td>
<td>2 (0.8)</td>
<td>0</td>
</tr>
<tr>
<td>Application site pruritus</td>
<td>2 (1.6)</td>
<td>2 (0.8)</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>4 (3.2)</td>
<td>2 (0.8)</td>
</tr>
<tr>
<td></td>
<td>2 (0.8)</td>
<td>0</td>
</tr>
<tr>
<td>Pruritus</td>
<td>2 (1.6)</td>
<td>2 (0.8)</td>
</tr>
<tr>
<td></td>
<td>1 (0.4)</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Discontinuation due to a TEAE, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>5 (4.0)</td>
<td>3 (1.2)</td>
</tr>
<tr>
<td></td>
<td>3 (1.2)</td>
<td>3 (2.4)</td>
</tr>
<tr>
<td></td>
<td>3 (1.2)</td>
<td>1 (0.4)</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Serious TEAE, n (%)*</td>
<td>2 (1.6)</td>
<td>1 (0.4)</td>
</tr>
<tr>
<td></td>
<td>2 (0.8)</td>
<td>3 (1.2)</td>
</tr>
<tr>
<td></td>
<td>3 (1.2)</td>
<td>1 (0.4)</td>
</tr>
</tbody>
</table>

TEAE, treatment-emergent adverse event.
* No serious TEAEs were related to RUX treatment.
PATIENT DISPOSITION DURING THE VC PERIOD

**TRuE-AD1**

- Randomized: n=631
- Discontinued: n=73 (11.6%)
  - Withdrawal by patient: n=31 (4.9%)
  - Lost to follow-up: n=24 (3.8%)
  - Adverse event: n=10 (1.6%)
  - Protocol deviation: n=2 (0.3%)
  - Noncompliance with study drug: n=1 (0.2%)
  - Physician decision: n=1 (0.2%)
  - Pregnancy: n=1 (0.2%)
  - Other: n=1 (0.2%)
- Completed: n=558 (88.4%)

**TRuE-AD2**

- Randomized: n=618
- Discontinued: n=57 (9.2%)
  - Withdrawal by patient: n=24 (3.9%)
  - Lost to follow-up: n=20 (3.2%)
  - Adverse event: n=5 (0.8%)
  - Protocol deviation: n=3 (0.5%)
  - Noncompliance with study drug: n=1 (0.2%)
  - Physician decision: n=1 (0.2%)
  - Pregnancy: n=0
  - Other: n=2 (0.3%)
- Completed: n=561 (90.8%)

* All randomized patients were included in the efficacy analysis. † Efficacy population consisted of 577 patients (vehicle, n=118; 0.75% RUX, n=231; 1.5% RUX, n=228).
PROPORTION OF PATIENTS WITH IGA-TS

Significantly more patients treated with RUX cream regimens vs vehicle demonstrated IGA-TS (primary endpoint); responses were time and dose dependent

TRuE-AD1

TRuE-AD2

SE, standard error.
*** P<0.0001.
†† Defined as patients achieving an IGA score of 0 or 1 with an improvement of ≥2 points from baseline.
PROPORTION OF PATIENTS ACHIEVING EASI-75

Significantly more patients treated with RUX cream achieved EASI-75 vs vehicle; responses were time and dose dependent.

TRuE-AD1

- Vehicle (n=126)
- 0.75% RUX (n=252)
- 1.5% RUX (n=228)

TRuE-AD2

- Vehicle (n=118)
- 0.75% RUX (n=231)
- 1.5% RUX (n=228)

*** P<0.0001.
Both strengths of RUX cream showed greater improvement in mean percentage change in EASI scores vs vehicle; statistical significance was observed at Week 2 and later.
Significantly greater itch reductions in itch NRS scores were observed within 12 hours of the first application of RUX cream (1.5%; \( P<0.05 \)) vs vehicle.
≥4-POINT IMPROVEMENT IN ITCH NRS

Significantly more patients treated with RUX cream demonstrated clinically meaningful reduction in itch (≥4-point improvement in itch NRS) vs vehicle.

**Patients in the analysis had an NRS score ≥4 at baseline.**

**P<0.001; ***P<0.0001.**
CONCLUSIONS

- Application of ruxolitinib cream brought about rapid (within 12 hours of initiation of therapy), substantial, and sustained reduction in itch
- Ruxolitinib cream showed superior efficacy vs vehicle in IGA-TS, EASI-75, and ≥4-point reduction in itch NRS score in these two phase 3 studies
- Ruxolitinib cream demonstrated a dual mode of action: antipruritic and anti-inflammatory
- No notable safety findings (either local or systemic) were associated with treatment, including on sensitive skin areas
- The successful outcomes of TRuE-AD1 and TRuE-AD2 support the potential of ruxolitinib cream as an effective and well-tolerated topical treatment for patients with AD
INCYTE TO COMMERCIALIZE RUX CREAM IN THE U.S.¹

NEAR-TERM OPPORTUNITY TO FURTHER DIVERSIFY REVENUE

Planning for commercial success

Targeting key prescribers
- 8,000 medical dermatologists

Dedicated division planned
- Commercial deployment expected in 2021
- ~150 field-based FTE’s

Expected launch
- 2021
- 2022

Atopic dermatitis
- Prevalence: ~10 million²
- Estimated duration of therapy: 4-6 months / year
- Eucrisa: ~$600 / month
- Limitations: Modest efficacy; stinging & burning

Vitiligo
- Prevalence: >1.5 million
- Estimated duration of therapy: 12 months / year
- Phototherapy: ~$1,000 / month
- Patient compliance

Current Therapies

1. If approved by FDA
2. Diagnosed and treated mild/moderate AD patients (aged ≥ 12 years)
3. Estimated WAC price of Eucrisa ~$600 per 60g tube; estimated cost of phototherapy based on lower price of two reimbursement codes (price can be as much as $20-25,000 per year, reimbursed price generally significantly lower)