This presentation contains forward-looking statements and information. The use of words such as “may,” “might,” “will,” “should,” “expect,” “plan,” “anticipate,” “believe,” “estimate,” “project,” “intend,” “future,” “potential,” or “continue,” and other similar expressions are intended to identify forward-looking statements. These forward-looking statements are predictions and involve risks and uncertainties such that actual results may differ materially from these statements. These risks and uncertainties include, among others: actions by the FDA and other regulatory agencies, results and timing of current and planned clinical trials, risks related to the commercialization of our products, our ability to manufacture sufficient quantities of products for clinical trials and commercial launch, and those other risks detailed from time to time under the caption “Risk Factors” and elsewhere in Krystal Biotech’s Securities and Exchange Commission (SEC) filings included in our Annual Report on Form 10-K for the year ending December 31, 2017, and in future filings and reports of Krystal Biotech. The Company undertakes no duty or obligation to update any forward-looking statements as a result of new information, future events or changes in its expectations.
Company Overview

- NASDAQ: KRYS; started operations in 2016 with headquarters in Pittsburgh, Pennsylvania.

- Established a proprietary fully-integrated HSV-1-based gene therapy platform and a pipeline of clinical and non-clinical effectors to target rare diseases and conditions. Zero royalty burden.

- Successful completion of Gem-1 (phase I) and Gem-2 (phase II) study of KB103.
  - Received regenerative medicine advanced therapy (RMAT) designation from the FDA
  - Priority Medicines (PRIME) designation awarded by EMA
  - Pivotal study anticipated to begin in 2H 2019

- Investigation New Drug (IND) application for KB105 submitted to FDA

- US Patents 9,877,990 (issued 1/16/18) and 10,155,016 (issued 12/18/18) covering pharmaceutical compositions and methods of their use.

- First GMP in-house manufacturing facility in Pittsburgh, PA complete. Plans to build a second GMP facility in motion.

- Insider ownership (management, employees, directors): approximately 30% of fully diluted shares outstanding (as of 6/30/19)
Fully-Integrated Vector Platform

Modified Herpes Simplex Virus 1 (HSV-1) vector well suited to treat skin diseases

**Proprietary Vectors**
and underlying cell lines support robust and flexible drug production

**Direct delivery**
Topical administration for open wounds and intradermal for intact skin

**Reproducible and Scalable Manufacturing**
using internally developed and validated protocols

**Non-integrating**
into the DNA making it safer

**Stability**
of vector beneficial to production and storage

**High Transduction Efficiency**
Transduces dividing and non-dividing skin cells

**Non-Replicating**
Safe for repeat administration; transient transgene expression, diluted by cell divisions

**Regulatory precedent**
HSV-1 used as backbone in Amgen’s Imlygic®, which is approved for melanoma and administered weekly to patients

**Significant payload capacity**
due to ~150Kb genome to accommodate multiple genes and effectors in the backbone
Krystal’s Unique and Straightforward Approach

“Off-the-shelf” gene therapy with repeat administration

Wild-type HSV-1 → Vector Platform → Therapeutic Vector → Drug Product

- Targeted mutagenesis of HSV-1, resulting in platform that is both safe and effective for delivery of effector(s) to human skin
- Modify platform via insertion of one or more therapeutic effectors of interest
- Formulate into a topical gel or intradermal solution
- Apply locally to the skin
1. Severe monogenic skin diseases

**Indications:** Dystrophic Epidermolysis Bullosa (DEB); Autosomal Recessive Congenital Ichthyosis (ARCI); Junctional Epidermolysis Bullosa (JEB); Netherton Syndrome (NS)

**Effectors:** COL7A1 (type VII collagen); TGM1 (transglutaminase-1); laminins; SPINK5 (serine protease inhibitor k kazal-type 5)

2. Aesthetic conditions

**Indications:** Fine lines; nasolabial folds; glabellar lines; depressed scars; UV-induced skin damage

**Effectors:** Collagens

3. Chronic skin diseases

**Indications:** Atopic dermatitis; psoriasis; rosacea; acne

**Effectors:** Anti-inflammatory antibodies and antibody fragments

**Krystal’s Approach:** Applicable to a Wide Range of Skin Diseases and Conditions
## Krystal’s Current Pipeline

<table>
<thead>
<tr>
<th>Product</th>
<th>Indication</th>
<th>Discovery</th>
<th>Preclinical</th>
<th>Phase I/II</th>
<th>Phase III</th>
<th>Marketed</th>
</tr>
</thead>
<tbody>
<tr>
<td>KB103†‡§</td>
<td>Dystrophic EB</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>KB105†</td>
<td>TGM1-deficient ARCI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>IND filed June 13th, 2019</td>
</tr>
<tr>
<td>KB301 / KB302</td>
<td>Aesthetic Skin Conditions</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>IND to be filed 2H 2019</td>
</tr>
<tr>
<td>KB104</td>
<td>Netherton Syndrome</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>IND to be filed 1H 2020</td>
</tr>
<tr>
<td>KB5XX</td>
<td>Chronic Skin Diseases</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

†: FDA Orphan Drug Designation;  
‡: FDA Rare Pediatric Disease Designation;  
*: Fast-track Designation;  
Δ: FDA RMAT designation;  
‡: EMA Orphan Drug Designation;  
§: EMA PRIME Designation.
Upcoming Milestones

• Commence pivotal phase III trial for KB103 (DEB); 2H 2019

• Commence phase I/II trial for KB105 (ARCI); 2H 2019

• File IND for KB301 (aesthetics); 2H 2019

• File IND for KB104 (Netherton Syndrome); 1H 2020

• Commence clinical trial for KB103 in EU; 1H 2020

• Break ground on second GMP manufacturing facility; 1H 2020
For treatment of dystrophic epidermolysis bullosa (DEB)

* RMAT designation;
Prime Eligibility;
Fast Track Designation Granted;
Orphan Drug Designation in US and EU;
Rare Pediatric Disease Designation in US;
Eligible for Priority Review Voucher.
Dystrophic Epidermolysis Bullosa (DEB)

“Butterfly Children” is used to describe young DEB patients because their skin is as fragile as a butterfly’s wings.

Dystrophic Epidermolysis Bullosa
A rare, genetic connective tissue disease that causes skin to tear or blister from minor contact
Caused by a mutation in the COL7A1 gene that codes for the COL7 protein
Without COL7 the epidermis does not anchor to the dermis

Epidemiology
Prevalence: Up to 125,000 people are affected by DEB worldwide1
Incidence: The incidence of DEB is 6.5 per million births in the US2

Current Standard of Care
There are no approved treatments for DEB
Existing therapies limited to expensive and time-consuming palliative treatments
Palliative treatments cost $200k – $400k annually3,4

Simple, Painless and Easy to Administer

**Off-the-shelf, Non-Invasive Modified HSV-1 Therapy**

**Drug Product Preparation**
- Modified Vector (KB103)
- Topical formulation

**Topical formulation**
- 1. Biopsy
- 2. Primary cell culture
- 3. Cell transduction & genomic integration
- 4. Selection & expansion
- 5. Engraftment or injection

**Competitive Approaches:**
- Autologous, *Ex Vivo* Cell Therapy

**Modified Vector**
- Fibroblast OR Keratinocyte

**Drug Product Preparation**
**Simple, Painless and Easy to Administer**

**Benefits of Krystal’s approach vs. autologous therapy:**
- Drug product ready for use in multiple patients
- Manufacturing and supply chain costs are lower
- Therapy can be administered by any dermatologist
- Outpatient; no hospitalization needed
- Does not require expensive, invasive, and time-consuming procedures or sophisticated medical teams

**Off-the-shelf, Non-Invasive Modified HSV-1 Therapy**

1. Modified Vector (KB103)
2. Topical formulation
3. Topical application
**KB103 Mechanism of Action**

1. **KB103 enters the compromised skin of DEB patients and transduces both keratinocytes and fibroblasts**
2. The drug enters the nucleus of transduced cells and the vector genome is deposited (episomally)
3. **COL7A1 transcripts are generated, which allows the cell to produce and secrete functional COL7 protein**
4. The secreted COL7 protein assembles into anchoring fibrils which hold the epidermis and dermis together

---

**KB103 Mechanism of Action Diagram**

**KB103**

1. **KB103 enters the compromised skin of DEB patients and transduces both keratinocytes and fibroblasts.**
2. The drug enters the nucleus of transduced cells and the vector genome is deposited (episomally).
3. **COL7A1 transcripts are generated, which allows the cell to produce and secrete functional COL7 protein.**
4. The secreted COL7 protein assembles into anchoring fibrils which hold the epidermis and dermis together.

---

**Untreated DEB Patient Skin**

- Keratinocytes
- Dermoepidermal Junction
- Anchoring Fibrils (Collagen VII)
- Fibroblasts
- Blister

**KB103-treated Skin**

- Keratinocytes
- Dermoepidermal Junction
- Anchoring Fibrils (Collagen VII)
- Fibroblasts
KB103 Status

- **2018**
  - Q4: Interim phase I data announced
  - Four additional patients (total of 6) dosed in Phase II study

- **2019**
  - 1H: Announced phase I/II data

- **2020**
  - 1H: Initiate pivotal phase III study
  - 2H: Initiate clinical study in EU
  - File Biologics License Application (BLA)
KB103 Clinical Data
A phase I study of KB103, a non-integrating, replication-incompetent HSV vector expressing the human collagen VII protein, for the treatment of dystrophic epidermolysis bullosa (DEB)

- Key objectives: Demonstrate efficacy and safety of KB103
- Primary Objectives: Expression of COL7, presence of anchoring fibrils, and safety
- Secondary Objectives: Change in wound area, duration of wound closure, time to wound closure
- Principal investigator: Dr. Peter Marinkovich, MD, Dermatologist, Stanford University
- Trial Design:
  - Randomized, open-label, placebo controlled
  - 2 wounds treated topically: 1 placebo, 1 active
  - 1 intact site treated intradermally
  - Patients were evaluated for COL7 expression by immunofluorescence and for the presence of anchoring fibrils by electron microscopy
  - Initial dosing at Day 0 and a repeat dose a month later; Patient 02 was additionally dosed on Day 14 and Day 42 by PI to understand impact of incremental dose escalation
Summary

• Results to date on 2 patients met all primary efficacy (presence of functional COL7 expression as early as Day 2 of treatment, observation of NC1 and NC2 reactive anchoring fibrils and continued expression following repeat administration) and safety endpoints (no adverse events, inflammation or irritation) in topically administered KB103 wounds.

• With respect to secondary endpoints – topically administered KB103 wounds closed in 2 weeks and remained closed through the last timepoint representing 5.7 and 6.6 months of closure, respectively, for Patients 01 and 02. Topically administered placebo treated wounds took 10 weeks to close in Patient 01 and did not completely close throughout the study in Patient 02.

• KB103 treated skin shows presence of functional COL7 expression and anchoring fibrils in both patients.

• Empirical observation that one patient discontinued use of bandages at the site of a KB103-treated area, an area which had required bandages for several months prior to administration.
KB103 continues to be well tolerated to date following first and repeat dose

- No treatment-related adverse events (serious or otherwise) were reported.

- No immune response or blistering observed around the sites of administration following first and repeat dose.

- Blood and urine samples collected throughout the study revealed:
  - No viral shedding
  - No adverse events associated with routine labs (chemistry and hematology)
  - No antibodies to COL7 were detected
GEM-2: Phase II Trial Design

Four patients enrolled in December 2018. Principal Investigator: Dr. Peter Marinkovich, Stanford University

**Key objectives:** Demonstrate efficacy and safety of KB103

- **Primary Clinical Objectives:** Safety and Wound healing (time to wound closure, % area of wound closure, duration of wound closure)
- **Secondary Mechanistic Objectives:** Expression of COL7, evidence of anchoring fibrils.

**Trial Design:**

- Randomized, placebo-controlled study; 4 patients enrolled in study – 2 adult 2 pediatric
- 3 wounds treated topically in each patient: 1 placebo, 2 active
- Total number of wounds treated in phase II study = 12 (3 wounds per patient * 4 patients)
- Initial front-loaded dosing for 5 days (3e8 pfu/day)
- Biopsies were based on PI discretion during site visits.
- Biopsied wounds were dosed one administration of 3e8 at site of biopsy, following a biopsy
- Each patient is on-study for approximately six months; three months of on-site visits followed by a 3-month at-home imaging period
### Phase II Trial

<table>
<thead>
<tr>
<th>Category</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total # of wounds</td>
<td>12</td>
</tr>
<tr>
<td># wounds analyzed</td>
<td>9</td>
</tr>
<tr>
<td># KB103 treated wounds</td>
<td>6</td>
</tr>
<tr>
<td># KB103 treated recurring wounds</td>
<td>4</td>
</tr>
<tr>
<td># KB103 treated chronic wounds</td>
<td>2</td>
</tr>
</tbody>
</table>

### Combined Phase I/II Trial

<table>
<thead>
<tr>
<th>Category</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total # of wounds</td>
<td>16</td>
</tr>
<tr>
<td># wounds analyzed</td>
<td>13</td>
</tr>
<tr>
<td># KB103 treated wounds</td>
<td>8</td>
</tr>
<tr>
<td># KB103 treated recurring wounds</td>
<td>6</td>
</tr>
<tr>
<td># KB103 treated chronic wounds</td>
<td>2</td>
</tr>
</tbody>
</table>

One patient in phase II trial dropped out of the study after 30 days due to an inability to travel resulting in analysis on remaining three patients (9 wounds).

Chronic wounds remain open for greater than or equal to 12 weeks while recurring wounds heal but easily open.
KB103 Phase II Efficacy Summary

- In the phase II study, 5 out of 6 wounds treated with KB103 closed completely (100% wound closure)

- The average time to 100% wound closure on 5 out of 6 wounds was 23.4 days. On recurring wounds, the average time to 100% wound closure was 19 days.

- Duration following complete (100%) wound closure on recurring wounds as measured on Day 90 was therefore 71 days.

- Preliminary results indicate that duration of wound closure at 120-day timepoint was 101 days. We shall provide a further update on final duration of wound closure prior to commencing pivotal trials.

- Molecular correction was established in all 3 patients in phase II trial and correlates to wound healing
In the combined phase I and phase II study, 7 out of 8 wounds treated with KB103 closed completely (100%).

The average time to 100% wound closure on all KB103 treated wounds in combined phase I and phase II study (7 out of 8) was 20.14 days (median 20 days).

In phase I study, the duration of wound closure on two patients following 100% wound closure as of the last follow up was 184 days (6.6 months) and 174 days (6.2 months).

In phase II study, preliminary results indicate that duration of wound closure at 120-day timepoint was 101 days. We shall provide a further update on duration of wound closure prior to commencing pivotal trials, once results are finalized.
For the treatment of Autosomal Recessive Congenital Ichthyosis associated with TGM1

* Orphan Drug Designation in US;
Rare Pediatric Disease Designation in US;
Eligible for Priority Review Voucher.
ARCI Associated With TGM1

Autosomal Recessive Congenital Ichthyosis (ARCI) associated with TGM1

A condition characterized by thick dry scaly skin, increased trans-epidermal water loss (TEWL), risk for dehydration, sepsis, skin malignancies, etc.

Caused by a mutation of TGM1 gene required for epidermal barrier formation

Prevalence1-8

<table>
<thead>
<tr>
<th>Region</th>
<th>Prevalence</th>
<th>US</th>
<th>EU</th>
<th>ROW</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1,791</td>
<td>3,168</td>
<td>18,018</td>
</tr>
</tbody>
</table>

New Cases/Year

<table>
<thead>
<tr>
<th>Region</th>
<th>New Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>US</td>
<td>22</td>
</tr>
<tr>
<td>EU</td>
<td>22</td>
</tr>
<tr>
<td>ROW</td>
<td>348</td>
</tr>
</tbody>
</table>

Current Standard of Care

There are no approved treatments for ARCI associated with TGM1
Existing approaches limited to time-consuming palliative treatments

References:
2. Dreyfus et al. Orphanet J Rare Dis. 2014 Jan 6;9:1;
6. Orphanet;
7. Foundation for Ichthyosis & Related Skin Types (FIRST);
8. National Organization for Rare Disorders (NORD).
ARCI Associated With TGM1

Current standard of care vs. Krystal’s approach

Does not address underlying genetic deficiency

Severe adverse effects
- Soft tissue calcification of the joints, e.g., around the spine
- Increased blood triglycerides and cholesterol, potentially inducing or exacerbating atherosclerosis
- Acute and chronic toxicities associated with long-term exposure

Oral retinoids

Particularly ill-suited for certain patient segments
- **Children**: doctors delay retinoid therapy as long as possible due to the bone growth defects (including premature termination of bone elongation) induced by retinoids
- **Women of childbearing age**: retinoids are teratogens (cause fetal abnormalities, miscarriages and severe birth defects) with potentially long half-lives; must be avoided by pregnant women or women who intend on becoming pregnant

Corrects molecular defects of disease
- KB105 encodes and expresses multiple functional copies of TGM1
- Direct delivery of TGM1 to appropriate skin substrata

KB105

Improved safety
- Avoids severe adverse events associated with long-term retinoid therapy

No systemic exposure to the drug product

Engineered for topical application
- Can be administered frequently

Suitable for all patient populations
- Including children and women of childbearing age
KB105 Mechanism of Action

1. KB105 enters permeabilized skin and transduces keratinocytes (native TGM1-producing cells).
2. KB105 is transported into the nucleus of transduced cells and the vector genome is deposited (episomally).
3. TGM1 transcripts are generated, which allows the cell to produce functional TGM1 protein that localizes to the cell membrane.
4. TGM1 crosslinks target proteins (e.g., filaggrin (FLG), involucrin (IVL), small proline-rich proteins (SPRs)) to aid in the formation of the cornified cell envelope.
5. This layer, known as the stratum corneum, acts as a mechanical barrier to protect against transepidermal water loss (TEWL) and entry of infectious agents.

Untreated ARCI Patient Skin  →  KB105-treated Skin
KB105 Preclinical: Immortalized TGM1-Deficient ARCI Keratinocytes

Robust TGM1 expression detected at the transcript and protein levels, with no obvious dose-dependent toxicity.
KB105 Preclinical: Primary TGM1-Deficient ARCI Keratinocytes

KB105 efficiently transduces primary TGM1-deficient ARCI keratinocytes
Dose-dependent increase in functional TGM1 detected in KB105 infected primary TGM1-deficient ARCI keratinocytes

TGM1 activity determined using a fluorescent synthetic peptide reporter assay measuring TGM1-mediated glutamine conjugation
Properly localized dose-dependent human TGM1 detected 48 hours after topical KB105 application in mice.

Loricrin is both a substrate for TGM1 and serves as a marker for the stratum granulosum/spinosum - indicates that TGM1 colocalizes with at least one native substrate and is expressed in the correct layer of skin.
KB105 Preclinical: *In Vivo* Repeated Topical Administration

Robust expression from single administration (D1 only) and repeat administration (D1 followed by D3 or D12)

Repeat dosing was well-tolerated – no morphological changes observed after repeat dosing
KB105 Preclinical: *In Vivo* Short-Term Pharmacokinetics (48 hours)

DNA and RNA levels peak around 8 hours, remain stable through the 48-hour timepoint.
KB105 was administered once weekly via topical application to the dorsal skin of immunocompetent mice. Five total weekly doses, followed by 4-week recovery (to assess the reversibility or persistence of any effects).

**Toxicity parameters**: mortality, clinical observations, body weights, food consumption, dermal observations, and clinical and anatomic pathology.

**Histological assessment**: application/dose site, sternum with bone, marrow, brain, epididymis, heart, kidneys, lesions, liver, lungs, axillary lymph node, inguinal lymph node, ovaries, oviducts, prostate, spleen, testes, thymus, uterus with cervix).

**Biodistribution (qPCR)**: Dose site, blood, femur with bone marrow, brain, heart, kidney, liver, lung, lymph nodes, spleen, ovaries and testes

<table>
<thead>
<tr>
<th>Group:</th>
<th>Number of Animals*:</th>
<th>Topical Dosing Regimen:</th>
<th>Termination:</th>
<th>Dose (PFU/day):</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>6 Males 6 Females</td>
<td>Days 1, 8, 15, 22, 29</td>
<td>Day 3 (Interim) Day 30 (Terminal) Day 63 (Recovery)</td>
<td>-</td>
</tr>
<tr>
<td>KB105</td>
<td></td>
<td></td>
<td></td>
<td>1.07x10^9</td>
</tr>
</tbody>
</table>

*For each termination time point.

**Group: Number of Animals**: Males, Females

**Termination**: Dose (PFU/day): Control, KB105
Repeat application of high dose topical KB105 was well tolerated and localized.

Five weekly administrations of $1.07 \times 10^9$ pfu/day KB105 via topical application to the dorsal skin of male and female mice were well tolerated.

- **NOAEL dose**: $1.07 \times 10^9$ pfu/day
- No KB105-related mortality, clinical observations, body weight or food consumption changes, macroscopic findings, or effects on organ weight parameters were noted.
- All animals survived until their scheduled necropsy.
- Minor increase in incidence of edema at the dose site in males between Days 16 and 30 of the dosing phase and persistence of erythema at the dose site during the recovery phase in females were not considered adverse based on severity.
- High copy levels of KB105 detected at (and limited to) the dose site of all KB105-treated animals
  - On Day 3, $3.6 \times 10^7$ copies/ug tissue
  - On Day 30 $1.3 \times 10^7$ copies tissue – demonstrates successful repeat dosing
KB105 Conclusions

- *In vitro* and *in vivo* proof-of-concept and safety established for KB105
  - KB105 efficiently transduces patient keratinocytes to express functional human TGM1.
  - Topical KB105 efficiently transduces permeabilized skin and expresses human TGM1 *in vivo* in mice, in a dose-dependent manner.
  - KB105-expressed TGM1 colocalizes with native TGM1 substrates, indicating delivery to the appropriate epidermal layer.
  - Biodistribution and toxicity data indicate that KB105 can be safely and repeatedly administered to the skin at high doses without systemic vector exposure.

- KB105’s robust production of TGM1 *in vitro* and *in vivo* supports its use in ARCI patients
KB105 Timeline to Clinical Readout

Phase I/II clinical trial to begin 2H 2019

2018

1H
• Preclinical studies initiated
• Pre-IND meeting

2H
• GLP safety and toxicity studies
• Orphan Indication Granted

2019

1H
• GMP manufacturing
• IND filing

2H
• Initiate phase I/II clinical study
• Interim clinical readout

2020

1H
• Initiate pivotal clinical study
KB104

For the treatment of Netherton Syndrome
Netherton Syndrome (NS)

Netherton Syndrome
A life-threatening condition characterized by red, inflammatory scaling on the face, shoulders, and back, as well as short, brittle, and broken “bamboo hair”.

Caused by loss-of-function mutations in the SPINK5 gene that is otherwise required for maintaining integrity and protective barrier function of the skin by regulating desquamation-involved proteases.

Current Standard of Care
There are no approved treatments for Netherton Syndrome
Existing approaches are limited to palliative treatments, including topical moisturizers, repair formulas, and steroids.

Prevalence

1. Orphanet Report Series, Rare Diseases collection, June 2018, Number 1;
3. Furio and Hovnanian. Biol Chem. 2014 395(9) 945-58; and

New Cases/Year

1. Orphanet Report Series, Rare Diseases collection, June 2018, Number 1;
3. Furio and Hovnanian. Biol Chem. 2014 395(9) 945-58; and
Established process conducted at Krystal’s end-to-end GMP facility

- Maintains control of IP/trade secrets relating to manufacturing process
- Adheres to internal process and production schedules, avoiding use of high demand gene therapy CMOs

Upstream Production Process

- Proprietary engineered vectors and complementary/supporting cell lines developed in-house are used in established methods for production of consistent batches
- Scalable from clinical phase to commercial

Downstream Purification Process

- Work conducted in an aseptic closed system process
- Process accommodates ever-expanding vector pipeline with minimal redevelopment effort between product candidates
- Compliant to global regulatory requirements
Currently working with Krystal on KB103 and KB105

**Dr. Peter Marinkovich**  
Department of Dermatology  
*Stanford University*  
Serving as lead clinical investigator in KB103 phase I/II trial

**Dr. Andrew South**  
Department of Dermatology,  
*Thomas Jefferson University*

**Dr. Joyce Teng**  
MD, PhD  
Clinical Professor, Dermatology Clinical Professor, Pediatrics  
*Stanford University*

**Dr. Keith Choate**  
MD, PhD  
Professor of Dermatology, Genetics, Pathology  
*Yale University School of Medicine*

**Dr. Amy Paller**  
MD  
Chair of Dermatology  
*Northwestern University*

**Dr. Alain Hovnanian**  
MD, PhD  
Director, INSERM Department on Genetic Skin Diseases  
*University Paris Descartes – Sorbonne Paris Cité*
Beyond Severe Monogenic Skin Diseases

Application of fully-integrated vector platform to treat aesthetic defects

Youthful Skin
- Smooth appearance
- Abundance of collagen

Aged Skin
- Lined and wrinkled appearance
- Lack of collagen

The characteristic features of skin aging are largely due to aberrant collagen homeostasis, resulting in a net collagen deficiency.

Vector Platform
- Modify platform via insertion of human collagen gene(s)

KB301
- Formulate into an intradermal solution

Off-the-shelf, Non-Invasive Modified HSV-1 Therapy

Fromowitz, J. "Update on Aging Skin"; Florida Society of Dermatology
Beyond Severe Monogenic Skin Diseases

Application of fully-integrated vector platform to treat complex, chronic skin conditions

**Chronic skin conditions**

KB500 series (Antibodies) for Chronic Skin Diseases (Atopic Dermatitis, Psoriasis, etc.)

**Vector Platform**

- Modify platform via insertion of anti-inflammatory antibody effectors

**KB500 series**

- Formulate into a topical gel

**Off-the-shelf, Non-Invasive Modified HSV-1 Therapy**

- Full-length Abs
- Ab Fragments (e.g., scFv-Fcs)

**Topical**