“Re-balancing the Skin Microbiome and Innate Immune System to Improve Skin Health”
MatriSys Bioscience

• Founded in 2015 to commercialize skin microbiome discoveries
• Focused on commercial product development to Proof-of-concept/Phase 2
• Laboratory in La Jolla, California
  – Microbial fermentation
  – Product formulation and development
  – Clinical development to Proof-of-concept/Phase 2
• Exclusive license to UCSD IP covering MSB-01 (S. hominis A9)
Value Proposition
MSB-01 microbiome dermatitis Program

MatriSys Bio

For Pharma partner
• Novel therapeutic approach
• IP protected
• Large chronic market
• Demonstrated killing of bad bacteria w low risk of toxicity and no toxicity seen in Phase I and 2 trials to date

For the Dermatitis Patient
• Current products less than ideal
• Topical, non-systemic application
• Non steroid approach (skin thinning/photo sensitivity)
• Great candidate for chronic Rx; we have seen no side effects in Phase I and 2 trials
Founding Team

Mark S. Wilson, CEO
- VP BD Halozyme
- 28+ years Bio experience
- Co-founder
- UC Berkeley Eng.
  UCLA MBA

Rich Gallo, MD, PhD,
Head of Scientific Advisory Board
- Chair, UCSD Dermatology
- Technology inventor
- Co-founder

Gilbert Keller, PhD,
VP Preclinical Sciences
- Genentech, Halozyme
- Co-founder
- U.Geneva/Einst.
- UCSD

Lou Bookbinder, PhD,
VP Product Development
- Halozyme, Intrexon
- Co-founder
- UCLA, UCSD
MatriSys Bio™ Microbiome Solution
Rebalancing the natural skin microbiome

S. hominis A9 intervention*

helpful bacteria peptides

bAMPs

“Good bugs”

Skin cells

Feedback

hAMPs

Innate immune system

PATHOGENS:
- Staph infection
- Super bugs/MRSA
- Acne
- Strep A & B
- Yeasts

= Bacteria
= Fungi
= Viruses
= bacterial Antimicrobial Peptides
= human Antimicrobial Peptides

* A natural beneficial microbe – not genetically engineered
<table>
<thead>
<tr>
<th>Product/Indication</th>
<th>Preclinical</th>
<th>Phase I</th>
<th>Phase II</th>
<th>Phase III</th>
<th>Registration</th>
<th>Approval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Microbiome – atopic dermatitis/eczema (Sh-A9)</td>
<td>MSB-01</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Microbiome – Bacterial infections</td>
<td>MSB-02</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Microbiome - Acne</td>
<td>MSB-04</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Microbiome – skin cancer/actinic keratosis</td>
<td>MSB-05</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Rosacea</td>
<td>MSB-03</td>
<td></td>
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</tr>
<tr>
<td>Netherton Syndrome*</td>
<td>MSB-03</td>
<td></td>
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</tr>
</tbody>
</table>

MSB-01 is an anhydrous topical formulation of *Staphylococcus hominis* strain A9 (Sh-A9)

MSB-05 is a topical formulation of *S. epidermidis* M034/M035 also referred to as the "Firmocidin" strain

* Expected to qualify as Orphan drug; Orphan application filed February 2017

NEW IP
## Cosmetic Extracts

<table>
<thead>
<tr>
<th>Active Ingredient</th>
<th>In vitro efficacy evaluation</th>
<th>In vitro safety evaluation</th>
<th>Formulation</th>
<th>Manufacturing process defined</th>
<th>Cosmetic clinical evaluation</th>
<th>Register active ingredient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Microbiome – Lantibiotic extract (eczema)</td>
<td>+ Lantibiotics</td>
<td>“Diseased skin”</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Microbiome – Lipoteichoic acid, or “LTA”,&amp; AIPs (inflammation, anti-toxin)</td>
<td>LTA/AIPs</td>
<td>“Healthy skin”</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Microbiome – Firmocidin (photo aging/UV)</td>
<td>Firmocidin</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

### Extracts from select proprietary beneficial bacterial strains
- +/- different lantibiotic peptides -
- AIPs (auto-inducing peptides)
- LTA (Lipoteichoic acid)  

}\ “Healthy skin”
Which is the largest interface between the microbiome and us?

**Skin**

؟️۸۱،۰۰۰م؟ \( m^2 \)

**GI tract**

۳۰ \( m^2 \)

Richard Gallo MD PhD
Potential options in Microbiome Therapeutics

Three approaches:

• Transfer “healthy” microbiome
  Pros: Applies large scale genomic information
  Cons: MOA unknown, many inactive microbes

• Engineer Microbes to express active
  Pros: Introduces known active compound
  Cons: Low expression, FDA risks unknown

• Apply commensal Biotherapeutic
  Pros: Known active, scientific rationale, clinical rationale, FDA
  Cons: Must have good IP counsel for lasting advantage
MatriSys’ technology is to re-balance the natural skin microbiome

- Healthy human skin produces beneficial peptides that protect your skin. This is the “innate immune system”
- MatriSys’ good bacteria also produce beneficial peptides that together protect your skin
  - Broad spectrum antibiotics kill these beneficial bacteria; a better treatment is needed
  - Our beneficial bacteria are proprietary (UCSD/MatriSys Bio) and only kill bad bacteria
  - Naturally potent microbes found on healthy skin, and not genetically engineered
The MatriSys Platform
Functional Screening for Microbiome Therapeutics

• *Staph aureus* is our first target – we have 12 proprietary strains of *S. hominis* and *S. epidermidis* that kill *S. aureus*.
• We have more than **10,000 additional clinical isolates** to explore (beneficial bacteria)
• We select pathogens (like MRSA), develop screening assays, and screen for harmless beneficial bacteria that kill pathogens, creating a mutual benefit
  • “Mutualism” is where both organisms benefit (man and the beneficial microbe)
Taxonomy – Strain is the key

Naming and classification of groups of organisms

**Example:**

*Staphylococcus*

*Staphylococcus hominis*

*Staphylococcus hominis hominis (OTU)*

*Staphylococcus hominis strain A9*
AMP Gene Systems Identified in Genomes of our functionally identified Strains
Hogocidin lantibiotic peptides
Hogo from Japanese (保護) – “protection”

Lanthionine is a nonproteinogenic amino acid with the chemical formula (HOOC-CH(NH₂)-CH₂-S-CH₂-CH(NH₂)-COOH). It is a thioether dimer of cystine, composed of two alanine residues that are crosslinked on their β-carbon via a sulfur atom. Despite its name, lanthionine does not contain the element lanthanum.
Atopic Dermatitis Program
MSB-01 (S. hominis strain A9)
MSB-01 (S. hominis A9) Target Product Profile

- Topical, live and naturally-derived microbiome therapeutic
- NOT autologous; this is a Universal Strain for all patients
- Potential for infrequent dosing at 1-2 times/week
- 4 Known mechanism of action:
  - Anti-inflammatory (lipoteichoic acid, decreases IL-4, IL-13)
  - Reduce S. aureus colonization (lantibiotic peptides)
  - Blocks S. aureus toxin production (auto-inducing peptides)
  - Bacterial AMPs synergize with human AMPs
- Appropriate for mild, moderate, and severe atopic dermatitis
- No immune suppression
Scientific Advisory Board

Richard Gallo, MD, PhD
- Chair, UCSD Dermatology
- Technology inventor
- Co-founder

Lawrence F. Eichenfield, MD
- Chief of Pediatric and Adolescent Dermatology at Rady Children’s Hospital
- Vice Chair of the Dept. of Dermatology
- Professor of Dermatology and Pediatrics (UCSD)

Peter M. Elias, MD
- Professor Emeritus, Dept. of Dermatology, UCSF
- Staff physician at the Veterans Affairs Medical Center

Amy S. Paller, MD
- Chair, Walter J. Hamlin Professor of Dermatology
- Director of the Northwestern University Skin Disease Research Center
Atopic dermatitis microbiome progression hypothesis

Cited in his blog - Francis Collins MD PhD, Director of the NIH

Proposed relationship among shifts in skin microbial diversity, the proportion of Staphylococcus, and disease severity

Key Concept #1:
Overwhelming data now shows essential role of the microbiome in AD

- Atopic Dermatitis:
  - Unmet therapeutic needs
  - *S. aureus* promotes disease severity
  - *S. aureus* hinders efficacy of other drugs
  - Past antimicrobial therapy did not kill *S. aureus* colonization
  - No treatments for sustained control of *S. aureus* in chronic patients available
  - Deficiency in natural antibiotics from host and microbiome
Concept #1 is based on solid, peer reviewed, science from many basic science and clinical groups

*S. aureus can directly drive AD-like dermatitis in mice:*
Many Refs now: Reviewed in Williams M. *et al.* Cell Host and Microbe. 2017

*Clinical severity is driven by S. aureus*
Many Refs now: See recent Brandwein M. *et al.* B. J Dermatol. 2018

*AD is lacking in innate anti-S. aureus activity:*
From Host: Ong *et al.* N Engl J Med 2002
From Microbiome: Nakatsuji *et al.* Sci Trans Med 2017
Key Concept #2:

*S. hominis* strain A9 (MSB-01) is ideal AD therapeutic

- MSB-01 *is* Safe: Isolated from healthy human skin
  - Missing from AD skin: Replacement therapy
  - Produces 3 validated actives; synergy with human AMPs
    1. Anti-inflammatory TLR inhibitor
    2. Anti-*S. aureus*- Selectively kills *S. aureus* and enables bacterial diversity
    3. Anti-*S. aureus* toxin- Inhibits pathogenic toxins
    4. Bacterial AMPs synergize with human AMPs
- Strong IP, strong preclinical, formulation stable, cell bank
Concept #2 is based on breakthrough discoveries from UCSD

*S. epidermidis* anti-inflammatory activity:

**CoNS kills S. aureus:**

**CoNS detoxifies S. aureus:**
Poster 636
MSB-01 is potent anti-Th2 and resolves AD in mice

SA= *S. aureus*,
SH= MSB-01 (*S. hominis* A9)

High throughput screen discovered anti-\textit{S. aureus} active in specific CoNS strains

This is the second activity that is \textbf{UNIQUE} to MSB-01

MSB-01 also produces a THIRD active molecule. This is a unique and potent anti-toxin against *S. aureus*.

This CoNS strain produces anti-toxin similar to MSB-01 but cannot kill *S. aureus*.

This shows **THIRD active** in MSB-01 can be effective alone!

New data presented IID 2018 Favorably reviewed STM:
Bacterial AMPs synergize with Human AMPs

**Fig. 6.** Detection of 

**Fig. 7.**

**Table 1.**

**Figure Legends:**

1. **Fig. 6.** Detection of *Sh*-lantibiotics in human skin swabs. Each point represents analysis of one individual. Bar, mean.

2. **Fig. 7.** Concentration of *Sh*-lantibiotics and human AMPs in skin swabs. Each point represents analysis of one individual. Bar, mean.

**Results:**

- *Sh*-lantibiotics are commonly found on healthy human skin and synergize with a host AMP.
- Bacterial AMPs synergize with human AMPs to exhibit bactericidal activity.
- CoNS colonies were screened. Only a single strain with antimicrobial activity was isolated from the three subjects.
- In cases where a single strain of CoNS was formulated, a trend toward an increase in bacterial abundance from baseline was seen in both untreated and vehicle-treated groups.
- Furthermore, we examined whether the dysbiosis seen in AD patients is associated with a decrease in the abundance of compatible bacteria.
- The relative abundance of compatible bacteria was significantly decreased in both untreated and vehicle-treated groups.

**Discussion:**

- The dysbiosis seen in AD patients is associated with a decrease in the abundance of compatible bacteria.
- Furthermore, we hypothesized that bacteria that then exacerbate the disorder.
- We further hypothesized that the loss of these protective strains might be functionally important because of a loss of these protective strains.
- We observed that several different bacterial species on the skin community of bacteria on the skin might contribute to the severity of AD.
- Moreover, patients with AD are frequently colonized by bacteria that then exacerbate the disorder.
- We hypothesized that the commensal bacteria residing on normal skin might contribute to the severity of AD.
- Furthermore, we examined whether the dysbiosis seen in AD patients is associated with a decrease in the abundance of compatible bacteria.
- The relative abundance of compatible bacteria was significantly decreased in both untreated and vehicle-treated groups.

**Conclusion:**

- Bacterial AMPs synergize with human AMPs to exhibit bactericidal activity.
- The dysbiosis seen in AD patients is associated with a decrease in the abundance of compatible bacteria.
- We further hypothesized that the loss of these protective strains might be functionally important because of a loss of these protective strains.
- We observed that several different bacterial species on the skin community of bacteria on the skin might contribute to the severity of AD.
- Furthermore, patients with AD are frequently colonized by bacteria that then exacerbate the disorder.
Summary of the pre-clinical data with MSB-01

- This strain survives well on healthy human skin but missing in AD
  (unlike other current proposed microbiome therapies)
- This species is anti-inflammatory (TLR-cross talk mechanism)
- This strain is selective anti- *S. aureus* (lantibiotic promotes survival of other CoNS)
- This strain will inhibit toxin from *S. aureus* (AIP peptide active even if *S. aureus* resistant)

*The live biotherapeutic offers advantage of multiple actives that is not possible with standard “drug” approach*
Great progress in the first 2 human clinical trials to test our microbiome therapeutic approach

- Clinical autologous PoC trials have been running simultaneously with development of MSB-01.

- This alternate approach with self-to-self transplant using biosimilars to MSB-01 was pursued by UCSD for path to FDA IND approval.

- These human clinical trials have been highly successful.
“First in human” skin microbiome transplant

1. Sample from AD subject
2. Screen for active
3. Isolate, expand and formulate
4. Randomize and blind with vehicle
5. Reapply with internal control
Human 1 day trial of strains similar to MSB-01 effective


= Autologous Microbiome Transplant
Second trial (BID X 7 days) was also effective and showed persistence after end of therapy

**Lesional** *S. aureus*

**Nonlesional** *S. aureus*

Off therapy *S. aureus* continues to drop! This suggests less frequent dosing will be effective.

Median of both arms of 5 (Active) or 6 (Vehicle)  
*P* value=two-tailed Mann Whitney U-test

![Graphs showing relative S. aureus CFU over time for lesional and nonlesional samples.](image-url)
Second trial also showed impressive clinical improvement with strains similar to MSB-01

Off therapy continued improvement again!
As predicted: This unique anti-\textit{S. aureus} therapy increased microbial diversity on human skin

This is bacterial transcriptomics:
Demonstrating gene expression by species that are alive on the skin
MatriSys Bio MSB-01 plan to Proof-of-Concept

- NIH Phase 1/2A MSB-01
  - Master & working Cell Banks
  - Engineering batches
  - cGMP Clinical batches
  - cGMP Clinical Supplies
  - MatriSys IND filing

- Phase 2 MSB-01
  - Potential NIH Phase 2 MSB-01

- Commercial formulation

- Value inflection point

Timing depends on funding

- Q3 2017
- Q4 2017
- Q1 2018
- Q2 2018
- Q3 2018
- Q4 2018
- Q1 2019
- Q2 2019
- Q3 2019
- Q3 2019
- Q3 2019
- Q3 2019
- Q3 2019
- Q3 2019
- Q3 2019

Frozen formulation
Skin Care and Dermatology Market

Total Available Market- Skin Care, non Rx
• In 2016, the global skin care market was estimated to be worth $121 billion  

Total Available Market- Rx Dermatology
• Worldwide market for prescription dermatological products reached $21.2 billion in 2015, growing at a rate of 7.9% 

Eczema products (includes Rx and non-Rx) are expected to grow to a value of $3.8 billion by 2018, at a compound annual growth rate (CAGR) of 8.2% 

Eczema Rx Market is $800 million market; 132m AD patients

Sources:
1. Statista.com, January 2016
3. GlobalData research (Eczema products include both Rx and non-Rx products)
# Competitive Landscape – atopic dermatitis

<table>
<thead>
<tr>
<th>Example Company</th>
<th>Valeant</th>
<th>GlaxoSmithKline</th>
<th>Pfizer/Anacor</th>
<th>Regeneron/Sanofi</th>
<th>Johnson &amp; Johnson</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Product Class</strong></td>
<td>Topical calcineurin inhibitors (&quot;TCI&quot;)</td>
<td>Topical steroids</td>
<td>PDE 4B inhibitor</td>
<td>Antibodies (ex. Dupilumab)</td>
<td>Barrier Creams (Rx/OTC)</td>
</tr>
<tr>
<td><strong>Mechanism</strong></td>
<td>-Blocks the formation of inflammatory molecules</td>
<td>-Anti-inflammatory and suppress immune system</td>
<td>-Inhibit phosphodiesterase 4B enzyme; Mild to Moderate only</td>
<td>--Block proteins that drive inflammation</td>
<td></td>
</tr>
<tr>
<td><strong>Effectiveness</strong></td>
<td>-Second Line Therapy -No better than steroids -Lose potency with time</td>
<td>-First Line Therapy -Decrease inflammation</td>
<td>~ 30% ISGA score of Clear (0) or Almost Clear (1) with a 2-grade improvement</td>
<td>-Significant disease burden decrease in patients within 12 weeks</td>
<td>Decrease need for steroids or immune suppressants</td>
</tr>
<tr>
<td><strong>Side Effects</strong></td>
<td>-Burning on application, skin infections</td>
<td>-Skin thinning, photosensitivity, acne, rosacea</td>
<td>Hypersensitivity Burning upon application</td>
<td>-Injection site reactions, inflammation of the nose &amp; throat, conjunctivitis</td>
<td>-Burning and stinging</td>
</tr>
<tr>
<td><strong>Safety Concerns</strong></td>
<td>-Black Box Warning cancer risk, suppresses immune</td>
<td>-Potential birth defects with very potent topical corticosteroids</td>
<td>Immune suppression</td>
<td>-Systemically suppresses the immune system</td>
<td>-Many safe for children -Allergies to excipients</td>
</tr>
</tbody>
</table>

*Dupixent® (Dupilumab) - for Atopic Dermatitis Inadequately Responsive to Topical Therapy*
Survey of practicing clinical dermatologists shows microbiome approach is very desirable

- Survey conducted at 2018 Society for Pediatric Dermatology
Survey of practicing clinical dermatologists shows microbiome approach is very desirable

<table>
<thead>
<tr>
<th>Question</th>
<th>Yes (%)</th>
<th>No (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Do you believe topical products can be as effective as a systemic?</td>
<td>66</td>
<td>34</td>
</tr>
<tr>
<td>For adult patients, would you prefer a microbiome-based therapy as an alternative to: a high potency topical steroid?</td>
<td>88</td>
<td>12</td>
</tr>
<tr>
<td>For adult patients, would you prefer a microbiome-based therapy as an alternative to: a systemic immunosuppressant?</td>
<td>90</td>
<td>10</td>
</tr>
<tr>
<td>Do you believe patients might be more compliant with a topical regimen if it was naturally derived microbiome based therapy?</td>
<td>84</td>
<td>16</td>
</tr>
<tr>
<td>Do you believe that a once-a-week dosing would promote compliance over daily dosing?</td>
<td>90</td>
<td>10</td>
</tr>
</tbody>
</table>

• Survey conducted at 2018 Society for Pediatric Dermatology
Average Yearly Cost of Therapy for AD

MSB-01 (S. hominis A9) Target Product Profile
- Topical, live and naturally-derived microbiome therapeutic
- Potential for infrequent dosing at 1-2 times/week
- Known mechanism of action to reduce *S. aureus* colonization
- Appropriate for mild, moderate, and severe atopic dermatitis
- No immune suppression
Strain Culturing

S. aureus Antimicrobial Activity

Living cells in culture are active

Freeze Dried Powder in Oil Formulation is active after 6 months

Freeze Dried Powder is active after 6 months

Living cells in culture are active

Zone of *S. aureus* Growth Inhibition

Sterile filtered Active Conditioned Medium is active

Product Formulations

Oil Ointment Lotion Anhydrous

S. aureus Antimicrobial Activity
Just a couple questions…

There is a large body of scientific literature implicating Staph. aureus as the cause of the inflammatory reaction in AD.

If Staph. aureus is the cause, why can’t I use bleach baths and broad spectrum antibiotics?

Hmm. And clinical studies don’t show improvements with bleach baths or broad spectrum antibiotics… Why ???
Infection or Colonization?

**Infection:** germs are in or on the body and make you sick (fever, pus, WBCs)

**Colonization:** germs in or on the body, but do not make you sick

- *S. aureus* was found to colonize the skin of 90% of patients with AD in 1974
- *S. aureus* has been proven to exacerbate disease state through several immune-mediated mechanisms, thereby leading to inflammation and sensitization
- Increased *S. aureus* abundance precedes flares in AD patients
- *S. aureus* can be an infection or colonizer, MRSA too
Failure of antibiotics therapy in Atopic Dermatitis is misleading

Oral and Topical Antibiotics for Clinically Infected Eczema in Children: A Pragmatic Randomized Controlled Trial in Ambulatory Care

Nick A. Francis, MD, PhD1⇑, Matthew J. Ridd, PhD2, Emma Thomas-Jones, PhD3, Christopher C. Butler, FRCPG4, Kerenza Hood, PhD3, Victoria Shepherd, MA3, Charis A. Marwick, PhD5, Chao Huang, PhD3, Mirella Longo, PhD6, Mandy Wootton, PhD7 and Frank Sullivan, FRSE8 the CREAM Trial Management Group

CONCLUSIONS We found rapid resolution in response to topical steroid and emollient treatment and ruled out a clinically meaningful benefit from the addition of either oral or topical antibiotics. Children seen in ambulatory care with mild clinically infected eczema do not need treatment with antibiotics.

7 day treatment with topical 2% fusidic acid TID , or floxacilllin QID
Cutibacterium acnes (P. acnes) makes biofilm

Biofilm makes bacteria less sensitive to antibiotics
Some Staph. Aureus strains make biofilms

Legend:
- Staphylococcaceae
- Firmicutes
- Actinobacteria
- Proteobacteria
- Bacteroidetes

A: Normal

B: Atopic Dermatitis Flare

npj Biofilms and Microbiomes (2016)2:3 ; doi:10.1038/s41522-016-0004-z
Gallo’s Bleach Bath Experiment

*S. Aureus* in PBS

*Immerse for 15 minutes*  

Infection is not Colonization!

*Bleach- 0.005%, 0.01%*

*S. Aureus* in agar

*Bleach- 0.005%, 0.01%*

*S. Aureus* on pigskin

*Bleach- 0.005%, 0.01%*
A commensal strain of *Staphylococcus epidermidis* protects against skin neoplasia


Aka “Firmocidin”
6-HAP exerts selective antiproliferative activity against cancer cell lines

6-N-hydroxyaminopurine is a nucleobase analog that competes with adenine to inhibit DNA synthesis

- F5178 lymphoma
- R16F10 melanoma
- Yac-1 lymphoma

- Pam212 murine cutaneous squamous cell carcinoma
- NHEK normal Human Epidermal Keratinocytes
Systemic administration of 6-HAP suppresses melanoma growth in mice

No apparent toxicity by
• weight
• blood count
• liver function

60% tumor size reduction of B16F10 melanoma cells implanted in mice
• repeated IV 6-HAP administration

Tumors at day 9 and day 13 in mice treated with 6-HAP
Skin colonization by *S. epidermidis* strain producing 6-HAP protects from UV induced neoplasia in mice

Effects of colonization by *S. epidermidis* MO34 on tumor incidence (A) and number (B) in hairless mice treated with DMBA (tumor initiator) and UV irradiation
Summary

• Novel therapy treating an unmet need – atopic dermatitis
• MSB-01 is standalone or complementary product
• True platform technology (10,000 microbial library)
• Earlier autologous clinical trials positive
  • Phase 1 autologous 99.1% reduction in *S. aureus*
  • Phase 2 autologous interim read-out positive (EASI)
• Universal product development underway
  • Phase 1/2a ongoing
  • Product stable at room temperature 1 ½ years
• Atopic dermatitis is a very large growing market
• MatriSys’ pipeline is growing
First Line of Defense

Therapeutics at the interface of the microbiome revolution and the innate immune system

Contact: Mark S. Wilson, President and CEO
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858.752.9003