therapeutically effective amount of a purified viable gram negative bacteria and a pharmaceutically acceptable carrier, wherein a) a lysate and/or component of the gram negative bacteria inhibits growth of *S. aureus* in an *in vitro* assay; b) the gram negative bacteria stimulates human keratinocytes; c) the gram negative bacteria induces cytokine expression from human cells; and d) the gram negative bacteria is non-pathogenic when administered to the skin of the subject. The pharmaceutical compositions are formulated for topical administration.

Methods of treating a topical dermatitis using these pharmaceutical compositions are also disclosed.

In one aspect, there is provided a use of a pharmaceutical composition comprising:

a therapeutically effective amount of at least one strain of *Roseomonas mucosa*, wherein the at least one strain of *Roseomonas mucosa* is purified, viable and isolated from skin of a donor subject; and

a pharmaceutically acceptable carrier in the manufacture of a medicament for treating atopic dermatitis in a subject in need thereof.

In one aspect, there is provided a bandage comprising the medicament as described herein.

In one aspect, there is provided a spray bottle comprising medicament as described herein.

In one aspect, there is provided kit for treating atopic dermatitis, comprising:

a) a first container comprising a therapeutically effective amount of at least one strain of *Roseomonas mucosa*, wherein the at least one strain of *Roseomonas mucosa* is purified, viable and isolated from skin of a donor subject;

b) a second container comprising a pharmaceutically acceptable carrier; and

c) instructions for applying the therapeutically effective amount of the purified viable gram-negative bacteria in the pharmaceutically acceptable carrier topically to the skin.

The foregoing and other features and advantages of the invention will become more apparent from the following detailed description of several embodiments which proceeds with reference to the accompanying figures.

BRIEF DESCRIPTION OF THE FIGURES

FIGs. 1A-1D: CGN isolates differ in presence and *S. aureus* inhibition between healthy volunteers and patients with AD. (A) Percentage of individuals with CGN isolate yield from HV (n=26) and AD (n=17) subjects. Individuals with multiple CGN isolates were counted per isolate for >100% total in HV; see Table 1 for details. (B) Eight strains of *S. aureus* isolated

from HV and AD patients were grown in the presence of either CGN supernatant or control media. Each data point represents the effect on *S. aureus* growth of supernatant from one CGN isolate compared to media control (HV isolates = 9, AD isolates = 7); shapes represent sourcing of *S. aureus* as either the participant's autologous *S. aureus*, or one *S. aureus* isolate from HV or AD patients. Data points with a * symbol represent one HV- and one AD-derived CGN isolate selected for subsequent human challenge and mouse model experiments. Data is alternatively presented by CGN species, see FIG. 4. (C) Healthy mouse ears were co-inoculated with *S. aureus* (strain SAAS9) and *R. mucosa* (*Rm*) from HV or AD patients, or *P. aeruginosa* from a HV for 10 days. Day 12 ears were homogenized and plated by serial dilution for CFU. Percent change in growth versus diluent (no CGN added) control is shown. (D) CGN CFU yield taken from same mice as panel C. Data shown are a combination of three or more independent experiments (B) or representative of two independent experiments (C-D) and displayed as mean + sem. SA = *S. aureus*, HV = Healthy Volunteer, AD = Atopic Dermatitis, CGN = Culturable gram negative, Rm = *Roseomonas mucosa*, Pa = *Pseudomonas aeruginosa*. Significance determined by Student's t test (B) or ANOVA with Bonferroni's correction (C-D).

FIGs. 2A-2D: CGN from HV enhances innate immunity and barrier function. (A) Cytokine analysis for in vivo human blister challenge on healthy controls showing cytokine

responses to a representative strain of *R. mucosa* from a healthy control versus one isolated from a patient with AD as compared to the saline control well (dotted line), N= 7. (B) Paired analysis of data presented in panel A showing the cytokine production in the blister well exposed to the HV sourced *R. mucosa* less the cytokine production in the blister well exposed to the AD sourced *R. mucosa* in the same human subject. (C) Mouse ears were inoculated daily for three days with 1e7 CFU *R. mucosa* from either HV or AD. Day 5 mRNA abundance for IL-1β and filaggrin standardized to GAPDH and compared to untreated mice is shown, N=4-5 mice per group. (D) Mouse backs were shaved and hair chemically removed on day 0. TEWL was then measured after daily application of 1e7 CFU *R. mucosa* from either HV or AD, N=4-5 mice per group. Data shown are a combination of 7 independent experiments (A-B) or representative of two or more independent experiments (C-D) and displayed as mean + sem (C-D) or mean and individual participants (A-B). HV = Healthy Volunteer, AD = Atopic Dermatitis, CGN = Culturable gram negative, Rm = *Roseomonas mucosa*, FLG = filaggrin, IL- = Interleukin, TEWL = Transepidermal Water Loss. Significance from diluent control (or as indicated) determined by ANOVA with Bonferroni's correction.

FIGs 3A-3D: CGN from HV improves outcomes in the MC903 mouse model of AD-like dermatitis. (A-B) Both ears of mice were inoculated daily with *R. mucosa* (*Rm*) from a HV or an AD patient, or *P. aeruginosa* (*Pa*) from a HV, or SAAS9 strain of *S. aureus* for 2 days prior to MC903 application. Then bacteria were co-applied with MC903 daily for 13 days. Day 14 ear thickness (A) for each ear and serum total IgE levels (B) are shown; N= 4-8 mice per group. (C-D) Mice were treated with MC903 for 14 days to induce AD-like dermatitis. Starting on day 13, mice were treated daily for 3 day with 1e6 CFU of live *R. mucosa* from a HV or AD patient (HVCGN and ADCGN), 1e6 CFU of killed *R. mucosa* from the same HV re-suspended in the supernatant from 3e6 CFU of autologous *R. mucosa* (Dead mix), or supernatant from 1e7 CFU of the HV *R. mucosa* (Sup). Day 21 visual redness (D) and ear thickness (C) are shown; N= 3-5 mice per group. Data shown are representative of three independent experiments and displayed as mean + sem. Significance determined by ANOVA.

FIG. 4: Species analysis of data from Fig. 1B. Eight strains of *S. aureus* isolated from HV and AD patients were grown in the presence of either CGN supernatant or control media. Each data point represents the effect on *S. aureus* growth of supernatant from one CGN isolate compared to media control (HV isolates = 9, AD isolates = 7). Significance between R. mucosa isolates determined by Student t-test.

FIGs. 5A-5C: Suction Blister Protocol. (A) Image of 3D printed blister induction device. (B) Blister results 2 hours after suction. (C) Challenge chamber placed over denuded blister areas,

We claim:

1. Use of a pharmaceutical composition comprising:

   a therapeutically effective amount of at least one strain of *Roseomonas mucosa*,

   wherein the at least one strain of *Roseomonas mucosa* is purified, viable and isolated from skin of a donor subject; and

   a pharmaceutically acceptable carrier in the manufacture of a medicament for treating atopic dermatitis in a subject in need thereof.

2. The use of claim 1, wherein the donor subject does not have atopic dermatitis.

3. The use of claim 1, wherein the medicament is formulated in a topical dosage form selected from the group consisting of a cream, gel, salve, foam, ointment, liquid, and a lotion.

4. The use of claim 1, wherein the at least one strain of *Roseomonas mucosa* is present in an amount of 10^4 to 10^{12} colony forming units.

5. The use of claim 1, wherein the at least one strain of *Roseomonas mucosa* is present in an amount sufficient for a reduction in *Staphylococcus aureus* in the subject.

6. The use of claim 1, wherein the at least one strain of *Roseomonas mucosa* comprises up to three strains of *Roseomonas mucosa*.

7. The use of claim 1, wherein the at least one strain of *Roseomonas mucosa* comprises up to five strains of *Roseomonas mucosa*.

8. The use of claim 1, wherein the pharmaceutically acceptable carrier comprises sucrose.

9. A bandage comprising the medicament of any one of claims 1-8.

10. A spray bottle comprising medicament of any one of claims 1-8.

11. A kit for treating atopic dermatitis, comprising:

    a) a first container comprising a therapeutically effective amount of at least one strain of *Roseomonas mucosa*, wherein the at least one strain of *Roseomonas mucosa* is purified, viable and isolated from skin of a donor subject;

    b) a second container comprising a pharmaceutically acceptable carrier; and

    c) instructions for applying the therapeutically effective amount of the purified viable gram-negative bacteria in the pharmaceutically acceptable carrier topically to the skin.

12. The kit of claim 11, wherein the donor subject does not have atopic dermatitis.

13. The kit of claim 11, wherein the at least one strain of *Roseomonas mucosa* is lyophilized.

14. The kit of claim 11, wherein the pharmaceutically acceptable carrier is a liquid.

15. The kit of claim 16, wherein the liquid is water.
16. The kit of claim 11, wherein the at least one strain of *Roseomonas mucosa* is present in an amount of 10^4 to 10^{12} colony forming units.
17. The kit of claim 11, wherein the at least one strain of *Roseomonas mucosa* is present in an amount sufficient for a reduction in *Staphylococcus aureus* in a subject.
18. The kit of claim 11, wherein the at least one strain of *Roseomonas mucosa* comprises up to three strains of *Roseomonas mucosa*.
19. The kit of claim 11, wherein the at least one strain of *Roseomonas mucosa* comprises up to five strains of *Roseomonas mucosa*.