SUPPLEMENTAL FIGURES AND TABLES



Supplemental Figure 1. *C. auris* colonization on mouse skin surface and within skin tissue compartment does not elicit overt skin inflammation or skin architecture disruption. Related to Figure 1. (A) Grocott's methenamine silver stain for fungi on ear pinna longitudinal section of a WT mouse topically associated seven days prior with *C. auris*. Clusters of *C. auris* cells (purple) can be seen on the skin surface (stratum corneum) and located around a hair shaft. 50 μm scale bar is displayed at the bottom right. (B-D) Hematoxylin and eosin staining of pinna skin transverse sections from a naïve WT mouse (B), mouse topically associated seven days prior with *C. auris* (D). epi: epidermis, derm: dermis, cart: cartilage, HF: hair follicle, SG: sebaceous gland. 100 μm scale bar is displayed at the bottom right of each panel.



Supplemental Figure 2. Intravenous injection of *C. albicans*, but not *C. auris*, is lethal to WT mice. Related to Figure 1.

 1×10^5 *C. albicans* SC5314, four genetically distinct clades of *C. auris*, and the NIH CC strain (South Asian clade), were injected into the tail vein of WT mice, and survival followed for 30 days. N=10 for each group.



Supplemental Figure 3. Gating strategy for flow cytometric analysis of IL-17A and IL-17F production by lymphoid cell subsets in the mouse skin. Related to Figure 2. After initial FSC/SSC gating of immune cells, singlets and live cells were selected (three upper left panels). CD45+ CD90.2+ lymphoid cells were delineated as TCR β^+ TCR $\gamma\delta^ \alpha\beta$ T cells, TCR β^- TCR $\gamma\delta^{low}$ $\gamma\delta$ T cells, and TCR β^- TCR $\gamma\delta^-$ innate lymphoid cells (ILCs). Among TCR β^+ cells, CD4 T cells and CD8 T cells were delineated as Foxp3⁻ CD4⁺ CD8 β^- and Foxp3⁻ CD4⁻ CD8 β^+ cells, respectively. Among ILCs, ROR γ t was used to delineate IL-17A- and IL-17F-producing ROR γ t⁺ ILCs. The total numbers of CD4 T cells, CD8 T cells, $\gamma\delta$ T cells and ILCs were quantified and presented in Figure 2A. The IL-17A-producing CD4 T cells (Th17 cells), CD8 T cells (Tc17 cells), $\gamma\delta$ T cells and ILCs were quantified and presented in Figure 2B. The IL-17F-producing CD4 T cells, CD8 T cells, $\gamma\delta$ T cells, $\gamma\delta$ T cells and ILCs were quantified and presented in Figure 2C.



Supplemental Figure 4. Immunological factors not associated with *C. auris* skin colonization. Related to Figures 2 and 3.

(A-C) C. auris skin colonization does not elicit Th1, Th2 or regulatory T cell responses.

Numbers of interferon gamma-producing T helper cells (A), IL-5/IL-13-producing T helper cells

(B), and regulatory T cells (C) are shown from naïve WT mice, WT mice colonized by *C. auris*

7 days after first topical association, and 14 days after first topical association.

(D-F) IL-22 is dispensable in regulating *C. auris* colonization.

(D) *C. auris* colonization on ear skin surface of WT and *II22^{-/-}* mice on days 4, 10 and 14 after topical association.

(E) *C. auris* colonization on ear and back skin surface of *Act1*^{-/-} mice and *Act1*^{-/-} mice injected with IL-22 neutralizing antibody on days 10 and 14 after topical association.

(F) *C. auris* residence in ear and back skin and intestinal compartments of *Act1*^{-/-} mice and *Act1*^{-/-} mice injected with IL-22 neutralizing antibody.

(G-I) Langerhans cell deficient, $Card9^{-/-}$ and $Rag2^{-/-}$ mice have normal *C. auris* skin colonization compared with WT mice.

(G and H) *C. auris* colonization on ear skin surface or disaggregated ear skin tissue of WT and Langerhans cell deficient (G) and *Card9^{-/-}* (H) mice.

(I) *C. auris* colonization on back skin surface of WT and *Rag2^{-/-}* mice on days 2, 8 and 15 after topical association.

(A-H) N=5 mice for each group. Numbers are shown with mean \pm SEM.

Supplemental Table 1. Within tissue residence by *Candida auris* in WT mice for up to 4 months. Related to Figure 1

| Group | CFU/Pi* | |
|-----------|----------|--|
| Day58_Pi | 40,5,5,0 | |
| Day98_Pi | 0,/,5,0 | |
| Day120_Pi | 10,0,5,0 | |
| Day153_Pi | 0,0,0,0 | |

/: mortality

* each value represents an individual mouse

WT: wild-type; Pi: pinnae

Supplemental Table 2. Summary of treatments not contributing to longer-term *Candida auris* colonization or higher fungal load in the skin. Related to Figure 1

| Experimental Treatment | Colonization sites |
|---------------------------------------------------------------------|--------------------|
| AMNV ^a pre-exposure for 4 weeks | Back skin |
| FLU ^b pre-exposure for 4 weeks | Back skin |
| AMNV+FLU pre-exposure for 4 weeks | Back skin |
| Diabetic mice (db/db) | Ear and back skin |
| High-fat (western) diet-fed mice (high body mass index) for 2 weeks | Ear and back skin |
| TET ^c pre-exposure for 4 weeks | Ear and back skin |
| TMP-SMX ^d pre-exposure for 4 weeks | Ear and back skin |

^aAMNV: Ampicillin, metronidazole, neomycin and vancomycin

^bFLU: Fluconazole

°TET: Tetracycline

^dTMP-SMX: Trimethoprim-sulfamethoxazole

Supplemental Table 3. Chrorhexidine retains C. auris genomic DNA on mouse skin. Related to Figure 4

| Time | Treatment | Average CFU | Average Ct |
|--------|---------------|---------------------|------------------|
| Day 3 | Chlorhexidine | ³⁵⁴⁰ —** | 16.9 |
| | Control | 7244 | 17.3 |
| Day 7 | Chlorhexidine | 44 | 22.5 — ** |
| | Control | 76 | 25.2 — |
| Day 11 | Chlorhexidine | 7 | 27.2 – * |
| | Control | 16 | 29.6 |
| Day 15 | Chlorhexidine | 6 | 29.8 – * |
| | Control | 19 | 32.0 |

Two-tail t-test was used to compare differences between groups.

N=5 mice for each group. *: *p*<0.05; **: *p*<0.01.