

Scientists zero in on the life-threatening fungus, *Candida auris*' ability to stick

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In 2009, a mysterious fungus emerged seemingly from out of thin air, targeting the most vulnerable among us. It sounds like Hollywood, but the fungus in question poses a very real threat. Scientists are scrambling

to figure out what makes the life-threatening fungus *Candida auris* tick—and why even the best infection control protocols in hospitals and other care settings often fail to get rid of it.

Researchers at the University of Michigan have zeroed in on *C. auris*' uncanny ability to stick to everything from skin to catheters and made a startling discovery. The paper, "A *Candida auris*-specific adhesin, SCF1, governs surface association, colonization, and virulence," has been published in *Science*.

The investigative team, led by Teresa O'Meara, Ph.D. of the U-M Medical School Department of Microbiology and Immunology and her graduate student Darian Santana, has discovered that *C. auris* is unlike any other known fungus in that it employs a type of protein, called an adhesin, that acts very similar to those used by oceanic organisms, such as barnacles and mollusks.

Their original hypothesis was that *C. auris* would use an adhesin from the families of sticky proteins used by other fungi like *C. albicans*. However, when they checked the usual suspects, namely proteins from the highly conserved ALS and IFF/HYR families, they came up mostly short, except for one protein, IFF4109, with a partial affect.

They then pivoted to a different screening method to systematically break the genome of *C. auris* and see which mutant lost its ability to stick to 96-well plastic plates—leading to the discovery of a new adhesin they named Surface Colonization Factor (SCF1).

"The new adhesin is only present in *C. auris* so we don't know where it came from evolutionarily. It doesn't look like it came from any other organisms by sequence similarity," said O'Meara. The bonds formed by Scf1, they revealed, are cation-pi bonds, which are among the strongest non-covalent chemical bonds in nature.

Said O'Meara added, "Much of the literature about this type of bond in nature comes from people trying to bioengineer glue that adheres underwater. Hence, they've looked to nature for inspiration."

Furthermore, the team discovered that SCF1 was associated with increased colonization and an enhanced ability to cause disease. Using mouse models, they demonstrated that a loss of both SCF1 and IFF4109 diminished the ability of a strain of *C. auris* to colonize skin and an indwelling catheter. What's more, strains designed to over express SCF1 saw enhanced virulence and more fungal lesions.

"We don't know why this adhesin is required to cause disease," said O'Meara. "It could be that they're required to attached to [blood vessels](#), or maybe they change the host-receptor interactions which has been true for the related fungus *Candida albicans*, but we don't know in this case."

O'Meara and her team plan to investigate the link between SCF1 and virulence in the hopes of exploiting it for a more effective anti-fungal therapy, as many strains of *C. auris* are resistant to current medications. The adhesin may also provide a clue about where *C. auris* came from, said O'Meara, with its barnacle-like adhesive properties suggesting an oceanic origin.

"Unlike SARS-CoV2, which emerged in one place, *C. auris* suddenly appeared in five distinct locations around the world. There's some [selective pressure](#) in the world that changed that allowed *C. auris* to go from not a threat at all to colonizing people."

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More information: Darian J. Santana et al, A Candida auris –specific adhesin, Scf1 , governs surface association, colonization, and virulence, *Science* (2023). [DOI: 10.1126/science.adf8972](https://doi.org/10.1126/science.adf8972)

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