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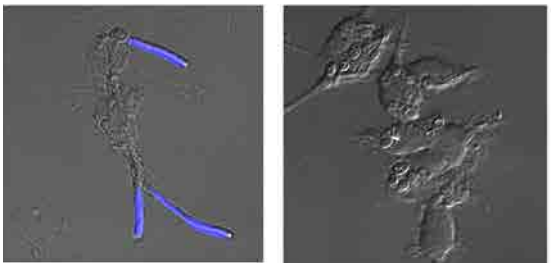
Microbial pathogens kill millions of people yearly, and the rise of drug resistance is an important concern. We focus on the human fungal pathogen *Candida albicans*, an important pathogen that affects a large proportion of the population by causing both superficial and invasive infections. Life-threatening infections with *Candida* number >400,000 yearly, with mortality of 15-50% despite the use of state of the art antifungal treatments.

The projects in our laboratory aim to understand the regulatory mechanisms that enable *C. albicans* to subvert immune responses and resist antifungal therapy through metabolic and developmental adaptation. We are particularly interested in how these pathways modulate the processes at the interface between host and pathogen.

To work on these problems, we use an interdisciplinary approach with molecular and cell biology, *ex vivo* and *in vivo* models of infection (immune cells, mouse models, and invertebrate animal infection systems), imaging, and the "omics" approaches of Systems Biology. We further collaborate with chemists and materials scientists to design new antimicrobial compounds, and surfaces for biomedical devices that are refractory to microbial colonization.

Research Projects

1. **Understanding innate immune evasion by Candida**
2. **Metabolic reprogramming and mitochondrial activity in host-pathogen interactions and virulence**
3. **Characterisation of transcriptional and posttranscriptional regulators of gene expression in Candida developmental transitions and pathogenicity**
4. **New antimicrobial compounds and surfaces against biofilm pathogens**



Left: Wild type Candida filaments lysing macrophages.
Right: If the formation of filaments is disrupted by a genetic mutation, macrophage lysis does not occur.

The image is adapted from Fig 2 in Uwamahoro et al mBio 2014, v5: e00005.

Selected significant publications:

1. Qu Y*, Locock K, Verma-Gaur J, Hay I, Meagher L, **Traven A***#. 2016. Searching for new strategies against polymicrobial biofilm infections: guanylated polymethacrylates kill mixed bacterial-fungal biofilms. *J Antimicrob Chemother* 71: 413 (*equal contr)

2. Verma-Gaur J, Qu Y, Harrison PF, Lo TL, Quenault T, Dagley MJ, Bellousoff M, Powell DR, Beilharz TH#, **Traven A#**. 2015. Integration of Posttranscriptional Gene Networks into Metabolic Adaptation and Biofilm Maturation in *Candida albicans*. *PLoS Genet* 11(10): e1005590

3. Uwamahoro N, Verma-Gaur J, Shen HH, Qu Y, Lewis R, Lu J, Bambery K, Masters SL, Vince JE, Naderer T#, **Traven A#**. 2014. The pathogen *Candida albicans* hijacks pyroptosis for escape from macrophages. *MBio* 5(2): e00003-14.

4. Hewitt VL, Heinz E, Shingu-Vazquez M, Qu Y, Jelicic B, Lo TL, Beilharz TH, Dumsday G, Gabriel K, **Traven A#**, Lithgow T#. 2012. A model system for mitochondrial biogenesis reveals evolutionary rewiring of protein import and membrane assembly pathways. *Proc Natl Acad Sci USA* 109(49): E3358-66.

5. Uwamahoro N, Qu Y, Jelicic B, Lo TL, Beaurepaire C, Bantun F, Quenault T, Boag PR, Ramm G, Callaghan J, Beilharz TH, Nantel A#, Peleg AY, **Traven A#**. 2012. The functions of Mediator in *Candida albicans* support a role in shaping species-specific gene expression. *PLoS Genet* 8(4): e1002613.

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