Slide 1

What's up fungus?

Are fungal emerging diseases increasing and with increasing consequences for biodiversity?

Slide 2

Aspergillus sydowii in Caribbean coral – has not been seen in Australia
Aphanomyces invadens (epizootic ulcerative syndrome) – regionally endemic in Australia, but range is expanding[1]
Aphanomyces astaci in crayfish (crayfish plague) – exotic to Australia
Fusarium solani complex in loggerhead sea turtles – fungus has been detected in Australian eggs, but mass mortalities have not occurred[2]

[1]Go *et al*. 2012. Detection of Aphanomyces invadans and epizootic ulcerative syndrome in the Murray-Darling drainage. AVJ 90:513-514.

[2]Sarmiento-Ramírez *et al.* 2014. Global Distribution of two fungal pathogens threatening endangered sea turtles. PlosOne http://dx.doi.org/10.1371/journal.pone.0085853.

Slide 3

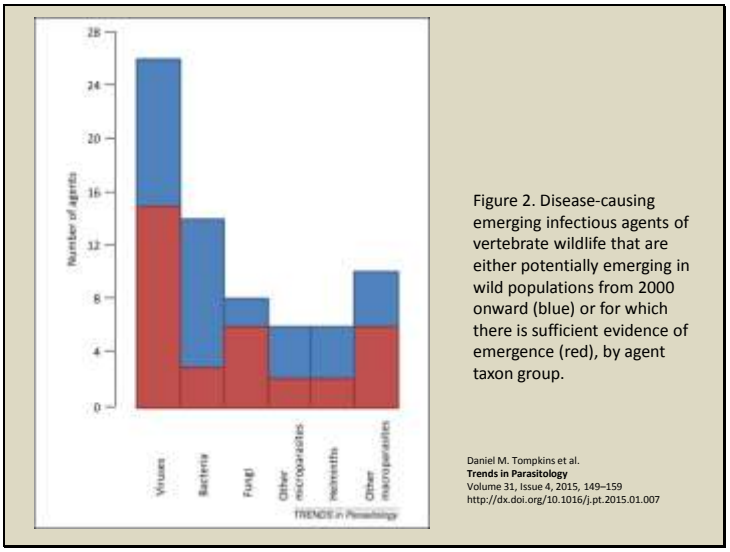
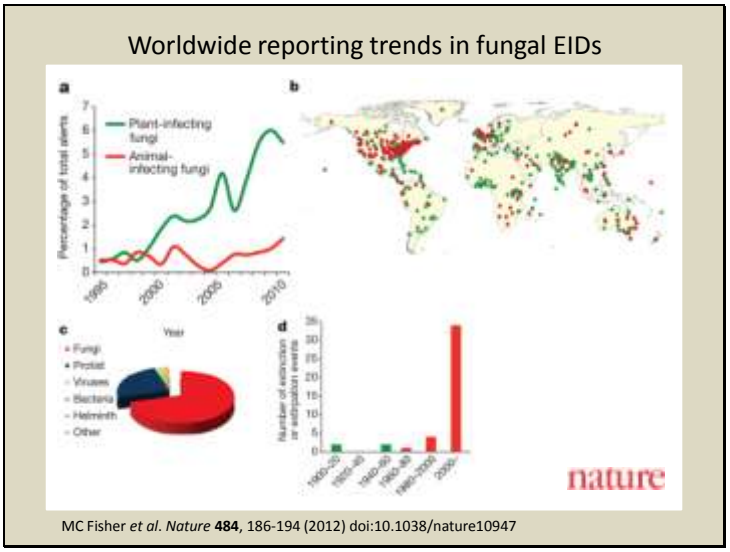


Figure 2. Disease-causing emerging infectious agents of vertebrate wildlife that are either potentially emerging in wild populations from 2000 onward (blue) or for which there is sufficient evidence of emergence (red), by agent taxon group.

Daniel M. Tompkins et al.
Trends in Parasitology
Volume 31, Issue 4, 2015, 149–159
http://dx.doi.org/10.1016/j.pt.2015.01.007

Fungi are not the most prevalent of the emerging infectious diseases of wildlife, still far surpassed by viruses.

Slide 4



a, **b**, Disease alerts in the ProMED database for pathogenic fungi of animals and plants (**a**), and the spatial location of the associated reports (**b**). **c**, **d**, Relative proportions of species extinction and/or extirpation events for major classes of infectious disease agents (**c**) and their temporal trends for fungal pathogens (**d**).

"We cannot discount the idea that sampling bias owing to increasing awareness of pathogenic fungi as EIDs may contribute to the patterns that we document. However, because of our observation that increases in the amount of disease caused by fungi are seen across many sources of data, including disease alerts, the peer-reviewed literature and previously noted patterns in human fungal EIDs, we believe that these trends are real." (Fisher *et al.* 2012)

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Why are they so good at this?

- High virulence – particularly in naïve hosts

High virulence – nearing 100% mortality in naïve populations of frogs and bats infected with chytridiomycosis and white nose syndrome respectively. Evolutionary theory would predict that pathogens co-evolve with their hosts, rather than extirpate them and generally we do see this as pathogen transmission is typically density dependent and therefore pathogens fade out before the host is entirely lost. However, when a pathogen is introduced to a naïve host, the time to co-evolve and pathogen dynamics is not sufficient to prevent extinction. Fungi often have a high reproductive potential and in a large host population this effect can result in all individuals becoming infected before the population is driven to the low densities at which the pathogen can no longer spread (Fisher *et al.* 2012)

eg. Fire salamanders in Netherlands dropped to 4% of original population within 4 years following introduction of *Bsal*

Slide 6

Why are they so good at this?

- Long-lived environmental stages

Fungi have remarkably resilient dispersal stages (a feature that they share with some spore-forming bacteria, such as *Bacillus anthracis*). The ability to survive independently outside of their host, as a free-living saprophyte or as durable spores in the environment, is probably the most important feature in driving the emergence of pathogenic fungi, owing to an increased risk of transporting the inocula to naive hosts (Fisher *et al.* 2012)

eg. White nose syndrome is believed to have been introduced to the US through transport of soil on cavers' boots.

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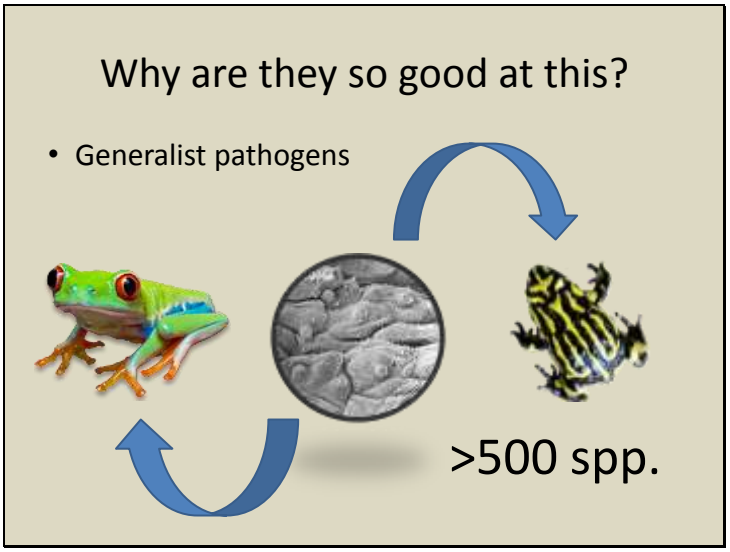
Why are they so good at this?

- Tolerant of extreme environmental variability

Aspergillus sydowii are terrestrial soil fungi which can be washed through freshwater drainage systems into the marine environment and subsequently infect corals.

Slide 8



Fungi exhibit the broadest spectrum of host ranges for any group of pathogens (Fisher *et al*. 2012).

eg. *B. dendrobatidis* is known to have infected over 500 host species.

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Fungal barriers to reproduction evolve more rapidly between lineages that are newly diverging than between geographically separated allopatric lineages. This process is termed reinforcement. What it means is that when humans facilitate the mixing of geographically separate fungi, they can undergo genetic exchange, resulting in rapid evolution (Fisher *et al.* 2012).

eg. *Cryptococcus gattii* outbreaks in northwestern North America.

Engelthaler *et al.* 2014. *Cryptococcus gattii* in North American Pacific Northwest: Whole-population genome analysis provides insights into species evolution and dispersal. mBio vol. 5 no. 4 e01464-14

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Other drivers

- Trade and transport

Modeling indicates that although fungi may currently be restricted in location by abiotic and biotic factors, the potential ranges of pathogenic fungi may be much larger than their realised range (Fisher *et al*. 2012).

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Other drivers

- Environmental change

Climate change may be providing more opportunities for fungi to express greater virulence

Slide 12

Fungi

Not just secondary invaders

Slide 13

References

- Engelthaler *et al.* 2014. *Cryptococcus gattii* in North American Pacific Northwest: Whole-population genome analysis provides insights into species evolution and dispersal. mBio vol. 5 no. 4 e01464-14
- Fisher *et* al. 2012. Emerging fungal threats to animal, plant and ecosystem health. Nature 484: 186-194.
- Go *et al*. 2012. Detection of *Aphanomyces invadans* and epizootic ulcerative syndrome in the Murray-Darling drainage. AVJ 90:513-514.
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