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Review

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Bacterial mycophagy: definition and diagnosis of a unique bacterial–fungal interaction

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Summary

This review analyses the phenomenon of bacterial mycophagy, which we define as a set of phenotypic behaviours that enable bacteria to obtain nutrients from living fungi and thus allow the conversion of fungal into bacterial biomass. We recognize three types of bacterial strategies to derive nutrition from fungi: necrotrophy, extracellular biotrophy and endocellular biotrophy. Each is characterized by a set of uniquely sequential and differently overlapping interactions with the fungal target. We offer a detailed analysis of the nature of these interactions, as well as a comprehensive overview of methodologies for assessing and quantifying their individual contributions to the mycophagy phenotype. Furthermore, we discuss future prospects for the study and exploitation of bacterial mycophagy, including the need for appropriate tools to detect bacterial mycophagy *in situ* in order to be able to understand, predict and possibly manipulate the way in which mycophagous bacteria affect fungal activity, turnover, and community structure in soils and other ecosystems.

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I. What is (bacterial) mycophagy?

Mycophagy, from the Greek mykes (= fungus) and phagein (= to eat), can be broadly defined as 'feeding on fungus'. Synonymous with the terms fungivory and mycetophagy, it covers the practice of purposefully consuming fungal tissue. Mycophagy has been reported for a wide variety of organisms, such as primates (Hanson *et al.*, 2006), pigs (Bertault *et al.*, 2001), rodents (Frank *et al.*, 2006), birds (Simpson, 1998), mollusks (Silliman & Newell, 2003), insects (Mueller *et al.*, 2005; Tuno, 1999; Robertson *et al.*, 2004), mites (Melidossian *et al.*, 2005), and nematodes (Bae & Knudsen, 2001). These fungivores have in common that they ingest fungal material, such as fruiting bodies ('sporocarps', i.e. mushrooms or truffles), spores, mycelium and/or hyphae, for internal digestion and nutrient extraction. Owing to their small size and different anatomy and physiology, fungal and bacterial fungivores rely on other mechanisms to derive nutrition from fungi. Fungal mycophagy, also known as mycoparasitism (Barnett, 1963; Jeffries, 1995) has been studied quite extensively in several species, especially *Trichoderma* (Steyaert *et al.*, 2003). Mycophagous behaviour of the latter is characterized by: formation of coiled hyphal structures around the hyphae of the host fungus; penetration of the cell wall because of the production of enzymes that break down cell wall components (Zeilinger *et al.*, 1999); the release of antibiotics that permeate the perforated hyphae and prevent resynthesis of the host cell wall (Lorito *et al.*, 1996); and growth on the cytoplasm of host hyphae (Inbar *et al.*, 1996). Other fungal fungivores, such as *Gliocephalis hyalina* (Jacobs *et al.*, 2005), *Stephanoma phaeospora* (Hoch, 1978) and *Verticillium biguttatum* (Vandenboogert & Deacon, 1994) employ not a necrotrophic but a biotrophic strategy to derive nutrition from their host (Barnett & Binder, 1973). This interaction may or may not involve direct contact with or even penetration of the target fungus.

The term bacterial mycophagy has been coined only recently (de Boer *et al.*, 2005; Fritsche *et al.*, 2006) to describe the ability of bacteria to grow at the expense of living fungal hyphae. In the current review we will adopt this general definition of bacterial mycophagy, but with the qualification that the mycophagous bacterium must have an active role in obtaining food from fungi. For example, the mere consumption of nutrients that passively leak from a fungus or are released from hyphae damaged or dead through unrelated causes does not meet our strict definition of mycophagy. Also, the ability of some bacteria to lyse fungi (see section II.1) is not necessarily synonymous to mycophagy without direct or indirect evidence that such bacteria actively use fungal-derived nutrients to multiply. The active role of bacteria multiplying inside fungal hyphae (see section II.3) lies less in the ability to utilize cytoplasmic compounds for growth (which makes these bacteria, by definition, mycophagous), but more so in the aptitude to enter into and survive within this intracellular environment. In order to cover as much ground as possible, we define bacterial mycophagy as the demonstrable and quantifiable effect of bacterial phenotypic behaviours that make available nutrients from living fungi and allow the conversion of living fungal biomass into bacterial biomass. Within this context, several demonstrated or suspected examples of mycophagous bacteria are available in the literature (Table 1).

Bacterial fungivores may feature a wide range of adaptations that contribute to their mycophagous phenotype. In section II, we will discuss the types of interaction that bacteria can have with fungi (including oomycete fungi such as *Pythium* and *Phytophthora*, which are not true fungi; see Alexopoulos *et al.*, 1996) and that contribute to the mycophagy phenotype. For each, we will consider the specific types of adaptations that distinguish this interaction.

II. Strategies for bacterial mycophagy

The strategies used by bacteria to obtain nutrients from fungal tissue can be subdivided into three main categories (Fig. 1), similar to those used to classify bacterial interactions with plants or animals: extracellular necrotrophy, extracellular biotrophy, and endocellular biotrophy. Each of these interactions is characterized by a set of mycophagy determinants (Table 2). In necrotrophic interactions, bacteria secrete proteins or low molecular weight toxins that permeabilize and lyse fungal hyphae, or inhibit fungal metabolism, thereby killing fungal cells and releasing nutrients for bacterial growth. By contrast, extracellular biotrophs do not kill fungal hyphae, but live in close proximity, often colonizing hyphal surfaces, and using nutrients exuded from living fungal cells. Biotrophs are able to tolerate or suppress the production of anti-bacterial metabolites by fungal cells, and may be able to modulate fungal metabolism to promote nutrient release. Finally, endocellular biotrophs multiply inside living fungal cells, absorbing nutrients directly from the fungal cytoplasm. In practice, many interactions may involve one or more of these phases. For example, a biotrophic interaction may progress to necrotrophy as bacterial numbers increase, and an extracellular biotroph may penetrate into and grow inside fungal cells. However, for simplicity we will consider each of these strategies separately.

1. Necrotrophic interactions

The defining feature of necrotrophic interactions is host cell death, which can be caused by the loss of cell wall or membrane integrity, the inhibition of essential metabolic processes, or the induction of programmed cell death. As will be detailed later, all these strategies may be available to mycophagous bacteria, and necrotrophic bacteria may use a combination of two or more of these mechanisms to kill and consume fungal cells. There is likely to be considerable overlap between the molecular mechanisms involved in saprotrophic growth on dead fungal cells and necrotrophic growth on killed fungal

Table 1 Examples of bacteria with demonstrated or suspected mycophagous traits

Bacterial genus/species	Origin	Evidence for mycophagy	Target fungus or fungi	References
Aeromonas caviae	Healthy bean plant roots	Use of live mycelium as sole carbon source in liquid medium	*Rhizoctonia solani*, *Sclerotium rolfsii, Fusarium oxysporum* f. sp. *vasinfectum*	Inbar & Chet (1991)
Bacillus cereus, *Bacillus megaterium*, and *Pseudomonas* sp.	Soils artificially infested with *Fusarium oxysporum* f. *cubense*	Direct isolation of halo-forming bacterial colonies on inorganic salts medium containing live fungus as sole carbon source	*Fusarium oxysporum* f. sp. *cubense*	Mitchell & Alexander (1961)
Bacillus thuringiensis	Not specified	Local digestion of fungal cell walls, loss of protoplasm	*Fusarium roseum* var. *sambucinum*	Cherif *et al.* (2002)
Burkholderia cepacia complex	Mycosphere of *Pleurotus ostreatus*	Improved bacterial growth in co-culture with fungus	*Pleurotus ostreatus*	Yara *et al.* (2006)
Burkholderia gladioli pv. *agaricicola*	Mushroom farm	Rapid degradation of mushroom sporocarps	*Agaricus bitorqis*	Chowdhury & Heinemann (2006)
Collimonas spp.	Sandy coastal dune soils	Increase in bacterial CFUs in autoclaved, acid-purified beach sand that was artificially explored by fungal hyphae extending from a potato-dextrose agar disk	*Chaetomium globosum*, *Mucor hiemalis*	de Boer *et al.* (2001)
Myxobacterial spp.	Soil	Perforation of hyphal and conidial cell walls, invasion and emptying of the fungal cell content	*Cochliobolus miyabeanus*, *Rhizoctonia solani*	Homma (1984)
Nostoc punctiforme	Cyanolichens	Endocellular location	*Geosiphon pyriforme*	Schuessler *et al.* (1996)
Paenibacillus polymyxa	Soil	Attachment and accumulation of bacteria to fungal hyphae in dual culture	*Fusarium oxysporum*	Dijksterhuis *et al.* (1999)
Paenibacillus sp.	Mycorrhizosphere of *Sorghum bicolor*	Disorganization of fungal cell walls and/or cell contents	*Fusarium oxysporum* *Phytophthora parasitica* (not a true, but an oomycete fungus)	Budi *et al.* (2000)
Paenibacillus sp.	*Laccaria bicolor* S238N fermentor culture	FISH detection of physiologically active bacteria inside fungal hyphae	*Laccaria bicolor* S238N	Bertaux *et al.* (2003)
Pseudomonas aeruginosa	Clinical environment	Formation of bacterial biofilms on filamentous host cells during coincubation in spent bacterial medium	Filamentous *Candida albicans*	Hogan & Kolter (2002)
Pseudomonas stutzeri Ypl-1	Soil cultivated with ginseng	Lysis of cell walls and outflow of cytoplasm in dual culture of bacteria and fungal mycelium in potato-dextrose broth	*Fusarium solani*	Lim *et al.* (1991)
Pseudomonas tolaasii	Mushroom compost beds	Tolaasin-evoked disruption of fungal membranes, resulting in nutrient release	*Agaricus bisporus*	Soler-Rivas *et al.* (1999)
Staphylococcus aureus	Clinical environment	Bacterial-induced fungal cell death	*Cryptococcus neoformans*	Saito & Ikeda (2005)
Streptomyces albus	Not specified	Bacterial colonization of fungal hyphae in sterile sand	*Aspergillus niger*	Rehm (1958)
Streptomyces spp.	Spores of the arbuscular mycorrhizal fungus *Gigaspora gigantea* recovered from a maritime sand dune	Formation of internal projections and/or fine radial canals in spore walls	*Gigaspora gigantea*	Lee & Koske (1994)

FISH, fluorescence *in situ* hybridization.

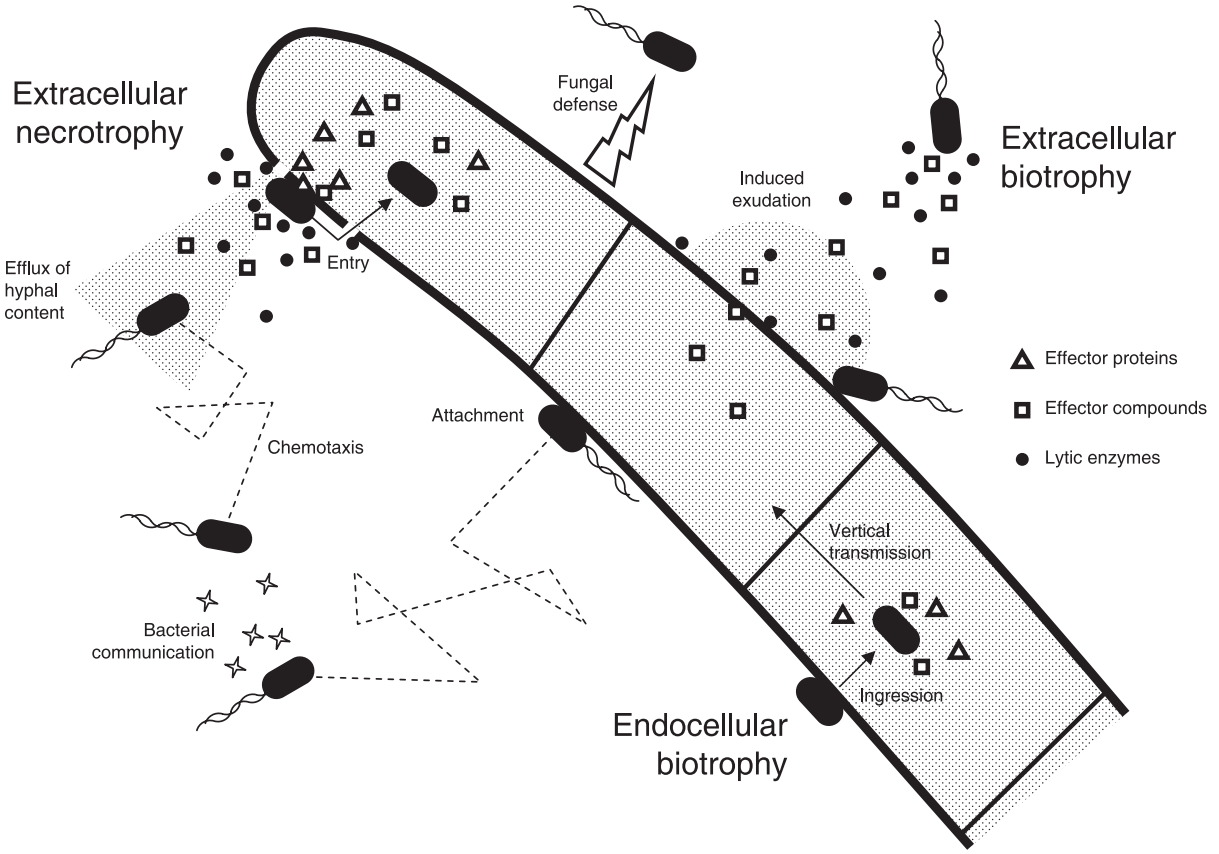

Fig. 1 Schematic representation of the three bacterial strategies to derive nutrition from fungi: extracellular necrotrophy, extracellular biotrophy and endocellular biotrophy. The actions/reactions ('interactivities') that are thought to play an important role in one or more of these strategies are indicated. A more detailed description of each of these interactivities is given in Section II. Parts 1–3.

cells. Fungal cells have a finite lifespan, and much of the fungal biomass available as a growth substrate for bacteria in natural environments may arise because of endogenous cell death mechanisms and abiotic stress rather than interactions with soil organisms. We define necrotrophic mycophagy as being restricted to examples in which bacteria actively kill fungal cells and subsequently assimilate fungal metabolites.

Bacterial lysis of fungal hyphae has been observed in a wide range of taxonomically distinct bacteria, including actinomycetes, β-proteobacteria, bacilli and myxobacteria (de Boer *et al.*, 2005). Much of the published literature on fungicidal factors produced by bacteria has been generated in the context of mushroom pathogenesis, biocontrol of fungal pathogens, and screens for fungicidal compounds of medicinal value. Very few studies have explicitly examined whether the use of these factors confers direct benefits in terms of facilitating bacterial assimilation of fungal metabolites. The clearest examples of links between fungal cell death and bacterial growth can be found in studies of mushroom pathogenic bacteria, which grow solely on nutrients available from mushroom cap tissue (Soler-Rivas *et al.*, 1999). Mushroom pathogenic bacteria cause a wide range of strain-specific symptoms, in terms of the colour of lesions (ranging from yellow to brown or purple) and the degree to which lesions are sunken or pitted, or have a matt or shiny appearance (Wells *et al.*, 1996; Soler-Rivas *et al.*, 1999; Godfrey *et al.*, 2001), which indicates that mushroom pathogenic bacteria use a diverse range of pathogenicity factors that affect the physical manifestation of disease.

Necrotrophic fungal mycoparasites such as *Trichoderma* spp. secrete a wide variety of cell wall degrading exoenzymes, such as chitinases, which play an important role in necrotrophic growth (Viterbo *et al.*, 2002; Benitez *et al.*, 2004). However, although chitinolysis is a common trait in bacteria that exhibit antifungal activity (Chernin *et al.*, 1995; Nielsen *et al.*, 1998; de Boer *et al.*, 2004, 2005; Hoster *et al.*, 2005; Ajit *et al.*, 2006; Leveau *et al.*, 2006), chitinase activity alone appears to be insufficient to account for bacterial lysis of fungal hyphae (Chernin *et al.*, 1995; Budi *et al.*, 2000; Zhang & Yuen, 2000; Kobayashi *et al.*, 2002). The complexity of the fungal cell wall makes it a formidable challenge as a primary target for bacterial attack, as bacteria would need to rapidly produce a wide variety of exoenzymes to degrade cell wall

Table 2 Putative bacterial mycophagy determinants

Putative determinant	Predicted targets and functions	Type of interaction
Flagella, type IV pili, chemotaxis mechanisms	Motility and chemotaxis towards fungal exudates	Necrotrophy, extracellular biotrophy
Pili, fimbriae, adhesins	Adhesion to fungal surfaces	Necrotrophy, extracellular biotrophy
Biosurfactants	Reduce surface tension and hydrophobicity, promote colonization of fungal surfaces, promote membrane lysis	Necrotrophy, extracellular biotrophy
Transport and assimilation of fungal exudates	Nutrition	Necrotrophy, extracellular biotrophy
pH tolerance	Tolerance to acidic fungal exudates	Necrotrophy, extracellular biotrophy
Antibiotic resistance	Resistance to antibacterial compounds produced by fungi	Necrotrophy, extracellular biotrophy
Extracellular polysaccharides	Stress tolerance, adhesion, colonization of fungal surfaces, carbon storage	Necrotrophy, extracellular biotrophy
Cell wall degrading enzymes (e.g. chitinase, glucanase, protease)	Degradation of fungal cell walls, nutrition, lysis of fungal cells, penetration into fungal cells, movement between fungal cells, promote germination of fungal spores	Necrotrophy, endocellular biotrophy
Pore-forming toxins (e.g. tolaasin)	Disrupt membrane transport, promote nutrient release, lysis of fungal cells	Necrotrophy
Lipase	Lysis of fungal cells, nutrition, degradation of lipid signalling molecules	Necrotrophy, extracellular biotrophy
Enzyme-inhibiting toxins (e.g. cyanide)	Inhibit fungal respiration and metabolism, kill fungal cells, modulate metabolic activity	Necrotrophy, extracellular biotrophy
Transport-inhibiting toxins	Inhibit or modulate membrane transport, kill fungal cells, promote nutrient release	Necrotrophy, extracellular biotrophy
Intracellular protein delivery (e.g. T3SS, T4SS)	Modulate fungal signal transduction and development, modulate fungal metabolism, promote or inhibit apoptosis	Necrotrophy, extracellular biotrophy, endocellular biotrophy
Apoptosis elicitors	Promote apoptosis, e.g. by mimicking fungal apoptosis signals	Necrotrophy
Degrade/mimic fungal quorum-sensing signals	Modulate fungal signal transduction and development	Necrotrophy, extracellular biotrophy
Indoleacetic acid (IAA)	Modulate fungal signal transduction and development	Necrotrophy, extracellular biotrophy
Nitrogen fixation	Provide fixed nitrogen to fungal host, promote growth of fungal host	Extracellular biotrophy, endocellular biotrophy
Photosynthesis	Provide fixed carbon to fungal host, promote growth of fungal host	Extracellular biotrophy, endocellular biotrophy
Vitamin synthesis	Provide essential vitamins for fungal host, promote growth of fungal host	Extracellular biotrophy, endocellular biotrophy
Degrade antibiotics and xenobiotics	Increase drug resistance of fungal host, promote growth and survival of fungal host	Endocellular biotrophy
Transport and assimilation of cytoplasmic or vacuolar nutrients	Nutrition	Endocellular biotrophy

components to the level needed to compromise structural integrity (de Boer *et al.*, 2005). For example, the cell wall of *Saccharomyces cerevisiae* contains 85% polysaccharides (mainly ß-1,3-glucan, ß-1,6-glucan, chitin and chitosan) and 15% proteins (mainly mannoproteins; Lesage & Bussey, 2006). Other chemicals commonly found in fungal cell walls include ß-1,4-glucan, mannan and dityrosine (Coluccio *et al.*, 2004), melanins (Nosanchuk & Casadevall, 2006) and hydrophobins (Linder *et al.*, 2005).The majority of molecular genetic studies of antagonistic and pathogenic interactions between bacteria and fungi have identified low molecular weight toxins as the primary causal agents of fungal inhibition and cell death (Soler-Rivas *et al.*, 1999; Haas *et al.*, 2000; Haas & Defago, 2005). For example, lipodepsipeptide toxins such as tolaasin appear to be the primary pathogenicity factors in brown blotch disease of mushrooms caused by *Pseudomonas tolaasii* (Rainey *et al.*, 1993; Soler-Rivas *et al.*, 1999; Lo Cantore *et al.*, 2006). However, Wells *et al.* (1996) identified 14 out of 219 strains of *Pseudomonas* from mushroom-casing soil that gave positive results in toxin synthesis assays, but were unable to cause symptoms when inoculated onto mushroom caps. This could indicate that toxin synthesis alone is insufficient for disease, or that variation between toxins has a significant effect on the outcome of interactions.

Tolaasin mediates membrane disruption via two routes: pore-formation or biosurfactant activity. Biosurfactant activity allows the pathogen to increase the 'wettability' of hydrophobic host surfaces in order to promote bacterial colonisation. Tolaasin

does this by the formation of an amphipathic left-handed α-helix in a hydrophobic environment (Jourdan *et al.*, 2003). Pseudomonads are known to produce a wide variety of biosurfactant compounds, including rhamnolipids, peptolipids and other lipodepsipeptides such as the syringopeptins. Toxins such as syringomycin or tolaasin are lethal to fungal cells at concentrations ranging from 0.8 to 200 μM (Sorensen *et al.*, 1996).

Toxin- and enzyme-secreting necrotrophs may use toxins as the primary mechanism for inhibiting and killing fungal cells and subsequently use chitinases, lipases, glucanases and proteases to saprotrophically degrade and assimilate fungal polymers. Nevertheless, several studies have shown that cell wall degrading exoenzymes can also act synergistically with bacterial toxins to enhance antifungal activity (Lorito *et al.*, 1994; Fogliano *et al.*, 2002; Woo *et al.*, 2002; Someya *et al.*, 2007). Proteases have been linked to mushroom pathogenesis by *Pseudomonas tolaasii* (Soler-Rivas *et al.*, 1999) and lipase activity is a common feature of mushroom pathogenic strains (Wells *et al.*, 1996).

Antagonistic bacterial–fungal interactions are typically assessed *in vitro* in terms of an unoccupied 'inhibition zone' between a bacterial colony and fungal hyphae cocultured on an agar plate. However, microscopic observations of bacteria–fungi interactions in more natural conditions clearly show that at least some antagonistic bacteria actively move towards and colonize the surface of fungal hyphae (Arora *et al.*, 1983; Lim & Lockwood, 1988; Grewal & Rainey, 1991; Singh & Arora, 2001; Bolwerk *et al.*, 2003; de Weert *et al.*, 2004). Detection of fungal-specific compounds may help bacteria find their way to the target. For example, fusaric acid (5-butylpicolinate), a secondary metabolite secreted by *Fusarium* strains, appears to be an excellent bacterial chemo-attractant (de Weert *et al.*, 2004). Bacteria may use a variety of mechanisms to attach themselves to fungal hyphae, including pili and exopolysaccharide (EPS) (Rainey, 1991; Sen *et al.*, 1996; Peng *et al.*, 2001). In one study, a *Streptomyces* strain was observed to penetrate and coil around fungal hyphae in a manner analogous to that of fungal parasites such as *Trichoderma* spp. (de Boer *et al.*, 2005). Biosurfactants may be important in attachment to fungal mycelia and spores, as they reduce surface tension and facilitate contact between bacteria and fungal cells (Nielsen *et al.*, 1999; Braun *et al.*, 2001).

Attachment to fungal surfaces plays a key role in the killing of *Cryptococcus neoformans* by *Staphylococcus aureus* (Ikeda *et al.*, 2007). Terminal deoxynucleotidyl transferase biotin-dUTP nick end labeling (TUNEL) assays for DNA fragmentation show that *C. neoformans* undergoes contact-dependent programmed cell death (apoptosis) when cocultured with *S. aureus* (Saito & Ikeda, 2005). Fungi undergo apoptosis in response to a variety of stimuli, including developmental programmes, aging, mating type incompatibility, and toxins (Marek *et al.*, 2003; Buttner *et al.*, 2006). Bacterial modulation of eukaryotic apoptosis is a well-characterized feature of bacterial interactions with plants and animals, but has not been extensively studied in the context of bacteria–fungi interactions, although some examples are known from fungi–fungi interactions. *Penicillium chrysogenum* produces a small, basic, cysteine-rich protein that is actively internalized by other fungi and induces programmed cell death (Leiter *et al.*, 2005). Any necrotrophic bacterium that acquires the ability to induce apoptosis also acquires the ability to rapidly and efficiently kill fungal cells, and it seems likely that more examples of bacteria-induced apoptosis in fungi will be discovered.

2. Extracellular biotrophic interactions

Bacteria do not need to kill fungal cells in order to obtain nutrients from fungi; neither is the presence of bacteria on fungal surfaces necessarily deleterious. Actively growing hyphae exude a complex mixture of low molecular weight metabolites that include organic acids such as oxalic, citric and acetic acid, peptides, amino acids, sugars and sugar alcohols (Griffiths *et al.*, 1994; de Boer *et al.*, 2005; Medeiros *et al.*, 2006). Some of these are secreted as waste products, whereas others are secreted as antimicrobial compounds or mobilizing compounds for phosphate and other minerals. Fungi also secrete a wide variety of iron-chelating siderophores, which may be degraded or assimilated by bacteria (Winkelmann, 2007). Numerous studies have shown that the bacterial communities associated with fungal hyphae, fungal spores, or with the mycorrhizosphere of mycorrhizal plants display fungi-specific differences in composition (de Boer *et al.*, 2005; Frey-Klett & Garbaye, 2005; Roesti *et al.*, 2005). This suggests that extracellular bacteria are under selection to develop fungi-specific traits that confer a competitive advantage during colonization of fungal surfaces. Such traits could be 'passive' traits, such as the ability to use nutrients that are specific to, or particularly abundant in fungal exudates, or the ability to tolerate antibacterial metabolites. Alternatively, they could be 'active' traits, such as the ability to alter fungal membrane permeability to increase or modify nutrient efflux (de Boer *et al.*, 2005). Chemotaxis, attachment and tolerance to fungal antimicrobial chemicals are all likely to be important traits for extracellular biotrophs.

Bacteria that grow on and tolerate fungal exudates without entering into a direct interaction with fungal cells can be defined as saprotrophs. As per our definition, a mycophagous extracellular biotroph can be defined as a bacterium that actively interacts with living fungal cells to promote bacterial growth, without lysing or killing host cells. Extracellular biotrophic interactions can be beneficial or detrimental to the fungal host.

One example of a beneficial extracellular association between fungi and bacteria can be seen in the form of lichens. Although many lichens are formed as a result of associations between fungi (mycobiont) and algae (photobiont), some lichens, known as cyanolichens, involve bipartite symbioses between fungi and cyanobacteria, or tripartite symbioses

between fungi, algae and cyanobacteria (Honegger, 2001; Richardson & Cameron, 2004; Kneip *et al.*, 2007). Many cyanolichens are restricted to, or are most common in, old growth and mature forests. The nutritional benefits provided to the fungal partner by association with photosynthetic and nitrogen-fixing cyanobacteria are clear, although the benefits to the photobiont are less well understood. In tripartite symbioses, cyanobacteria are concentrated in special areas called cephalodias, in which the mycobiont actively envelops the bacteria and incorporates them into the thallus where they are protected from high oxygen concentrations and where they can fix nitrogen efficiently (Honegger, 2001). Cyanolichens may provide a special example of mycophagy, in which the symbiont is primarily dependent on its fungal partner for essential minerals, such as phosphate and iron, rather than carbon and nitrogen (Kneip *et al.*, 2007), although it seems likely that cyanobacteria do take up organic nutrients from fungal partners, particularly at night (Rai *et al.*, 1981).

Biotrophic modulation of fungal physiology by extracellular bacteria is likely to take three main forms: modulation of fungal development; modulation of membrane permeability and nutrient efflux; modulation of fungal metabolism. There is a growing body of evidence to show that bacteria can and do modify fungal differentiation and development in a wide variety of ways. Bacteria-induced alterations to fungal development and differentiation include inhibition or promotion of germination, and alterations to foraging behaviour, hyphal branching, growth, survival, reproduction, exudate composition and production of antibacterial metabolites, each of which could benefit extracellular bacteria (de Boer *et al.*, 2005). For example, mycorrhizal helper bacteria (MHB) promote mycorrhization of plant roots and induce alterations in the architecture of mycorrhizal fungi (Garbaye, 1994; Frey-Klett & Garbaye, 2005; Aspray *et al.*, 2006; Frey-Klett *et al.*, 2007), and soil bacteria such as *Pseudomonas putida* have been shown to promote sporocarp formation in *Agaricus bisporus* (Rainey *et al.*, 1990). Mycorrhizal helper bacteria have also been shown to induce changes in the transcriptome of mycorrhizal fungi (Schrey *et al.*, 2005; Deveau *et al.*, 2007). At present, there is no clear evidence to show whether these developmental changes do benefit bacteria, as per our criteria for bacterial mycophagy. However, it seems likely that increased mycorrhization, and associated increases in nutrient availability for plants and fungi may benefit plants, bacteria and fungi in a tri-trophic interaction, while localized accumulation of nutrients in the developing sporocarp, or dispersal in association with fungal spores, could benefit sporocarp-inducing bacteria.

The mechanisms by which bacteria alter fungal development are poorly understood. However, some extracellular molecules secreted by bacteria have been shown to affect fungal development. For example, many plant-associated and rhizosphere bacteria secrete the auxin indole acetic acid (IAA), a molecule that is best known for its role in plant signal transduction (Quint & Gray, 2006). However, IAA has also been shown to act as a signal molecule in bacteria, mammals and fungi (Leveau & Lindow, 2005; Bianco *et al.*, 2006; Liu & Nester, 2006; Yang *et al.*, 2007) and to induce adhesion and filamentation of *Saccharomyces cerevisiae* (Prusty *et al.*, 2004).

One set of bacterial signals known to be detected by a wide variety of eukaryotes, including algae, nematodes, plants, fungi and mammalian cells, are quorum sensing (QS) signals (Hogan *et al.*, 2004; Wang *et al.*, 2004; Shiner *et al.*, 2005; Beale *et al.*, 2006; Wheeler *et al.*, 2006). Quorum sensing systems involving peptides and low molecular weight molecules such as farnesol, tyrosol, tryptophol and phenylethanol have also been reported to occur in fungi (Alem *et al.*, 2006; Blankenship & Mitchell, 2006; Sprague & Winans, 2006; Lee *et al.*, 2007). Interestingly, the bacterial QS signals 3-oxo-C12 homoserine lactone and *cis*-11-methyl-2-dodecenoic acid, and the fungal QS signal farnesol all inhibit filamentation in *Candida albicans* (Hogan *et al.*, 2004; Wang *et al.*, 2004). Bacteria, plants, algae and fungi have also been shown to degrade QS molecules and to produce QS inhibitors that inhibit or modulate signalling through QS mechanisms (Rasmussen *et al.*, 2005; Shiner *et al.*, 2005; Gonzalez & Keshavan, 2006; Karamanoli & Lindow, 2006). The fungal signal farnesol inhibits production of the *Pseudomonas* quinoline signal (Cugini *et al.*, 2007). Quorum sensing molecules and QS inhibitors may therefore play a key role in orchestrating interactions between fungi and bacteria.

3. Endocellular biotrophic interactions

Endocellular biotrophs are entirely dependent on their fungal host for nutrients, at least for the duration of their endocellular existence. Some endocellular biotrophs are vertically transmitted, while facultative endocellular biotrophs possess mechanisms for invading and subverting fungal cells. Many of the known examples of endocellular bacteria isolated from fungi belong to the β-proteobacteria, which also contains pathogens that colonise and survive in the cytoplasm of mammalian and amoebae cells, such as *Burkholderia mallei* and *Burkholderia pseudomallei* (Levy *et al.*, 2003). Both pathogenic and nonpathogenic *Burkholderia* spp. can colonize the interior of *Gigaspora decipiens* spores when applied to spore surfaces (Levy *et al.*, 2003), but the molecular mechanisms involved in this interaction and in most endocellular bacteria–fungi interactions are unknown. The fungus *Geosiphon pyriformis* forms multinucleated 'bladders' at its hyphal tips, which are colonized by the cyanobacterium *Nostoc punctiforme*. The primary function of these bladders is likely to be photosynthesis, but the symbiont also forms heterocysts, which suggests that it also fixes nitrogen (Schuessler & Kluge, 2001; Kluge, 2002). Experimental results suggest that there is a degree of recognition and host-specificity in the *G. pyriformis-Nostoc* symbiosis, as some strains of *Nostoc* are not incorporated into the fungus, and *Nostoc* has to be in the early primordial (immobile) stage

for endocytosis of free-living bacteria to occur (Kluge, 2002). *Nostoc* cells in a mature bladder are 10 times larger than free-living cells and have a higher concentration of photosynthetic pigments. Interestingly, although the association between *G. pyriformis* and *Nostoc* appears to be mutually beneficial, changes in the growth medium, such as an increased level of phosphate, can result in the cyanobacterial symbiont 'overpowering' the fungus and terminating the symbiosis (Kluge *et al.*, 2002).

The symbiosis between *G. pyriformis* and *Nostoc* is in some respects a relatively primitive and unstable symbiosis, which involves horizontal acquisition of a free-living bacterium. By contrast, the vertically transmitted obligate endocellular bacterium *Candidatus* Glomeribacter gigasporarum colonizes the spores of *Gigaspora margarita* at densities ranging from 3700 to 26 000 bacteria per spore (Jargeat *et al.*, 2004). *Ca.* G. gigasporarum has an estimated genome size of 1.35 Mb, and was originally classified into the genus *Burkholderia*, but has subsequently been assigned to a new taxon, *Glomeribacter* (Bianciotto *et al.*, 2003; Jargeat *et al.*, 2004). The small genome size suggests that this bacterium, like other obligate pathogens and symbionts, is entirely dependent on fungal cells for many metabolic functions.

An increasing number of studies have shown evidence to support the hypothesis that endocellular bacteria provide important biochemical functions for their fungal hosts in exchange for their exclusive niche (Minerdi *et al.*, 2001). Lumini and collaborators (2007) cured a strain of *G. margarita* of its endogenous endocellular bacteria, and found that although the fungus could still colonize plants and complete its lifecycle under laboratory conditions, the cured strain showed altered spore morphology, reduced presymbiotic hyphal growth and reduced branching, which is associated with reduced competitive fitness. A primary function of endocellular bacteria may be to provide nutritional benefits for fungi, for example, by fixing nitrogen or synthesizing essential nutrients, as noted for the cyanobacterial symbioses described earlier (Jargeat *et al.*, 2004). Although symbiotic nitrogen fixation has been most widely studied in relation to α-proteobacteria and cyanobacteria, many β-proteobacteria are also able to fix nitrogen, both as free-living bacteria and in symbioses with plants (Moulin *et al.*, 2001; Chen *et al.*, 2003, 2005; Elliott *et al.*, 2007), and it is possible that some endosymbiotic β-proteobacteria also fix nitrogen.

A second symbiotic function of endocellular bacteria could be as a source of antimicrobial molecules that protect fungi against predation and parasites, or promote pathogenesis towards eukaryotic hosts. For example, the rice seedling blight pathogen *Rhizopus microsporus* contains endocellular bacterial symbionts belonging to the genus *Burkholderia* which can be cultured *in vitro* and which produce the toxins rhizoxin and rhizonin (Partida-Martinez & Hertweck, 2005; Scherlach *et al.*, 2006; Partida-Martinez *et al.*, 2007a). Bacteria may also protect fungi against antifungal chemicals. Zygomycetes have become an increasingly problematic source of opportunistic, drug-resistant infections in hospitals in recent years and researchers have speculated that the emergence of these strains coincides with the acquisition of endocellular, drug-resistant bacteria (Chamilos *et al.*, 2007).

4. Effector proteins – tools to promote necrotrophic and biotrophic mycophagy?

Colonization of animal and plant hosts by extracellular and endocellular bacteria frequently involves the use of protein secretion systems such as the type III and type IV secretion systems (T3SS and T4SS), which can deliver proteins, or in the case of *Agrobacterium*, proteins and DNA, directly into the cytoplasm of host cells (Preston *et al.*, 2005; Backert & Meyer, 2006; Cambronne & Roy, 2006; Galan & Wolf-Watz, 2006; Angot *et al.*, 2007). T3SS- and T4SS-secreted effectors could have a wide range of roles in bacteria–fungi interactions, based on their functional characterization in animal and plant cells, which include altering fungal development, inducing or suppressing apoptosis, and suppressing antibacterial defence mechanisms (Christie *et al.*, 2005; Guiney, 2005; Suparak *et al.*, 2005; Grant *et al.*, 2006; Gurlebeck *et al.*, 2006; Pilatz *et al.*, 2006; Pizarro-Cerda & Cossart, 2006; Schlumberger & Hardt, 2006; Angot *et al.*, 2007; He *et al.*, 2007; Ninio & Roy, 2007). T3SS-mediated-modulation of cellular processes could play a particularly important role in endocellular colonization by bacteria such as *Burkholderia* spp., which are known to use T3SSs to promote endocellular colonization of animal cells (Ulrich & DeShazer, 2004; Pilatz *et al.*, 2006; Ribot & Ulrich, 2006; Stevens *et al.*, 2002). However, at present, there is no experimental evidence to show that mutants impaired in T3SS or T4SS are impaired in mycophagy. The only direct evidence of an ecological role for T3SS in bacterial interactions with soil microorganisms comes from studies involving other soil eukaryotes, such as the oomycete pathogen *Pythium ultimum* (Rezzonico *et al.*, 2005) and the amoebae *Dictyostelium* (Pukatzki *et al.*, 2002). Nevertheless, several lines of indirect evidence support the hypothesis that intracellular protein delivery could be used by mycophagous bacteria. The first line of evidence comes from the observation that nonflagellar T3SS genes and T3SS-secreted effector genes are present in soil and rhizosphere bacteria belonging to the genera *Pseudomonas*, *Burkholderia*, *Ralstonia*, *Chromobacterium* and *Rhizobium*, including strains that are known to interact with fungi (Ferguson *et al.*, 2001; Preston *et al.*, 2001; Smith-Vaughan *et al.*, 2003; Betts *et al.*, 2004). In addition, numerous studies have shown that T3SS-secreted effectors used by bacterial pathogens of plants and animals are biologically active when expressed in fungal cells (Valdivia, 2004). For example, the *Shigella* effector IpaH9.8 interrupts pheromone response signalling in yeast by promoting the proteasome-dependent destruction of the MAPKK Ste7 (Rohde *et al.*, 2007). Effectors secreted by the plant pathogen

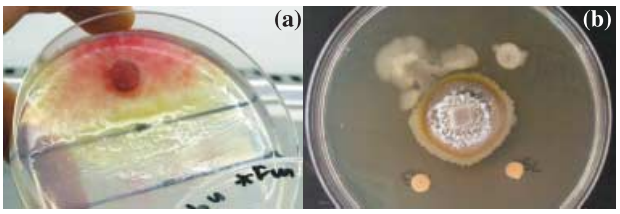

Fig. 2 (a) Accumulation of bacterial biomass at the interface between *Collimonas* bacteria (inoculated in the centre of the plate between the two black lines) in confrontation on water agar with the fungus *Fusarium culmorum* (growing from the agar plug on the top half of the plate). (b) *Pseudomonas* sp. NZ104 (inoculated top-left) is seen growing towards and encircling the fungus *Magnaporthe grisea* (centre of the plate).

Pseudomonas syringae can suppress apoptosis in yeast cells (Jamir *et al.*, 2004), and the YopE effector protein of *Yersinia* severely inhibits the growth of *Saccharomyces cerevisiae* when expressed in yeast cells from an inducible promoter (Lesser & Miller, 2001). Intriguingly, Ferguson and collaborators (2001) reported that genes encoding the YopE-related effector ExoS of *P. aeruginosa* were more common in soil isolates than in clinical isolates. *Pseudomonas aeruginosa* also secretes a phospholipase, ExoU, which rapidly kills yeast cells when expressed as a transgene (Rabin & Hauser, 2003; Sato *et al.*, 2006; Sitkiewicz *et al.*, 2007). Yeast has also been used as a host to study the cellular localization of T3SS effectors (Sisko *et al.*, 2006).

Numerous studies have shown that the *Agrobacterium* T4SS can be used to deliver proteins and DNA into fungal cells, and *Agrobacterium* has been widely used as a tool for fungal transformation (Bundock *et al.*, 1995; de Groot *et al.*, 1998; Schrammeijer *et al.*, 2003; Blaise *et al.*, 2007). This clearly demonstrates that T4SS could be used by bacteria for protein delivery, and even fungal transformation in natural environments. T4SS-secreted effectors have primarily been studied in the context of bacterial interactions with protozoan and mammalian cells, where they perform functions analogous to the T3SS-secreted effectors discussed above (Christie *et al.*, 2005; Ninio & Roy, 2007). However, as with T3SS effectors, the targets and biological activities of numerous T4SS-secreted effectors have also been shown to be functionally conserved in yeast, indicating that T4SS effector proteins could perform similar functions in bacterial interactions with fungi (Campodonico *et al.*, 2005; Ninio & Roy, 2007; Schrammeijer *et al.*, 2003; Garcia-Rodriguez *et al.*, 2006).

III. Practical definitions of bacterial mycophagy

While it is quite easy to define bacterial mycophagy as the ability of bacteria to feed on living fungi, it is by no means a trivial task to establish through experimentation whether a bacterial species or isolate is mycophagous or not. Direct observation of fungivorous behaviour, as has been done for animals (Lee & Widden, 1996; Hanya, 2004) is not practical or even possible for bacteria. Their small size requires the use of high-magnification microscopes, and bacteria typically lack an easily recognizable or measurable reaction to the presence of fungal food. Other techniques that work for animals, for example analysis of stomach or scat content for indirect evidence of mycophagy (Currah *et al.*, 2000; Frank *et al.*, 2006), obviously do not apply. What follows is a list of established or suggested approaches that can be used individually or in combination to provide proof of mycophagous behaviour, and to identify genes and/or proteins that contribute to this behaviour. The list is by no means exhaustive, but serves to provide the phenomenon of bacterial mycophagy with a first set of more practical definitions.

Demonstrate an increase in bacterial biomass or numbers in the presence of fungus as the only source of nutrients This approach of offering live fungi to bacteria has been used successfully (Mitchell & Alexander, 1961; Inbar & Chet, 1991), although great care should be taken to avoid false-positive interpretation (de Boer *et al.*, 2005). The mycophagous nature of *Collimonas* species was assessed by introducing collimonads as a suspension into autoclaved, acid-purified beach sand. Bacteria were left to starve for 1 wk, after which fungal hyphae of *Mucor hiemalis* or *Chaetomium globosum* were allowed to infiltrate into the soil matrix from potato-dextrose agar. Numbers of *Collimonas* bacteria in the sand were estimated by plate counting and shown to be higher in sand inoculated with fungi than in uninoculated sand. This assay can be adapted to work on water agar plates, containing no substrate for the bacteria other than fungal inoculum on the same plate (Fig. 2), although controls lacking fungal inoculum should be included to avoid false-positive bacterial growth on components in the agar preparation. As an alternative means of quantifying cell numbers, real-time polymerase chain reaction (PCR) can also be used (Höppener-Ogawa *et al.*, 2007). Evidence for an active role of bacteria in obtaining nutrients must be deduced from control assays (e.g. by using mycophagous mutants of the same strain or other nonmycophagous strains; de Boer *et al.*, 2001). An important consideration is that the conversion of fungal to bacterial biomass does not need to result in a proportional increase in bacteria numbers. Given the low carbon-to-nitrogen ratio of bacteria compared with fungi, a fungal meal to a bacterium is in essence nitrogen-limited, which leads us to predict that bacterial fungivores might have adapted to be efficient at storing excess carbon. The strategy of using EPS for nutrient storage has been reported for other bacteria (Laue *et al.*, 2006). Experimental evidence for this prediction in bacterial fungivores is currently lacking, although it has been noted (Fig. 2) that bacteria of the genus *Collimonas* and *Pseudomonas* become slimy during growth on *Fusarium culmorum* and *Magnaporthe grisea*, respectively, perhaps suggesting the incorporation of excess carbon into the bacterial EPS layer.

Demonstrate a nutrient flux from fungus to bacterium Several approaches are available for this, mostly based on stable or radioactive isotopes. One example involving the fungal mycoparasite *Eudarluca caricis* is based on measurements of naturally occurring isotope abundances. Comparison of ^{15}N in this fungus, its rust fungus host (*Melamspora medusae*), and the plant host of *M. medusae* (*Populus trichocarpa*) revealed an increasing $^{15}N/^{14}N$ ratio (Nischwitz *et al.*, 2005), confirming that *E. caricis* feeds on *M. medusae* and not on nutrients obtained directly from the plant. Another approach involves the experimental labeling of fungi with a stable isotope and subsequent detection of label in the fungivore (Ruess *et al.*, 2005). Although these types of methodology have not yet been applied to bacterial fungivores, proofs of principle for an isotopic approach towards bacterial mycophagy are plentiful. For example, there are several reports on the use of, for example, ^{13}C-labeled carbon sources to demonstrate their specific utilization by bacterial species or whole communities (Singleton *et al.*, 2007), and ^{13}C-labeled *Escherichia coli* were used to identify bacteriovores in an agricultural soil (Lueders *et al.*, 2006). Incorporation of stable isotopes in bacteria can be detected by various methods, including Raman spectroscopy (Huang *et al.*, 2007) and analysis of biomolecules such as phospholipids or nucleic acids (Boschker & Middelburg, 2002).

Demonstrate the existence and activity of bacteria living and multiplying inside fungal hyphae Endocellular bacteria have been detected and quantified by staining with fluorescent dyes or by the use of fluorescent *in situ* hybridization (Bianciotto & Bonfante, 2002; Bianciotto *et al.*, 2004). The most convincing evidence for the ability of these bacteria to multiply inside their host is the microscopic observation of dividing bacterial cells inside fungal hyphae, spores and other structures (Bianciotto *et al.*, 2004). Indirect proof comes from viability stains for bacterial cells (Bertaux *et al.*, 2003), and from the use of reverse-transcription PCR on RNA isolated from fungal tissue to reveal active bacterial gene expression (Bianciotto & Bonfante, 2002).

Demonstrate the ability of bacteria to destabilize the fungal cell wall Bacteria that adopt a necrotrophic approach to mycophagy rely at some point on their ability to weaken the fungal cell wall and/or membrane. There are several methods available that allow for the observation or quantification of cell wall integrity. Electron microscopy can reveal cell wall distortions and even holes after attack by bacterial (Budi *et al.*, 2000) and fungal (Siwek *et al.*, 1997; Picard *et al.*, 2000) antagonists. A recently developed assay to assess cell wall integrity is based on a *gfp* bioreporter strain of *Aspergillus niger* (Hagen *et al.*, 2007) and was used to confirm the induction of cell wall stress by the antifungal protein AFP from *Aspergillus giganteus*, which specifically inhibits chitin synthesis in sensitive fungi. Indirect assays for the disruption of cell wall and membrane are based on the detection of cytoplasmic content leaked from damaged fungi, for example detection of an easily measurable fungi-specific enzyme activity (Jewell *et al.*, 2002).

Identify mutants that exhibit reduced or abolished mycophagy There are several ways to exploit bacterial mutants to discover the mechanism(s) underlying mycophagy, and to confirm that mycophagy is an active process. One is based on random transposon mutagenesis and screening of the resulting library for mutants with an altered mycophagous phenotype. The relative success of this screening depends, among other things, on the compatibility of the screening assay with a high-throughput format. No truly high-throughput assays for bacterial mycophagy exist, which hampers current efforts to identify genes involved in this phenotype. Alternatively, one can hypothesize which bacterial properties are likely to contribute towards mycophagy and then screen for mutants affected in those properties. Such mutants may subsequently be tested in a mycophagy assay, to assess the effect of the gene disruption. For fungal mycoparasites, similar approaches have confirmed the involvement of chitinase activity (Woo *et al.*, 1999; Brunner *et al.*, 2003), *N*-acetyl-beta-D-glucosaminidase activity (Brunner *et al.*, 2003) or the hyperosmotic stress response (Delgado-Jarana *et al.*, 2006). In a bacterial example, several transposon mutants of *Collimonas* were isolated which lacked chitinolytic activity on chitin agar plates (Leveau *et al.*, 2006), but none were shown to be significantly affected in mycophagy. Notably, mutants need not always have a reduced mycophagous phenotype: it was shown for *Trichoderma virens* that a knockout in the *tvk1* locus increased the expression level of mycoparasitism-related genes (Mendoza-Mendoza *et al.*, 2003). A potentially very interesting but yet unexplored role in mutant-based approaches is reserved for mutants that are unable to synthesize essential compounds such as amino acids or nucleic acids: these should not be affected in mycophagy as long as the essential compound is provided in their fungal diet. Thus, systematic screening of auxotrophic mutants in a mycophagy assay could be used to determine the nutritional value of fungi to bacteria that feed on them. In this context, it is interesting that a *pyrB* mutant of antifungal *Pseudomonas putida* 06909, which is unable to synthesize its own pyrimidine, was not able to grow in the presence of *Phytophthora parasitica* (Lee & Cooksey, 2000), suggesting that this interaction did not provide the bacterium with sufficient amounts of pyrimidine to sustain growth.

Determine transcriptional profiles of bacteria during mycophagy Microarrays allow the genome-wide interrogation of gene expression levels of bacteria in interaction with their hosts (Cummings & Relman, 2000). In the study of bacterial mycophagy, they would assist in the discovery of genes involved in but not absolutely required for mycophagy and in the analysis of temporal changes in gene expression during the

different phases of mycophagy. However, a microarray strategy requires a (partial) genome sequence and, until now, very few bacterial species have been chosen for sequencing based specifically on their interactions with fungi. The sequenced strain *Burkholderia xenovorans* LB400 was formerly named *Burkholderia fungorum* LB400 for its relatedness to *Burkholderia* species found associated with fungal hyphae, but was primarily sequenced for its ability to aerobically degrade environmental contaminants. Ongoing genome projects include those for the mycophagous bacterium *Collimonas fungivorans* (de Boer *et al.*, 2004) and the mycorrhizal helper bacterium *Pseudomonas fluorescens* BBc6 (Deveau *et al.*, 2007). There is a clear need for additional genome sequences from suspected or confirmed bacterial fungivores, not in the least to be able to fully exploit the strength of comparative genomics for the discovery of mycophagy-related genes, for understanding the distribution and diversity of such genes among different bacteria, and for appreciating the evolutionary forces that shaped mycophagy in these different genomes. A list of candidates for genome sequencing also should include bacterial endosymbionts of fungi: the inability of some to be cultured has not proven to be a major obstacle for isolation and analysis of their genomic DNA (Jargeat *et al.*, 2004).

Demonstrate the ability of bacteria to modulate fungal physiology This can be achieved by different means, one of which would be the identification of changes in gene expression in fungi that are being fed upon. The feasibility of such an approach is confirmed by similar studies on bacterial–fungal interactions. For example, a suppressive subtractive hybridization approach was used to identify changes in gene expression of fly agaric (*Agaricus muscaria*) in response to the presence of a *Streptomyces* species (Schrey *et al.*, 2005). Other studies have revealed the effect of *Streptomyces* (Becker *et al.*, 1999) or *Pseudomonas* (Deveau *et al.*, 2007) on expression profiles of ectomycorrhizal fungi. A list of the first 50 microarray studies in filamentous fungi (Breakspear & Momany, 2007) does not include data on mycoparasitized fungi, but does contain the names of several fungal species that in other studies have been shown to be susceptible to mycophagy or antifungal activity. The availability of fungal microarrays and microarray data can be exploited to deduce what mechanisms underlie a mycophagous assault by bacteria (e.g. based on comparison with microarray data obtained from the same fungus under controlled conditions of various stresses). Obviously, changes in mycophagy-induced fungal physiology may not only be interpreted from transcriptional profiling, but can also be measured and quantified from the analysis of the proteome, metabolome, or secretome (e.g. as changes in enzyme activity), protein profiles and exudation patterns.

Utilize bioreporter technology for spatial and temporal analysis of bacterial mycophagy Bacterial bioreporters are useful tools in microbial ecology (Leveau & Lindow, 2002). Fluorescent reporter proteins such as green fluorescent protein (GFP) have revolutionized the *in situ* observation of bacterial–fungal interactions (Toljander *et al.*, 2006; Kamilova *et al.*, 2007; Partida-Martinez *et al.*, 2007b), by facilitating the quantification of bacterial cells and assessment of their location relative to their host. Fluorescent proteins can also be used to label fungi and fungal proteins, often in contrasting colours (Nahalkova & Fatehi, 2003). Another type of bioreporter is one that carries a reporter gene fused to a promoter with known responsiveness to a specific condition (e.g. the availability of a particular carbon source or the growth status of the bacterium). Such reporters can be used to probe the physical, chemical and biological microenvironment in which bacterial–fungal interactions take place. They have been used with great success to map nutrient availability to bacteria in the rhizosphere and phyllosphere (Leveau & Lindow, 2002), and should do equally well in describing the mycosphere during the different phases of bacterial mycophagy. A third kind of bioreporter addresses the need to know whether a gene of interest is expressed, or not, during the process of mycophagy. This is achieved by cloning the gene's promoter element upstream of a promoterless reporter gene and interpreting the expression of reporter protein as a measure for gene activity. A related type of application is aimed at identifying those genes that are specifically induced during the interaction of bacteria with their hosts. This is often referred to as *in vivo* expression technology or IVET (Rediers *et al.*, 2005). In a relevant example (Lee & Cooksey, 2000; Ahn *et al.*, 2007), IVET was used to study the colonization of the root-rotting oomycete *Phytophthora parasitica* by *P. putida* 06909. Several *P. putida* promoters were identified, corresponding to genes for diacylglycerol kinase, bacterial ABC transporters, outer membrane porins and proteins with unknown function. The elevated expression of these genes during colonization of *Phytophthora* could be confirmed by *lacZ* reporter gene fusions to the recovered promoter fragments (Lee & Cooksey, 2000). In summary, bioreporter technology has the potential to contribute significantly towards an increased understanding of the processes and mechanisms that underlie bacterial mycophagy.

IV. Future directions for the study of bacterial mycophagy

This review aimed to provide an overview of the current knowledge on the phenomenon of bacterial mycophagy. Briefly, one may conclude that there is ample direct and indirect evidence to acknowledge the existence of bacterial behaviours that, as per our definition, make available nutrients from living fungi and allow their conversion into bacterial biomass. Furthermore, it has become clear that bacteria can engage in mycophagous interactions in a variety of ways, so that the term 'bacterial mycophagy' really represents an amalgam of many different, serially overlapping bacterial activities, all

converging on the same objective, that is, to derive nutrition from fungi.

We conclude this review by listing some of the priorities and challenges that remain on the path to a fuller appreciation of bacterial mycophagy. These include the need for: development of unambiguous and high-throughput methods to screen environmental isolates or mutants for mycophagous phenotypes; an improved understanding of the possibilities and limitations of exploiting bacterial mycophagy for the control of fungal infections (e.g. in agricultural, medical and preservation applications); additional genomic data on bacteria with a mycophagous lifestyle. Another priority is to obtain a more comprehensive insight into the occurrence of mycophagy-related phenotypes across the bacterial domain. Obviously, there exists a bias in our knowledge, which is also reflected in this review, towards fungivores in soil and the phytosphere. Relatively little is known about fungal–bacterial interactions in other environments, such as marine and freshwater habitats, or in mixed infections of humans and animals. Applying our practical definitions of mycophagy to these environments might reveal novel fungivorous players and mechanisms.

Another challenge lies in demonstrating the relative impact of bacterial mycophagy on the structure and activity of fungal communities in nature. Graphical representations of food webs in soils and other environments typically lack an arrow pointing from fungi to bacteria, which would represent the trophic interaction that is bacterial mycophagy. It is too early and presumptuous to insist that such an arrow should be drawn without knowing more about the contribution of bacterial mycophagy in relation to all the other arrows pointing to and from fungi and bacteria. In other words, we need to be able to estimate parameters such as the turnover time of living fungus by bacteria and the amount of bacterial biomass derived from living fungal biomass in natural environments. To begin to address these questions, there is a clear need for the establishment of methods that allow the detection and quantification of bacterial mycophagy *in situ*. A promising approach is the use of stable isotope probing (Neufeld *et al.*, 2007) to identify in a culture-independent manner, using labelled fungus as bait, bacteria that exhibit mycophagous behaviour *in situ*. This would be analogous to the efforts that have been made to determine which microorganisms prey on bacteria (Lueders *et al.*, 2006) or which are the prime benefactors of plant root exudates in the rhizosphere (Leake *et al.*, 2006; Lu *et al.*, 2007). Such studies could reveal much of the diversity and specificity of bacterial fungivores in a particular environment, especially when used in combination with a metagenomic approach (Friedrich, 2006). By way of prediction, we would expect that bacterial mycophagy plays a more prominent role in fungus-featuring environments that are otherwise characterized by low alternative nutrient availability. Indeed, culturable representatives from the mycophagous genus *Collimonas* have been isolated from nutrient-poor soils such as from the dunes on the island of Terschelling, the Netherlands (de Boer *et al.*, 2001, 2004), and from natural grassland and heathland (Höppener-Ogawa *et al.*, 2007) but also forest soils (Aspray *et al.*, 2005; Mannisto & Haggblom, 2006).

The potential of bacterial mycophagy as a biocontrol strategy needs to explored more intensively. *Collimonas fungivorans* showed efficient biocontrol towards *Fusarium oxysporum* f. sp. *radicis-lycopersici*, the causative agent of tomato foot and root rot, but it is unclear whether this involved mycophagous behaviour (Kamilova *et al.*, 2007). It is worth noting that mycophagous biocontrol agents are in essence positive-feedback biocontrol agents: they inhibit the growth of harmful fungi by feeding on them, thus supporting their own growth, which in turn leads to greater biocontrol activity.

An important challenge will be to reveal the evolutionary processes that gave rise to and shaped the various expressions of bacterial mycophagy. Obviously, bacteria and fungi have coinhabited this planet for a very long time, during which there have been many opportunities for the two to interact in a variety of ways. Any one of these interactions, including bacterial mycophagy, has been subject to the process of natural selection and at least part of this process might be reconstructed through comparative genomics of bacterial fungivores to identify which genes underlie this phenotype and how they ended up and evolved in different bacterial species. It will be an interesting exercise to compare the evolution of mycophagy in bacteria with that of other fungus-feeding organisms. With insects, there are examples of evolutionary transitions from one fungal host to another (Robertson *et al.*, 2004), and it has been suggested that mycophagy is a behaviour serving as an evolutionary stepping stone to phytophagy (Lindquist, 1998; Robertson *et al.*, 2004) or predation/parasitism on other insects (Whitfield, 1998; Leschen, 2000). Also, as more and deeper insights are becoming available on the mechanisms that underlie bacterial mycophagy, it will be exciting to start hypothesizing on what types of counter-adaptations fungi have evolved (e.g. resisting bacterial attack by the production of antibiotics and changes in cell wall composition and architecture or, conversely, initiating and sustaining symbiotic relations with beneficial endobacteria).

Finally, the notion that mycophagous bacteria might be beneficial to other fungivorous organisms needs closer examination. The foregut of several species of fungivorous marsupials is enlarged and harbours bacterial endosymbionts that ferment or predigest fungal biomass (Claridge & Cork, 1994). Subsistence of the earthworm, *Enchytraeus crypticus*, on the fungus *Aspergillus proliferans* is enhanced by cofeeding with *Streptomyces lividans* pCHIO12 which overproduces an exochitinase that degrades the fungal hyphae (Kristufek *et al.*, 1999). While these examples need further investigation to see if they meet our criteria of mycophagous bacteria, they still illustrate a possibly broader impact of the phenomenon of bacterial mycophagy on the biology, ecology and evolution of other organisms.

In conclusion, the study of bacterial mycophagy has a future that is characterized by many technical and conceptual challenges. Meeting these challenges will place this unique bacterial–host interaction in a broader ecological context, expand our ability to exploit it for control of unwanted fungi and, above all, feed our growing appreciation for the enormous diversity and adaptability of bacteria when it comes to finding ways to acquire food in order to thrive and survive.

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