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Review

The roles of sexual and asexual reproduction in the origin and dissemination of strains causing fungal infectious disease outbreaks

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ABSTRACT

Sexual reproduction commonly refers to the reproductive process in which genomes from two sources are combined into a single cell through mating and then the zygote genomes are partitioned to progeny cells through meiosis. Reproduction in the absence of mating and meiosis is referred to as asexual or clonal reproduction. One major advantage of sexual reproduction is that it generates genetic variation among progeny which may allow for faster adaptation of the population to novel and/or stressful environments. However, adaptation to stressful or new environments can still occur through mutation, in the absence of sex. In this review, we analyzed the relative contributions of sexual and asexual reproduction in the origin and spread of strains causing fungal infectious diseases outbreaks. The necessity of sex and the ability of asexual fungi to initiate outbreaks are discussed. We propose a framework that relates the modes of reproduction to the origin and propagation of fungal disease outbreaks. Our analyses suggest that both sexual and asexual reproduction can play critical roles in the origin of outbreak strains and that the rapid spread of outbreak strains is often accomplished through asexual expansion.

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Contents

1. Introduction

Fungi are heterotrophic eukaryotic microorganisms with thick cell walls made of chitin and cellulose. Fungi show great diversity in morphology, lifecycle and ecology, and are commonly known as molds, mushrooms, yeasts, rusts, etc. Based on the estimated fungi to plant ratio in Britain and assuming that such a ratio holds in the rest of the world, it was estimated that globally, there are about 1.5 million fungal species (Hawksworth, 2001). However, directly analyses of environmental DNA suggested that there could be as many as 5.1 million fungal species but only about 100,000 of them have been described so

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far (Blackwell, 2011). A large number of known fungal species can cause diseases to plants and animals and this ability is widely distributed in all major phylogenetic groups (James et al., 2006). These pathogenic fungi can cause a wide array of diseases ranging from rust in plants (Saunders et al., 2012) to aspergillosis in humans (Lin et al., 2001) and chytridiomycosis in amphibians (Berger et al., 1998).

For fungi to cause damage to host tissue they need to be able to do the following: (i) adhere to host external surfaces, (ii) penetrate host external surfaces and migrate to targeted tissue and/or organ, (iii) circumvent host defense system and multiply (Richardson, 1991). Pathogenic fungal species have evolved through different genetic pathways to acquire genes associated with host adherence, penetration, and survival; a process that is largely driven by gene duplication, mutation, and selection (Moran et al., 2011). Over the past several decades, because of the increasing number of immunocompromised hosts, increased global travel, and rapidly changing environmental conditions due to anthropogenic activities, pathogenic fungi have become a significant worldwide threat to plant and animal health. Indeed, the threat posed by emerging infectious diseases, including those caused by pathogenic fungi, has been rising over the last two decades (Fisher et al., 2012 and Jones et al., 2008). Some of these outbreaks have resulted in grave consequences (Chiller et al., 2013; Kentish, 2015; Malkin, 2014; Moskin, 2009; Peláez et al., 2012 and Skerratt et al., 2007a). To make matters worse, fungal species that were previously obscure are now known to be at the root of very deadly outbreaks. For example, since February 2006, an emerging cutaneous fungal disease has ravaged bat populations in northeastern United States and eastern Canada (Blehert, 2012; Blehert et al., 2009 and Leopardi et al., 2015). It is estimated that the white-nose syndrome has killed over 5 million bats and spread to 30 US states and 5 Canadian provinces. The emergence and re-emergence of such fungal diseases have grave consequences on food security and ecosystems health. It was estimated that crop losses caused by low-level persistent plant fungal diseases could feed about 600 million people if averted (Fisher et al., 2012). Consequently, understanding the origin and the mechanism by which these outbreaks are disseminated is paramount.

Several factors can lead to the emergence of fungal outbreaks, including changes in host populations, in pathogen population, in the environment from which host-pathogen interact, or a combination of all three factors. In this review, we focus on the fungal factor. Specifically, we are interested in the roles of sexual and asexual reproduction in the emergence and spread of fungal outbreak strains. Different from the majority of plants and animals, about 20% of all described fungal species are known to predominantly or exclusively reproduce asexually, the remaining 80% or so can reproduce both sexually and asexually (Lee et al., 2010 and Xu, 2004). Such characteristics make pathogenic fungi excellent model organisms for studying interplays between sexuality and clonality in outbreak initiation and propagation.

2. Search criteria

Electronic bibliographic databases were the main sources for our literature review. We searched databases such as ProMed, PubMed, and Google Scholar for fungal infectious disease outbreaks. Searches were conducted very broadly and included terms “outbreaks,” “molecular epidemiology,” “population structure,” “sexual,” “asexual,” “reproduction,” “role,” “epidemic,” “fungi,” and “infectious diseases.” Articles that contained at least 50% of a combination of search terms were further reviewed. Authors and co-authors of relevant articles were profiled and references of relevant articles were also reviewed.

3. Tradeoffs between sexual and asexual reproduction

A broad definition of sex that accommodates both conventional sex in eukaryotes and lateral gene transfer is used here to refer to any natural process that amalgamates genes from two or more sources in a single cell (Xu, 2004). Reproduction in the absence of gene amalgamation from two or more sources is referred to as asexual. Most pathogenic fungi are known reproduce both sexually and asexually (Feretzaki and Heitman, 2013; Tibayrenc and Ayala, 2012; Xu, 2004, 2010). However, the roles of sexual and clonal reproduction in the origin and propagation of strains responsible for fungal infectious diseases outbreaks are still poorly understood. In addition, recent genomic and population genetic analyses have shown that many so called “asexual” fungi actually possess intact and functioning genes related to sexual reproduction or have been shown to recombine in nature (Kück and Pöggeler, 2009). These recent findings have prompted biologists to seek an understanding of underlying conditions where one reproductive form may be favored over another.

Life-history theory hypothesizes that evolution in reproductive development is shaped by a need to achieve optimal population fitness (Nespolo et al., 2009). For example, a study published by Liu et al. (2008) showed that available nutrient levels could influence the trade-offs between sexual reproduction, clonal propagation and vegetative growth in the aquatic plant *Sagittaria pygmaea*. Similarly, a handful of other studies have revealed rather unique situations in which the selection for asexual or sexual reproduction in a population is often accompanied by trade-offs in non-selected traits to maintain optimal population fitness (Nespolo et al., 2009; Ronsheim and Bever, 2000; Sutherland and Vickery, 1988). In fungi, external factors such as temperature, light, quality and quantity of nutrients, water availability, aeration, and pH are known to influence the interplay between asexual and sexual reproduction (Chamberlain and Ingram, 1997; Xu, 2004).

Generally speaking, high and/or novel virulence trait(s) is a characteristic commonly associated with outbreak strains of most microorganisms (Baldwin et al., 2004; Taha et al., 2002; Valway et al., 1998 and Warny et al., 2005). For example, adaptations to host defense by changing virulence and transmissibility are vital mechanisms by which pathogens initiate outbreaks (Deitsch et al., 1997; Finlay and Falkow, 1997; Subramanian et al., 2001). Over the last few decades several hypotheses have been put forth to explain why sexual reproduction is ubiquitous even though it can be costly. For example, the reproductive output per individual in asexual reproduction is twice that for sexual reproduction (Otto and Lenormand, 2002). Nonetheless, most pathogenic fungi continue to reproduce sexually, at least occasionally (Lee et al., 2010). One hypothesis states that sexual reproduction within populations is maintained to remove deleterious recessive mutations accumulated as part of the DNA replication process, through a process known as Muller's Ratchet (Kondrashov, 1988). An alternative hypothesis, also known as the Red Queen Hypothesis, states that the maintenance of sexual reproduction within populations promotes adaptation in ever-changing environments (Hamilton and Zuk, 1982; Lively et al., 1990; Morran et al., 2011). The environmental changes refer to not only those of abiotic factors but also biotic factors, including interacting species.

Although sexual reproduction can accelerate adaptation to novel or changing ecological niches, adaptation to stressful or new environments can still occur in the absence of sex. For example, a study on a group of asexual rotifers showed genetic diversification that was approximately equivalent to that found in related sexual groups (Fontaneto et al., 2007). More interestingly the study demonstrated differentiation in feeding morphology among these asexual aquatic animals. Indeed, among the ancient asexual bdelloid rotifers, no male rotifers or traces of meiosis have ever been reported. The study concludes that sex is not necessary for adaptation and genetic differentiation in these rotifers.

In the following sections, we review some of the major contemporary fungal infectious disease outbreaks and analyze the relative roles of sexual and asexual reproduction in the origin and spread of these outbreaks. Specifically, we sought to answer the following question: (i) can asexual fungi initiate outbreaks? (ii) Is sex absolutely necessary for the initiation of outbreaks in sexual fungi? (iii) Are there trade-offs between sexual and asexual reproduction during outbreak initiation

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