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Diverse strain immune reactivity shapes fungal inflammation or tolerance

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For over a century microbiology and immunology have classified microorganisms in pathogenic or non-pathogenic. This definition, clearly relevant at the level of strain and species for most bacteria, has never been probed in fungal species. Understanding the nature of fungal pathogenesis will result in developing more effective therapies for fighting invasive fungal infection. Currently, several studies attempt to address pathogenicity mechanisms using different strains as a model. This study was designed to explore the immune-based diversity of Aspergillus spp strains and Saccharomyces cerevisiae fungal strains comparing different fungal life stages, from conidia to hyphae to spores. Our results show a wide strain-dependent variation of the immune response elicited indicating that different isolates possess diverse virulence and infectivity. Moreover, in contrast to the *S. cerevisiae* yeast cell-induced Th1 response, dendritic cells stimulated with yeast spores induce cellular responses shifted towards Th17 differentiation. The switch between spores and yeast is crucial for the commensalism of *S. cerevisiae* and depends on the use of a different receptor repertoire. We demonstrate that the differential recognition of specific mannan structures is one of the master regulator of the discrimination between harmful and harmless fungi. The in-vitro preliminary classification and characterization of fungal biodiversity in inducing immune responses led us to start the investigation on how/if the different cell mediated immunogenicity could result in differences in trained immunity properties of the tested *S. cerevisiae* strains. Thus, the definition of markers of inflammation or pathogenicity cannot be generalized. Understanding the role of cell wall composition in different strains and variation in the balance between tolerance and inflammation responses might lead to fully understand the boundaries between safety and pathogenicity.

Evidence for aging theories from the study of a hunter-gatherer people (Ache of Paraguay)

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For the study of a species, it is fundamental to observe it under natural conditions. For the human species, the closest condition to the natural one is that of the residual hunter-gatherer populations, which is equivalent to the human condition in the Paleolithic period. One of the few studies of this kind was conducted in late seventies on a small tribal population of Paraguay, the Ache. Data from this population turn out useful for considerations about evolutionary hypotheses on aging phenomenon. In natural conditions, after juvenile ages, Ache show an age-related increasing mortality, as "civilized" populations, and a mean duration of life (ML) of 38.8 years. Without the age-related increase of mortality, ML would be 87.75 years. The ratio between the two values is 2.260 and is in accordance with the values from other studies on mammal and bird species. In short, age-related increasing mortality limits strongly ML in natural conditions for many vertebrate species. Moreover, data for our species are also in agreement with the observation about an inverse correlation between extrinsic mortality (0,01%/year for our species) and the deaths due to the age-related increasing mortality (about 67% for our species), a relation that disproves non-adaptive aging theories and supports adaptive aging hypotheses, which predict a direct and an inverse relation, respectively. Another important information is given by the causes of death for Ache. When this people lived in the wild, the main causes of deaths for modern populations (heart attacks, diabetes, hypertension, etc.) were absent, and almost all deaths derived from accidents, violence, intoxications and infections. Cases of death by cancer were not reported, and only in the group aged 60+ years (age reached by about 31% of the population) some isolated cases of illness attributed to unspecified causes or to "old age" could have been the result of neoplastic diseases. For many gerontologists, the age-related decline of vital functions is well explained as a consequence of the gradual decline of cell turnover, genetically determined and regulated by the declining duplication capacities of stem cells. For the adaptive interpretation of aging, this is not a difficulty, and indeed it is a necessary mechanism to reduce life span. In contrast, for non-adaptive theories of aging, the progressive decline of cellular capabilities