The current progress in medicine, related to the advent of anticancer therapies, the development of transplantology and the use of immunosuppressive drugs or antibiotics with a broad spectrum of action, apart from their enormous benefits, have promoted a significant increase in the incidence of fungal infections that can pose serious global problems for human health. One of the main groups of fungal pathogens in humans is the yeasts of the *Candida* genus that live in a commensal state on the skin and mucous membranes of most healthy people. However, when physical protective barriers are damaged or the host immune mechanisms are significantly weakened, these yeasts cause a number of diseases. Common harmless surface infections, such as candidiasis of the skin, nails, mucous membranes of the mouth or throat, are well known; however, multi-organ and systemic infections, often directly life-threatening, caused by these yeasts attract a special attention.

Although *C. albicans* is the most common cause of candidiasis in humans, there have been some alarming changes in the epidemiology of infections over the past decades, including an increase in the frequency of candidiases caused by other species of the genus *Candida*, collectively referred to as "non-albicans" and characterized by drug resistance and the tendency to recur. A particular attention is paid to the *C. glabrata* species, which is currently the second most common cause of candidal infections. Despite being closely related to non-pathogenic yeast *Saccharomyces cerevisiae*, commonly used in the food and brewing industries, this species can cause infections with a mortality rate of up to 49%.

So far, little is known about this species' virulence mechanisms, but in many aspects, *C. glabrata* appears to employ essentially different strategies than other fungal pathogens to achieve the common goals, which include the host cell invasion, nutrient extraction and immune response evasion. One example of this uniqueness is the inability of *C. glabrata* to produce a filamentous form, which in the case of other *Candida* species, promotes the infection of the host organism. Despite this, *C. glabrata* can persist inside the host organism for a surprisingly long time without any infection symptoms. One hypothesis is that *C. glabrata* attracts macrophages and uses them as a "Trojan horse". Hiding in macrophages protects *C. glabrata* from the host's immune system and allows the yeast to pass unnoticed to the organs. Another example, highlighting the uniqueness of *C. glabata* is the inability to secrete typical yeast hydrolytic enzymes belonging to the aspartic protease (Saps) family. By the degradation of numerous host proteins, these enzymes ensure the acquisition of nutrients and open the way to invasion into the host's tissues; therefore, they belong to the main factors determining *Candida* virulence. Interestingly, *C. glabrata* does not produce the Saps but exposes a family of proteinases called yapsins (Yps). Yps functions are poorly understood; however, due to the mutant's phenotypic features of the mutant with a *YPS* gene deletion, it has been postulated that Yps are key players in the virulence of *C. glabrata*.

Since *C. glabrata* proteinases have not been isolated or characterized so far, this project's main aims is to determine the role of extracellular Yps in gaining the pathogenic advantage by *C. glabrata* in the host organism. In the first stage of the study, it will be checked whether Yps can damage the epithelial cell monolayer and degrade host proteins. In the next step, the Yps contribution to avoidance of the host's immune response will be analyzed, using macrophage cells and neutrophils. In a final step, using an alternative animal model – *Galleria melonella* larvae, it will be checked whether Yps can induce an immune response against itself.

Detailed understanding of the mechanisms used by *C. glabrata* to obtain nutrients and defend the fungal cells against the host immune system could contribute to the development of methods to combat infections caused by this species.