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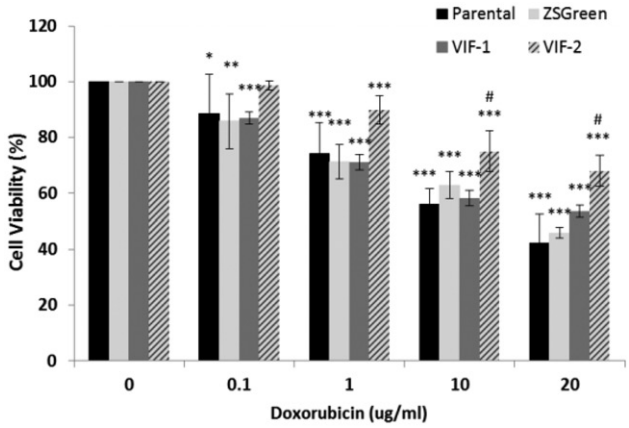

Figure 1. Viability of cells treated with doxorubicin for 24 h. Values are presented has mean ± SEM of three independent MTT assays. *p-value <.05 in relation to untreated cells from the same cell line. #p-value <.05 in relation to HCC1806 parental cell line.

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Evaluation of antifungal susceptibility in clinical isolates of the *Candida glabrata* complex

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ABSTRACT

Introduction: *Candida glabrata* is classified as an emerging threat due to its resistance profile to antifungal drugs. Associated to this, there is also the fact that recently, new species of *Candida* sp. phylogenetically related to *Candida glabrata* have been discovered: *Candida bracarensis* and *Candida nivariensis* [1]. Once that is only possible to identify these species through molecular methods [2], that identification represents a crucial step, since these species have been associated with a higher virulence and resistance to antifungals, in particular to the azole class [3], including the new extended-spectrum triazoles [4]. The aim of this study is to characterise *C. glabrata* clinical isolates from a culture collection.

Materials and methods: Seventy clinical isolates from the "Micoteca IUEM" were used, presumedly classified as *Candida* sp. Their phenotypic identification was performed, and all isolates classified as *Candida glabrata* were subjected to molecular identification through the PCR technique followed by electrophoresis, to verify the presence of cryptic species. Susceptibility tests were performed using the disc diffusion method in order to evaluate the susceptibility of the complex to fluconazole and voriconazole.

Results: Phenotypic identification showed that only 43 (61%) corresponded to *C. glabrata*. Molecular identification of these 43 isolates was carried out but led to inconclusive results. Susceptibility tests showed that one of the 43 samples of *C. glabrata* lost viability, 13 (31%) were sensitive to fluconazole, 12 (29%) were dose-dependent intermediates and 17

(40%) were resistant to fluconazole. Testing voriconazole, only 2 (5%) were resistant and the great majority, 40 (95%), was shown to be sensitive to voriconazole.
Discussion and conclusions: This study showed that resistance to fluconazole is increasing and needs to be resolved quickly, since resistance in most cases was verified. However, voriconazole appears to be a good option for resistance to fluconazole, because it has been shown to be effective in the vast majority of strains resistant to fluconazole. One of the negative implications of this study is the fact that it is not possible to identify the users who have this resistance. Finally, it is important to highlight the need to produce new antifungal agents with different mechanisms of action, in addition to moderate and optimise the use of existing drugs.

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HIV Vif protein in docetaxel treatment of breast cancer cells

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ABSTRACT
Introduction: Breast cancer is one of the most frequently diagnosed cancers in the world and is also the leading cause of death in women diagnosed with this disease. Recently, APOBEC3 proteins have been identified as potent mutagenic agents of genomic DNA associated with the onset, progression and treatment resistance of various types of cancer. On the other hand, Vif1 from HIV-1 and Vif2 from HIV-2 are proteins encoded by HIV-1 and HIV-2, respectively, that during viral infection plays a crucial role in the inhibition/degradation of APOBEC3. In this work it was tested the hypothesis that both Vif1 and Vif2 mediated APOBEC3 inhibition will increase the cytotoxicity of docetaxel in a triple-negative breast cancer cell line.
Materials and methods: Breast cancer HCC1806 cell line was used as well as two new derived cell lines, with vif1 and vif2 genes integrated and expressing vif in fusion with Zs Green fluorescent protein, mentioned hereinafter simply as VIF-1 and VIF-2 cells. Cell viability assays were performed by MTT reduction after 24 h and 48 h exposure of cells (HCC1806, VIF-1 and VIF-2) to different concentrations of docetaxel.
Results: Our results in the presence of docetaxel for 24 h have shown that cell viability decreases in all cell lines around 30% or 40%. Moreover, there is no significant differences in cell viability of parental cell line HCC1806 and any of the modified lines (VIF-1 and VIF-2) at any of the docetaxel concentrations tested (p-value $>$ .05 – independent sample t-test). Additionally, after 48 h exposure to docetaxel, cell survival also decreases significantly to values between 38 and 43% (Figure 1), but again we could not see significant differences in cell viability among HCC1806, VIF-1 and VIF-2 cells treated to any tested concentration of docetaxel.
Discussion and conclusions: The hypothesis that Vif will enhance triple negative breast cancer cells sensitivity to treatments with docetaxel was not verified. The lack of effect of Vif in cells sensitivity in the presence of docetaxel may be explained by the fact that cell target of docetaxel is Microtubules and Vif proteins do not interfere with Microtubules. Probably, the presence of Vif will only alter sensibility of breast cancer cells when using drugs that affect the DNA, the known target of APOBEC3. Therefore, treatment with drugs independent of DNA seems to be also independent of APOBEC3 levels in cells.