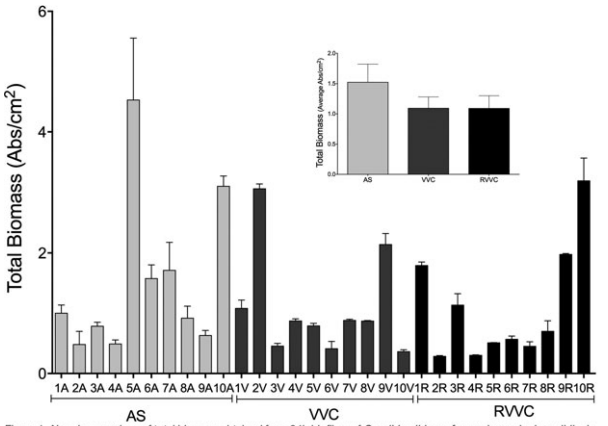

Figure 1. Absorbance values of total biomass obtained from 24h biofilms of *Candida albicans* from vulvovaginal candidiasis. AS: asymptomatic; VVC: vulvovaginal candidiasis; RVVC: recurrent vulvovaginal candidiasis. The inset represents the mean absorbance for all analyzed groups

Table 1. Range of MIC and MIC50, MIC90 of 30 clinical isolates of patients with VVC front of the antifungal fluconazole and nystatin.

	MIC (μg/mL)					
	Fluconazole			Nystatin		
Clinical groups	MIC_{50}	MIC_{90}	Range	MIC_{50}	MIC_{90}	Range
AS	0,25	0,5	0,125 - 0,25	4	4	1 - 4
VVC	0,25	0,5	0,25 - 0,5	4	4	4
RVVC	0,25	4	0,25 - 2	2	4	2 - 4

MIC_{50} and MIC_{90} were defined as capable of inhibiting 50% and 90% growth of the isolates, respectively. The range values correspond respectively to lower and higher MIC of each drug.
VVC - vulvovaginal candidiasis. RVVC - recurrent vulvovaginal candidiasis. AS - asymptomatic.

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Assessment of *in vitro* biofilm formation and antifungal susceptibility of *Candida albicans* isolates from vulvovaginal candidiasis

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Objectives Vulvovaginal candidiasis (VVC) is an inflammation of the genital mucosa, which mainly affects the vulva and vagina. *Candida* spp. are considered commensal fungus, however, when there is imbalance in the microbiota or the host immune system is compromised, these can become pathogenic. *C. albicans* is responsible for most cases of VVC and is able of expressing mechanisms which allow the colonization or infection in the host. These factors related yeasts, including the growth of strains resistant to antifungal agents and virulence attributes (such as biofilm formation) are important in the development of VVC. In this sense, the objective of this study was to evaluate the *in vitro* biofilm formation and susceptibility to antifungal of *C. albicans* isolates from patients with vulvovaginal candidiasis.
Methods For the study were analyzed 30 clinical isolates of *Candida albicans*. The clinical isolates were separated in groups of 10 samples of the according to symptoms presented by the patients: asymptomatic (AS), vulvovaginal candidiasis (VVC) and recurrent vulvovaginal candidiasis (RVVC). For all isolates were analyzed biofilm formation and minimal inhibitory concentration (MIC) for fluconazole and nystatin. The MIC was performed according to M27-A3 protocol of the Clinical Laboratory Standards Institute. Biofilm forming ability was assessed through quantification of total biomass by crystal violet (CV) staining, performed on 96-well microplates containing a cellular suspension of 1×10^7 cells ml^{-1} and incubated for 24 h at 37°C.
Results Antifungal susceptibility testing is showed in table 1. The isolates were tested to the two antifungals. The MIC raging from 0.125 to 2 μg ml^{-1} for fluconazole and 1 to 4 μg ml^{-1} to nystatin. The figure 1 show the quantification of the total biomass. It was evident that all the *C. albicans* isolates were able to form biofilm, although differences occurred depending on the isolated and consequently the group. Importantly it was noted that, in general, VVC and RVVC groups had similar capacity biofilm formation. On the other hand, these groups had less total biomass (average Abs = 1,091 ± 0.88) compared with AS group (average Abs = 1,521 ± 1.32).
Conclusion Although all the samples analyzed are sensitive to antifungals tested research of resistant strains is relevant, since recurrences are related to cases of VVC. Nystatin and fluconazole were effective in small concentrations for the isolates analysed. All samples were able to form biofilm and the average of the group of asymptomatic patients greater than the others. Thus, the capacity to forming biofilm is an important virulence factor in the persistence of microorganisms in infectious processes and represent an increase in resistance to antifungal and host defense.