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HOLOTHURIN AND CASPOFUNGIN-INDUCED ALTERATIONS IN TTOLL-LIKE RECEPTOR 4 EXPRESSION IN THE VAGINA OF RATTUS NORVEGICUS WISTAR WITH CANDIDIASIS

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Abstract

Toll-like receptor 4 is a marker that indicates whether or not tissues have immunological or pro-inflammatory responses. Candida albicans has the potential to aggravate the tissue that makes up the epithelium. After candidiasis, the antifungal properties of holothurin and caspofungin have the ability to block toll-like receptor 4.

A total of 48 white rats Rattus norvegius Wistar were divided into four positive control groups (P1) and given topical C. albicans after being grown in yeast extract peptone dextrose in the vagina of white rats Rattus norvegius Wistar. P2 and P3 groups were given 3500 g holothurin and 140 g caspofugin topically in the vagina of animal models at 12-, 24-, and 48-hour intervals. Immunofluorescence was used to analyse the study results both quantitatively and qualitatively by attaching the imaging. After that, the data was processed using the SPSS statistical software version 23.

Toll-like receptor 4 expression decreased significantly in the treatment group compared to the positive control group (p0.05). This demonstrates that holothurin (P1) and caspofungin (P2) treatments reduced toll-like receptor 4 expression in C. albicans at 0.25 and 6.375 at 12 hours, 0.62 and 3. at 24 hours, and 1.68 and 4.18 at 48 hours. The mean difference in toll-like receptor 4 expression in the positive control group, on the other hand, did not differ statistically when compared to the negative (healthy) control group. This demonstrates that the treatment group's holothurin and caspofungin have the potential to reduce toll-like receptor 4 expression.

Holothurin has a potential effect compared to caspofungin on experimental animals with candidiasis experiencing significant changes in suppressing the number of toll-like receptor 4 in vaginal epithelial tissue of Rattus norvegicus Wistar.

Keywords: *TLR4, candida albicans, holothurin, caspofungin, antifungal.*

Introduction

C. albicans invades host cells via necrosis (passive and involuntary cell death caused by the uncontrolled release of inflammatory cell contents), apoptosis (an active and purposeful process of autonomic cell disassembly that avoids the beginning of inflammation), and pyroptosis (cell death caused by species infection) [*Krysan D et al., 2014*]. Necrotic dead cells release lactate dehydrogenase during

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