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Influence of the vaginal microbiota in the expression of IDO enzyme in cervical tissue

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Cervical cancer (CC) is a multifactorial disease, in which the principal risk factor is the infection by the *Human papillomavirus* (HPV). However, some observations suggest HPV is not the only determinant for CC development and other factors may be involved, such as unbalanced immune system and cervico-vaginal dysbiosis. For instance, HPV-positive women with different degrees of cervical intraepithelial neoplasia have major bacterial dysbiosis, categorized mostly like bacterial vaginosis. The purpose of this study is to evaluate the influence of the vaginal microbiota on the expression of indolamine 2,3-dioxygenase (IDO) enzyme at an early stage of cervical carcinogenesis in cervical tissue. We used the "K14E7" CC model mice, which consists of hormonal treatment and the HPV E7 oncoprotein expression; CC model mice with low-abundance modified vaginal microbiota due to the use of a Broad-spectrum Antibiotic Cocktail (BAC) treatment during 6 days; and FvB control mice (genetic background of the K14E7 mice without E7 protein expression). We found that vaginal microbiota differs between CC model mice and the FvB mice. The relative abundance of *Proteobacteria* and *Actinobacteria* phyla is smaller, while the relative abundance of *Firmicutes* and *Cyanobacteria* phyla is larger in CC model mice with respect to FvB mice. We observed the relative microbiota abundance in the CC model mice treated with BAC is similar to the FvB mice. We also determined that CC model mice overexpressed IDO enzyme in cervical tissue compared to the FvB mice. Moreover, our results suggest that higher IDO expression is found in the CC model mice with BAC treatment compared to the CC model mice as well as to FvB mice. Based on our findings, we suggest the vaginal microbiota is different in the FvB mice compared to the CC model mice but is similar to CC model mice with BAC treatment and also this vaginal microbiota influence IDO expression in cervical tissue. This research was financed by CONACyT-163235 INFR-2011-01 and FONSEC SS/IMSS/ISSSTE-CONACYT-233361 to JGM; by SEP-CONACYT-242506 to ECM and by SEP-CONACYT-236767 to PGV.

Biography

Aida Aide Luna Perez has completed her bachelor´s degree in Experimental Biology in Guanajuato University in the faculty of chemistry. She completed her undergraduate thesis in molecular oncology area at the Instituto Nacional de Medicina Genomica. Later she made a research stay at the Cancer Research Center in Salamanca, Spain. She is currently finishing her master's degree in the Department of Genetics and Molecular Biology at CINVESTAV, Mexico.

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