

The relationship of *Trichomonas vaginalis* with sex hormones for women in Babylon / Iraq

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ABSTRACT

The current study was conducted in the period from October 2014 to April 2015; Studies were conducted to determine the correlation between some steroid hormones, particularly sex hormones (estrogen, progesterone, and testosterone) in women infected with *Trichomonas vaginalis* in Hilla province. The results showed that there is a significant difference in the rate of estrogen and progesterone, where estrogen and progesterone rates increased in the infected women compared with non- infected. And also showed no significant differences in testosterone rates between women with and non-infected with the parasite.

Key words: *Trichomonas vaginalis*, Estrogen, Progesterone, and Testosterone

INTRODUCTION

Trichomoniasis is the most common sexually transmitted disease, caused by a motile flagellate non-invasive parasitic protozoan, *T. vaginalis*^{6,1}. Women with trichomoniasis may have several associated problems such as premature labor, low birth weight, greater risk of tubal infertility, atypical pelvic inflammatory disease, amplified HIV transmission acquisition, and increased risk of cervical cancer¹².

Numerous studies have examined the influence of hormones on infectious diseases and there is now a wealth of data relating to the more specific effect of the sex hormones, estrogen and progesterone, on urogenital infections, Although it is difficult therefore to draw general conclusions regarding predominant effects of specific hormones, there is the suggestion that estrogen enhances the pathogenicity of many urogenital micro-organisms¹¹.

T. vaginalis has specific androgen and estrogen receptors, which suggest that steroid hormones could directly affect *T. vaginalis*⁵, and also the presence of hormone-binding proteins in numerous micro-organisms including protozoan lends support to the hypothesis that hormones may directly alter the virulence of micro-organisms¹³.

Animal models of *T. vaginalis* vaginitis require estrogenization to establish infection¹⁰; it was also found that, in women volunteers, a high estrogenic state is required to establish a *T. vaginalis* vaginal infection². Trichomonad movement and attachment to cells appears to be affected by the presence of estrogen¹⁵ and protein containing estrogen binding sites have been identified in *T. vaginalis* and other trichomonads pathogenic in non-human species¹⁴.

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Studies *in vitro* of *T. vaginalis* with mammalian cell cultures have demonstrated a contact dependent cytopathic effect. Organisms were able to kill target cells by direct contact without phagocytosis. At least four proteins have been identified in cell adherence. Additional, *T. vaginalis* has been shown to produce a cell-detaching factor that causes detachment of cultured mammalian cells and likely the sloughing of vaginal epithelial cells seen in clinical disease⁹.

The amount of cell-detaching factor produced by the flagellates appeared to correlate with the severity of the clinical infection and therefore be a virulence marker in *T. vaginalis* pathogenesis. Experimental evidence also suggests that the symptoms of trichomoniasis may be influenced by the vaginal concentration of estrogens; the greater the concentration the less severe the symptoms⁹. So it is the aim of the study was to detect the level of sex hormones (Estrogen, Progesterone, and Testosterone) in women infected with *T. vaginalis*.

MATERIALS AND METHODS

It was selected blood samples from 30 women infected with *T. vaginalis* and 30 other blood samples from non-infected women (control), therefore from October 2014 to April 2015 in Babylon hospitals and private laboratories in Babylon province. Some information was taken from patients such as name, age, address, Is a regular menstrual cycle? In any phase of the month? What type of contraceptive user found that? The presence of thyroid disease or not?, Date of sampling. Have been estimated concentration of the three hormones (estrogen, progesterone, testosterone) in the blood serum samples of women previously saved degree (-20 m) using a Mini VIDAS device.

Measure the level of estrogen in the blood serum

It was measured the concentration of estrogen in the blood serum using the Mini VIDAS device with kit ready-analysis of this hormone by following the instructions supplied with the kit for hormone estrogen³.

Measure the level of progesterone in the blood serum

It was measured the concentration of progesterone in the blood serum using the Mini VIDAS device with kit ready-analysis of this hormone by following the instructions supplied with the kit for hormone progesterone³.

Measure the level of testosterone in the blood serum

It was measured the concentration of testosterone in the blood serum using the Mini VIDAS device with kit ready-analysis of this hormone by following the instructions supplied with the kit for hormone testosterone³.

Statistical analysis

The result has been counted by Qui – square under significance level ($p < 0.05$) to compare between infection rate¹.

RESULTS AND DISCUSSION

There are a lot of studies on several aspects specific to incidence of this parasite, but we did not find any study on the relationship between the incidence of this parasite and influence in sex hormones for females only laboratory studies (*in vitro*) specific to certain hormones, but not all, so the conditions of this study and samples vary all those studies, so consider this study is the first concerning this aspect.

The results showed that there are significant differences in estrogen rate among women infected and non-infected where the estrogen rate for infected women is highest 1.43 than non-infected women¹, and the p value 0.02 below the level 0.05 statistic as in Table(1) and Figure (1), The studies on the effect of hormones differ in parasite *T. vaginalis* in women with it, since the hormones are responsible for regulating the growth of organisms such as cellular differentiation and physiological functions and others so Recently, research is being done with the effects of hormones on the host parasites.

The role of estrogen in influencing the parasite is unclear and controversial, According to an earlier study¹⁵ the estrogen decrease the growth of parasite, and their attachment to mammalian cells *in vitro*, and acted as a chemo repellent, therefore this study didn't agree with the results of the current study. While the results of the current study agreed with the findings of the researcher⁷. Who has studied the relationship between the estrogen and *T. vaginalis in vitro*, which he found the high concentrations of 17 β -Estradiol and Estriol promoted the reproduction of *T. vaginalis*, the researcher instructed the reason for

this situation that 17- β -Estradiol changes in pH and make it suitable for the growth of the parasite, and that agreed with this findings study. As for the Estriol, it has not changed in pH, So the reason may instructed researcher in increased parasite growth at the high concentration of this hormone is that it affects directly in its own hormone receptors on the surface of the parasite, which may play an important role in the growth and reproduction of the parasite, as previously proposed by Ford *et al*⁵.

The results also agreed with the findings of Martinotti and Savoia¹⁰ who found that the 17- α –Estradiol, 17- β -Estradiol. They were motivated for the growth of the parasite. As the results also agreed with Markel and Voges⁹, who suggested that the greater the concentration of estrogen increased the lack of unity of symptoms, the researcher instructed the reason for this situation that 17- β -Estradiol reduce the effectiveness of Cell Detaching Factor (CDF).

From this we conclude by the findings of the study that there is a positive correlation between estrogen and *T. vaginalis*, a greater incidence of parasite increased secretion of the estrogen may be defensive as a means to reduce the symptoms of the parasite.

As for the hormone progesterone results of the statistical analysis showed that there were significant differences between the hormone progesterone rate for women infected and non-infected, Where the hormone increased the rate in infected women 6.56 for non- infected 3.71, and the p value 0.04 below the level 0.05 statistic as in Table (1) and Figure (2). And therefore present study results did not agree with Martinotti and Savoia¹⁰. That progesterone inhibits *T. vaginalis* growth *in vitro*. Baeten *et al*⁴., found that women who use contraception which enters progesterone in composition reduce their incidence of *T. vaginalis*, and therefore did not agree with the results of the current study, The reason may be due to that not in all cases the concentration of hormones that affect the incidence of the parasite as can sometimes be the parasite is able to produce a change in the concentration of the hormone to a host through a change to one of the immune response of the gland endocrine host paths after infection parasite⁸.

Results of statistical analysis also showed no significant differences in testosterone levels between women infected and non-infected women with the parasite, Where it was mentioned the hormone for infected women rate 0.06 and for and non-infected women rate 0.03, and the p value 0.24 below the level 0.05 statistic as in Table (1) and Figure (3). The reason may be due to the low concentration of this hormone in women for being a male sex hormone.

Table (1): Changes in hormonal indicators in women with *Trichomonas vaginalis*

Hormones		Number	mean	Std. Error Mean	P value
E ₂	Patients	30	*1.43	11.86	0.02
	Control	30	1	8.45	
PRG	Patients	30	*6.56	1.24	0.043
	Control	30	3.71	0.59	
TES	Patients	30	0.41	0.06	0.24
	Control	30	0.5	0.03	

Figure (1): The difference in estrogen concentration rate among women infected and non-infected with *Trichomonas vaginalis*

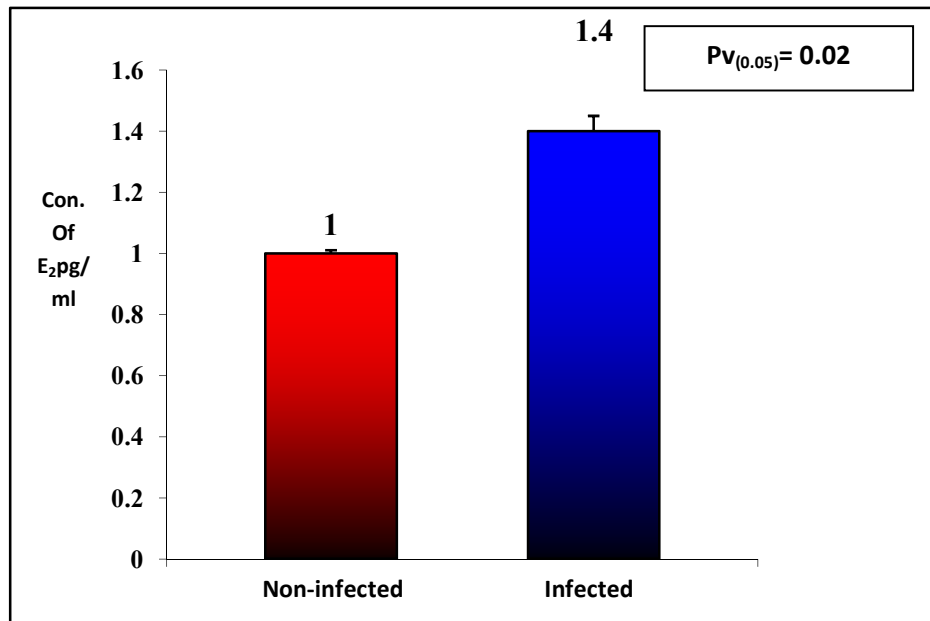


Figure (2): The difference in progesterone concentration rate among women infected and non-infected with *Trichomonas vaginalis*

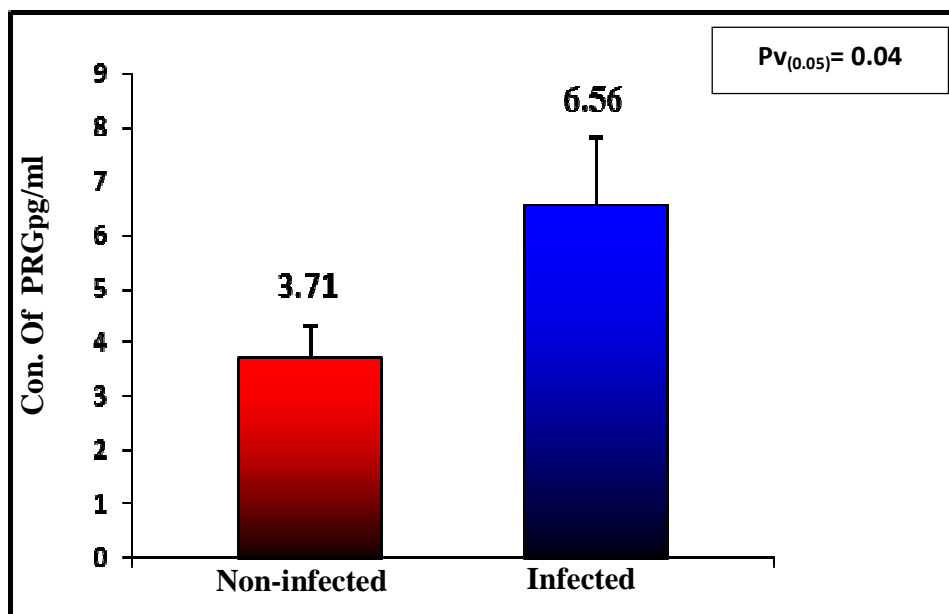
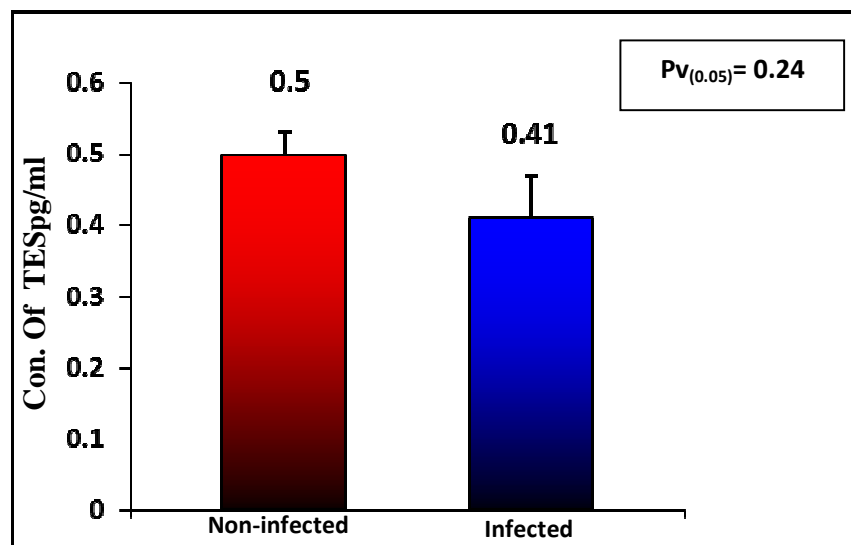


Figure (3): The difference in progesterone concentration rate among women infected and non-infected with *Trichomonas vaginalis*



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