

Assessment of Lipid Profile and Cytokine (IL-13, TGF- β) with sICAM-1 in Patients Infected with Intestinal Parasite (*Entamoeba histolytica*, *Giardia lamblia* and *Blastocystis hominis*)

Esra I. Wahid-Aldeen*, Neama A. Ahmad, Sahlah Kh. Abbas

Department of Biology, College of Science, University of Kirkuk.

*Corresponding Author: Esra I. Wahid-Aldeen

Abstract

Tests were assessed in sera of 150 patients infected with intestinal parasites of *E.histolytica* (50 case), *Giardia lamblia* (50 case) and *Blastocystis hominis* (50 case), as well as 20 apparently healthy controls whom attended to Children Hospital , Kirkuk General Hospital And Azadi teaching Hospital in Kirkuk city-Iraq, from February- August 2017. Biochemical parameters include a lipid profile, while immunological parameters include serum level of IL-13, TGF- β and soluble intracellular adhesion molecule-1 (sICAM-1) . Biochemical parameters showed a significant correlation between low serum lipid levels in infected intestinal parasites especially in the level of cholesterol was significantly decreased in patients infected with *G.lamblia* (107.36 ± 10.82 mg/dl) and *E.histolytica* (110.88 ± 9.45 mg/dl) compared to control group and patients infected with *B.hominis* ($P \leq 0.05$) . The level of T.G. and VLDL also showed significantly decreased in patients infected with *E.histolytica* (57.52 ± 8.73 mg/dl) compared to control and patients infected with *B.hominis*, while all patients showed a significant decrease in HDL level. Serum level of IL-13 and sICAM-1 were significantly increased in all patients infected with intestinal parasite, while there was a significantly increased serum level of TGF- β in patients infected with *B.hominis* and *E.histolytica*, the current study conclude that intestinal parasite work to consumed fat from the host because it cannot synthesize it by itself , and this result suggest a role of IL-13 pathogenesis of intestinal parasitic infection ,also the increase in the level of TGF- β and sICAM-1 in *B.hominis* patient may suggest that TGF- β causes increased expression of endothelial ICAM-1 and neutrophil-mediated intestinal injury.

Keywords: *Entamoeba histolytica*, *Giardia lamblia*, *Blastocystis hominis*, sICAM-1, IL-13, TGF- β , Lipid Profile.

Introduction

Intestinal parasitic infections caused by parasitic protozoa are one of the main causes of health problems worldwide, especially in tropical and subtropical countries. About 3.5 billion people with intestinal parasites and about 450 million have clinical symptoms [1].

A number of intestinal parasites have been reported in different parts of the world such as *Giardia lamblia*, *Dientamoeba fragilis*, *Entamoeba histolytica*, *Blastocystis homini*, *Isospora belli*, *Cyclospora cayetanensis*. However, *E.histolytica* and *G.lamblia* parasites are the major parasites in global public health [2] the common symptoms associated with the infection included abdominal discomfort, vomiting, and diarrhea [3], but in severe cases may cause

several complications such as feeling distressed, bloating, Fatigue, malnutrition, malabsorption, intestinal ulcers, gastroenteritis, weight loss, cysts, and even death [4] Cholesterol is one of the main components of cellular membranes, which plays a key role in regulating the cell membrane, dynamically, functionally and categorically.

The relationship of cholesterol levels in the serum of a man with parasites has drawn the attention of many researchers. Especially after a laboratory study, and concluded that *G.lamblia* and , *E.histolytica* could grow in high-fat media in the absence of serum [5]. Furthermore, studies have shown high levels of lipoproteins such as high-density

lipoprotein (HDL), low-density lipoprotein (LDL) and total cholesterol in patients with malaria infections [6] The interleukin-13 (IL-13) is an important cytokines in regulating immune response and Inflammatory, secondary structural characteristics IL-13 are similar to that of IL-4 [7, 8, 9], the central regulator of IgE synthesis, and excessive mucus secretion, as well as one of the mediators of allergy reaction [8], with a number of studies indicated that the cystic fibrosis has a role in causing skin allergies Urticarial [10, 11, 12] reported in an exploratory study of 92 children with chronic allergies, 5.4% of whom had parasitic infections, including Giardia and Cystic fibrosis.

Transforming growth factor- β (TGF- β) is a protein produced by a large number of immune cells. Its primary functions include the regulation of inflammatory processes, especially in the gut [13]. TGF- β plays a crucial role in stem cell differentiation, T cell differentiation [14, 15]. Numerous researches have been done on this cytokines, especially in autoimmune diseases and infectious diseases [16]. Indicate that TGF- β is an important factor in controlling the amoebic infection by modifying and regulating the function of the Mononuclear cells (MN). In a study, the researchers concluded that parasites can induce T-cells to secrete high levels of TGF- β [17].

While soluble intracellular adhesion molecular (sICAM-1) is one of the intracellular adhesion molecules found in low concentrations, expressed by several cell types, including white blood cells and endothelial cells, and play an important role in both autoimmune and acquired immune responses, and enhance.

The migration of white blood cells across transendothelial tissues to inflammation sites [18, 19]. Accordingly, the present study aimed to determine the serum level of total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL), low density lipoprotein (LDL) and very low density lipoprotein (VLDL) together with the assessment levels of IL-13, TGF- β and sICAM-1, in a sample of intestinal parasitic patients.

Material and Methods

Subject

The study was carried out on 150 patients infected with intestinal parasites, who were attended to Children Hospital, Kirkuk General Hospital And Azadi teaching Hospital for diagnosis and treatment during the period February –August 2017, after laboratory investigation direct wet mount and clinical examination, the patients were distributed into three group which were infected with *E.histolytica* (50 case), *Giardia lamblia* (50 case) and *Blastocystis hominis* (50 case) and their age range between (6 months-55 years) year, as well as 20 apparently healthy controls matched patients for age and gender were enrolled in the study.

Assessment of Lipid Profile and IL-13, TGF- β AND ICAM-1 Serum Level

Serum of all patients and healthy control were assessment for biochemical test included lipid profile by using enzymatic method, and for immunological testes included levels of IL-13, TGF- β and sICAM-1 by ELISA method using commercially available (kits Elabscience, china).

Statistical Analysis

All parameters were analyzed using the statistical package for social science (SPSS) version 13. The data were given as Mean \pm S.E. and Duncan's test used to assess differences between means, the differences were considered significant when probability value was ≤ 0.05 .

Results and Discussion

Biochemical Parameters

The results in Table (1) showed a decrease in the level of cholesterol in those infected with *E.histolytica* and *Giardia* compared with the control group, so that the difference was statistically significant (Probability = 0.04). However, there were no significant differences between patients with *B.hominis* and control group (P = 0.67).

When comparing the level of cholesterol among the patients infected with studied parasites, the results showed a significant reduction in the serum level of patients infected with *E.histolytica* and *G.lamblia* compared patients infected with *B.hominis* ($p \leq 0.05$). The level of triglycerides in patients infected with *E.histolytica* decreased significantly (57.52 ± 8.73 pg/ml) compared

with control group (probability = 0.03), and with those infected with *E.histolytica* compared to those infected with the *B.hominis* (69.6 ± 18.22 pg/ml), (probability ≤ 0.05), While level of HDL ,the results showed a decrease in HDL levels in all patients above

compared with control group ,and numerical reduction in the levels of LDL in all patients . On the other hand the infection with the *E.histolytica* lead to the reduction in the level of VLDL (P=0.02) compared with control group and with *B.hominis* infection

Table 1: Mean sera level of lipid profile in patient infected with intestinal parasites and controls

Parameters		Mean \pm S.E.(mg/dl)		Blastocystis
		<i>E.histolytica</i>	Giardia	
TC	Patients	110.88 \pm 9.45	107.36 \pm 10.82	144.1 \pm 19.53
	Control		135.5 \pm 19.53	
	P.V.	0.04	0.04	0.67
TG	Patients	57.52 \pm 8.73	63.63 \pm 5.72	69.6 \pm 18.22
	Control		77.25 \pm 4.15	
	P.v.	0.03	0.08	0.6
HDL	Patients	26.4 \pm 2.9	25.39 \pm 2.16	24.05 \pm 2.98
	Control		40.75 \pm 2.05	
	P.v.	0.006	0.001	0.003
LDL	Patients	57.46 \pm 55	60.9 \pm 6.9	66.9 \pm 12.66
	Control		70.25 \pm 4.51	
	P.v.	0.1	0.3	0.8
VLDL	Patients	11.78 \pm 1.68	13.41 \pm 1.62	29.65 \pm 11.25
	Control		18.3 \pm 1.6	
	P.v.	0.02	0.06	0.056

Different letters indicate a significant difference (P \leq 0.05) between Mean in one row, similar letters represent to no significant differences (P \geq 0.05) between these means

The present study, showed a clear reduction in the level of lipid with *G.lambliia* and *E.histolitica* infection. The result agree with [5] who pointed out that parasites used lipid to grow inside the host and recorded a decrease in the level of cholesterol ,HDL and TG in Giardiasis and Amoebiasis patients.

Parasites get fat and HDL cholesterol from the upper part of the intestines. However, the mechanisms followed by parasites to do so are unclear so far, and many scientific references have shown a change in the lipid profile in most patients with parasites, especially in those inflammations [20] and during Encystation, cholesterol is consumed by the parasite because it cannot be synthesized by itself [21] While the level of TG and HDL were normal in the same patients, because *G. lambliia* consumed the cholesterol of the host in the biosynthesis of the cell wall and the parasite is unable to synthesis cholesterol by itself , as well as the TG and HDL do not affect the growth of parasite and that the level of LDL and VLDL depends mainly on the level of cholesterol [22] The results of this study were agreed with [22 23], who recorded a decrease in blood cholesterol level in individuals with *G. lambliia* infection, In a recent study, [24] reported a reduction in blood cholesterol level in the case of *G.lambliia* parasites, which use

cholesterol in the cellular membrane structure, they pointed out that *G.lambliia* infection affects fat standards. It causes poor absorption of fat in the intestines, for the loss of fat with stool, causing Steatorrhea, in addition to causing low levels of HDL, with a significant increase in levels of triglycerides and LDL. When *E.histolitics* colonizes is in the bowel, the level of cholesterol absorption in the intestines decreases, while serum cholesterol is depleted in the case of hepatic abscess produced by amoebic infection [25] noted a decrease in the level of triglycerides in patients with *B.hominis*, although there were no significant differences, while the level of cholesterol increased slightly in the case of the infected and compared to non-infected, and this result agreed with the findings of the current study.

In addition, cholesterol is a catalyst for growth of amoeba in laboratory experiment , when serum was replaced with cholesterol which resulted in heavy growth in the medium .In contrast, the trophozoite stage of *G.lambliia* can be stimulated to encystation by starving the parasite of cholesterol by using lipoprotein-deficient serum [22, 23, 26].

Immunological Parameters

Cytokines

As show in the Table (2), the mean level of IL-13 was significantly higher in sera patients infected with intestinal parasites included: *E.histolytica*, giardia and *B.hominis* compared with control group. TGF- β also showed a significant increase in the serum level of patients infected with *E.histolytica*

and *B.hominis*. While there was a significant difference in the Mean serum level of TGF- β among the patients infected with studied parasites. On the other hand there were significant increases in the serum level of sICAM-1 in all patients compared with control group.

Table 2: Mean sera level of IL-13, TGF- β and sICAM-1 in patient infected with intestinal parasite and control

Parameters		Mean \pm S.E. (mg/dl)		Blastocystis
		<i>E.histolytica</i>	Giardia	
IL-13	Patients	490.10 ^a \pm 96.4	158.60 ^a \pm 29.4	421.11 ^a \pm 67.2
	Control		53.6 \pm 8.4	
	P.V.	0.0005	0.004	0.0001
TGF- β	Patients	7.55 ^b \pm 6.34	0.59 ^c \pm 0.17	13.18 ^a \pm 3.14
	Control		2.04 \pm 1.25	
	P.V.	0.01	0.2	0.006
sICAM-1	Patients	4879.08 ^b \pm 254.1	5376 ^{ab} \pm 313.5	5729.5 ^a \pm 436.8
	Control		3839.87 \pm 173.6	
	P.V.	0.002	0.0006	0.001

Different letters indicate significant difference ($P \leq 0.05$) between Mean in one row, Similar letters represent to no significant differences ($P \geq 0.05$) between these means

The results of the current study were agreed with [27], which recorded a significant increase in the concentration of IL-13 in patient infected with *G.lamblia*, also the results of the current and consistent with [28], who recorded on mice CBA were infected with *E.histolytica* and the level of IL-13 increased statistically significantly as compared to control group with three days.

In another study [27] to determine the level of IL-13 in patients infected with Giardia and compare with control group ,the result showed in increased significantly in the level of IL-13 (6 fold) in the case of *G.lamblia* infection(42.37 \pm 27.79 pg/ml) in compere with healthy people (7.00 \pm 3.91 pg/ml) and this level did not reduced when used anti-parasites in the serum of patients (41.41 \pm 31.07 pg/ml).

The IL-13 is characterized by inflammatory conditions and allergies. IL-13 plays a key role in intestinal infections and as an essential part of the immune response in the intestinal mucosa. It acts as a catalyst for Goblet cells in the intestinal mucosa for the production of substances which in turn attracts esinophile and thus increases the production and secretion of IgE. IL-13 also increases peristalsis and increases the secretion of Epithelia [9, 27, 29]. Many clinical studies deal with the relationship between parasites and symptoms linked with skin allergic ,in spite of that some parasites is one of the activated factor to produce IgE [12, 30].

The study by [31] proved that 38.8% from infected person with *E.coli* , *G.lamblia* and *B.hominis* are suffering from urticarial in computation with 1.1% of control, the serum level of IgE increased 4 time in patients infected with protozoa ,which they suffering from allergy in compare with these which they do not infected with parasite .the study [32] in Saudi Arabic established that 13.7% of samples from patients having a chronic allergic and skin infection , were infected with *B.hominis*.the result of [11] observed 92 children with chronic allergies between the ages of 4-15 years, 5.4% of whom were infected with parasitic infections, including *B.hominis* and Giardia. This explains why IL-13 concentration elevated in patients with giardia And *B.hominis*.

However, [10] which stimulates parasite antigens to activate specific copies of Th₂ lymphocytes causing the production of specific cellular cytokines such as IL-3, IL-4, IL-5, and IL-13, leading to Isotype Switching from IgM to IgE production. This study recorded elevated level of TGF- β which consistent with [21], which showed a significant increase in the concentration of TGF- β in people who had symptoms of parasitic infection compared to control groups.the researches [33] concluded that the IL-10 and TGF- β were associated with development of *E.histolytica* infection as it has an important role in activating the cellular immune response ,leading to the appearance symptoms. In another study

[16], mononuclear cells (MN) were exposed to *E.histolytica* then treated with TGF- β which stimulated leukophagocytosis. The low level of TGF- β in *G.lambli*a patients may explain by the fact that it is non-invasive parasite, and does not cause congenital malformations in the gastrointestinal tract of the intestinal tract, but it reduces the surface area of absorption causing diarrhea [34, 37].

The results of the current study agree with [38], which recorded a significant increase in cytokines TGF- β in the case of *B.hominis* infection, [17] indicated that parasites can induce T cells to secrete high ratios of cytokines TGF- β , since *B.hominis* colonizes the intestine, making it in constant contact with mucosal immune factors. TGF- β is an important mediator in the immune system of the mucosa. It plays a key role in the activation of immune cells [39].

The adhesion molecule sICAM-1 are present in normal cases with low concentrations on the surface membrane of white blood cells and epithelial tissue cells, when inflammation occurs in the epithelial tissue where it is stimulated and concentration greatly increased; it is important to interfere with other cellular and cellular inter-inflammatory receptors, the WBC migrates to the site of infected tissues and interacts with ICAM-1 molecules resulting in ICAM-1 / LFA-1 [18, 40]. In addition to its large role in facilitating the transmission of immune and inflammatory cells during tissue [41].

The high concentration of the ICAM-1 adhesion molecules can be explained by the increased inflammatory process. The gene responsible for the coding of ICAM-1 molecules is associated with responses to inflammatory processes. It was found that

mice lacking a concentration of ICAM-1 molecules had a weak response to inflammation in the case of pulmonary diseases [4 42]. However, its role remains unclear in the case of intestinal parasites, and may be considered as markers of activation of epithelial cells in localized and systemic infections [4].

Relationship between the Studied Variables

In the study of the correlation between all studied variables in each parasite, (Table 9) shows a positive relationship between TGF- β concentration with triglycerides and VLDL in the case of *E.histolytica* ($P \leq 0.05$). Literary texts did not mention a relationship between TGF- β and the serum lipid level. The results showed a positive relationship between TG and VLDL in the case of *E.histolytica* ($P \leq 0.05$), and in the case of *G.lambli*a ($P \leq 0.001$), VLDL is mainly composed of triglycerides and 55% , And therefore any change in the concentration of the latter causes a change in focus directly ⁴³The results showed a significant correlation between HDL and LDL ($P \leq 0.05$) in *G.lambli*a parasites.

The link between the HDL and the LDL may be due to the fact that HDL consumption by *G.lambli*a led to a decrease in serum concentration, accompanied by increased LDL concentration due to lack of consumption by parasite. In the case of *B.hominis* infection, LDL was shown to be cholesterol - related with LDL ($P \leq 0.05$) [44]. LDL is composed of 55% cholesterol and is responsible for the transfer of cholesterol from cells to the liver. The TGF- β concentration showed a positive association with IL-13 in the serum. This result was consistent with [45], where TGF- β was reported to enhance IL-13 function.

Table 3: Relationship between the studied immunological variables

Samples studied	Correlation coefficient	R	p-value
<i>E.histolytica</i>	Triglyceride	1.000	0.000
	VLDL		
	Triglyceride	0.897	0.039
	TGF- β		
	VLDL	0.897	0.039
	TGF- β		
Giardia	HDL	0.960	0.010
	LDL		
Blastocystis	Cholesterol	0.954	0.046
	LDL		
	IL-13	0.984	0.016
	TGF- β		

Conclusions

The study showed low levels of fat, especially cholesterol and HDL in cases of parasites studied, where parasites use fat in the bio-construction of their cell membrane. The current study showed a close relationship between the high concentration of cytokines IL-13 and TGF- β . Among the cases of

intestinal parasites studied, the immune response to parasites. The current study showed a correlation between the high concentration of IL-13 and TGF- β , indicating a correlation between their functions. Also the correlation between TGF- β level and serum levels of lipid in intestinal parasitic infection sufficient to clear the protozoa.

References

1. Who (2000) World Health Organization. Intestinal Parasites, Available at: <http://www.who.int/ctd/intpara/burdens.htm>. 2000.
2. Abdullah I, Tak H, Ahmad F (2016) Predominance of Gastrointestinal Protozoan Parasites in Children: A Brief Review. *J. Heal. Educ. Res Dev.*, 04(04): doi:10.4172/2380-5439.1000194
3. Schunk M, Jelinek T, Wetzel K, Nothdurft HD (2001) Detection of *Giardia lamblia* and *Entamoeba histolytica* in stool samples by two enzyme immunoassays. *Eur. J. Clin. Microbiol. Infect. Dis.*, 20(6):389-391. doi:10.1007/PL00011279
4. Yao FR, Qiao JY, Zhao Y, Zhang X, Yang JH, Li XQ (2005) Experimental infection of mice with *Blastocystis hominis*. *Zhongguo ji sheng chong xue yu ji sheng chong bing za zhi= Chinese J. Parasitol. Parasit Dis.*, 23(6):444-448.
5. Bansal D, Bhatti HS, Sehgal R (2005) Altered lipid parameters in patients infected with *Entamoeba histolytica*, *Entamoeba dispar* and *Giardia lamblia*. *Br. J. Biomed. Sci.*, 62(2):63-65. doi:10.1080/09674845.2005.11732686
6. Faucher J-F, Ngou-Milama E, Missinou M, Ngomo R, Kombila M, Kremsner PG (2002) The impact of malaria on common lipid parameters. *Parasitol. Res.*, 88(12):1040-1043.
7. Zurawski G, de Vries JE (1994) Interleukin 13, an interleukin 4-like cytokine that acts on monocytes and B cells, but not on T cells. *Immunol Today*, 15(1):19-26.
8. Rael EL, Lockey RF (2011) Interleukin-13 signaling and its role in asthma. *World Allergy Organ J.*, 4(3):54.
9. Seyfizadeh N, Seyfizadeh N, Gharibi T, Babaloo Z (2015) Interleukin-13 as an important cytokine: A review on its roles in some human diseases. *Acta Microbiol. Immunol. Hung.* 62(4):341-378.
10. Pasqui AL, Savini E, Saletti M, Guzzo C, Puccetti L, Auteri A (2004) Chronic urticaria and *blastocystis hominis* infection. A case report. *Eur. Rev. Med. Pharmacol. Sci.*, 8:117-120.
11. Chansakulporn S, Pongpreuksa S, Sangacharoenkit P, et al (2014) The natural history of chronic urticaria in childhood: a prospective study. *J. Am Acad. Dermatol.*, 71(4):663-668.
12. Lepczyńska M, Chen W, Dzika E (2016) Mysterious chronic urticaria caused by *Blastocystis* spp.? *Int. J. Dermatol.*, 55(3):259-266.
13. Letterio JJ, Roberts a B (1998) Regulation of immune responses by TGF-beta. *Annu Rev Immunol.*, 16(1):137-161. doi:10.1146/annurev.immunol.16.1.137
14. Li MO, Flavell RA (2008) TGF- β : A Master of All T Cell Trades. *Cell*, 134(3):392-404. doi:10.1016/j.cell.2008.07.025
15. Massagué J, Xi Q (2012) TGF- β control of stem cell differentiation genes. *FEBS Lett.*, 586(14):1953-1958.
16. Moraes LCA, França EL, Pessoa RS, et al (2015) The effect of IFN- γ and TGF- β in the functional activity of mononuclear cells in the presence of *Entamoeba histolytica*. *Parasites and Vectors*, 8: 1. doi:10.1186/s13071-015-1028-6
17. Correale J, Farez M (2007) Association between parasite infection and immune responses in multiple sclerosis. *Ann Neurol.*, 61(2):97-108.
18. Yang L, Froio RM, Sciuto TE, Dvorak AM, Alon R, Luscinskas FW (2005) ICAM-1 regulates neutrophil adhesion and transcellular migration of TNF- α -activated vascular endothelium under flow. *Blood*, 106(2):584-592. doi:10.1182/blood-2004-12-4942
19. Lawson C, Wolf S (2009) ICAM-1 signaling

- in endothelial cells. *Pharmacol. Rep.*, 61(1):22-32. doi:10.1016/S1734-1140 (09) 70004-0
20. Lauwaet T, Davids BJ, Reiner DS, Gillin FD (2007) Encystation of *Giardia lamblia*: a model for other parasites. *Curr. Opin. Microbiol.*, 10(6):554-559.
 21. Bansal D, Sehgal R, Chawla Y, Malla N, Mahajan RC (2005) Cytokine mRNA expressions in symptomatic vs. asymptomatic amoebiasis patients. *Parasite Immunol.*, 27(1- 2):37-43.
 22. Maani N, Jabir DM (2017) Study of lipid profile alteration in the patients infected with *Giardia lamblia* and compare the results with healthy individuals. *AL-Qadisiyah Med. J.*, 9(15):119-129.
 23. Luján HD, Mowatt MR, Nash TE (1997) Mechanisms of *Giardia lamblia* differentiation into cysts. *Microbiol Mol. Biol. Rev.*, 61(3):294-304.
 24. Alhuchaimi SN, Mahmood TA, Abdullateef SF, Khadum EJ (2017) Association between Serum Cholesterol Level and *Giardia lamblia* Infection among Children with Acute Diarrhea in Al-Najaf Governorate. *kufa J. Nurs. Sci.*, 7(1):41-46.
 25. Chen T-L, Chan C-C, Chen H-P, et al (2003) Clinical characteristics and endoscopic findings associated with *Blastocystis hominis* in healthy adults. *Am J. Trop. Med. Hyg.*, 69(2):213-216.
 26. Gillin (1996) Cell biology of the primitive eukaryote *Giardia lamblia*. *Annu Rev Microbiol.* 50(1):679-705.
 27. Matowicka-Karna J, Kralisz M, Kemona H (2011) Assessment of the levels of nitric oxide (NO) and cytokines (IL-5, IL-6, IL-13, TNF, IFN- γ) in giardiasis. *Folia Histochem Cytobiol.*, 49(2):280-284.
 28. Guo X, Stroup SE, Houpt ER (2008) Persistence of *Entamoeba histolytica* infection in CBA mice owes to intestinal IL-4 production and inhibition of protective IFN- γ . *Mucosal Immunol.*, 1(2):139-146. doi:10.1038/mi.2007.18
 29. Mannon P, Reinisch W (2013) Republished: Interleukin 13 and its role in gut defence and inflammation. *Postgrad Med J.*, 89(1054):448-456.
 30. Lynch NR, Hagel IA, Palenque ME, et al (1998) Relationship between helminthic infection and IgE response in atopic and nonatopic children in a tropical environment. *J Allergy Clin Immunol.*, 101(2):217-221.
 31. Farthing MJG (2003) Immune response-mediated pathology in human intestinal parasitic infection. *Parasite Immunol.*, 25(5):247-257.
 32. Farooq M, Khodari Y, Zagloul D, Othman RAM (2011) Prevalence of intestinal parasites and bacteria among food handlers in a tertiary care hospital. *Niger Med J.*, 52(4):266. doi:10.4103/0300-1652.93802
 33. García- Zepeda Ea, Rojas- López A, Esquivel- Velázquez M, Ostoa- Saloma P (2007) Regulation of the inflammatory immune response by the cytokine/chemokine network in amoebiasis. *Parasite Immunol.*, 29(12):679-684.
 34. Fahmy ZH, Aly E, Mohamed AH (2014) The effect of medium chain saturated fatty acid (monolaurin) on levels of the cytokines on experimental animal in *Entamoeba histolytica* and *Giardia lamblia* infection. *African J. Pharm. Pharmacol.*, 8(4):106-114.
 35. Faubert G (2000) Immune response to *Giardia duodenalis*. *Clin Microbiol Rev.*, 13(1):35-54, table of contents. doi:10.1128/CMR.13.1.35-54.2000
 36. Eckmann L (2003) Mucosal defences against *Giardia*. *Parasite Immunol.*, 25(5): 259-270. doi:10.1046/j.1365-3024. 2003. 00634.x
 37. Scott KGE, Yu LCH, Buret AG (2004) Role of CD8+ and CD4+ T lymphocytes in jejunal mucosal injury during murine giardiasis. *Infect Immun.*, 72(6):3536-3542. doi:10.1128/IAI.72.6.3536-3542.2004
 38. Chan KH, Chandramathi S, Suresh K, Chua KH, Kuppusamy UR (2012) Effects of symptomatic and asymptomatic isolates of *Blastocystis hominis* on colorectal cancer cell line, HCT116. *Parasitol Res.*, 110(6):2475-2480.
 39. Town T, Laouar Y, Pittenger C, et al (2008) Blocking TGF- β -Smad2/3 innate immune signaling mitigates Alzheimer-like pathology. *Nat. Med.*, 14(6):681.
 40. Stanciu LA, Djukanovic R (1998) The role of ICAM-1 on T-cells in the pathogenesis of asthma. *Eur. Respir J.*, 11(4):949-957.
 41. Fang FC (1997) Perspectives series: host/pathogen interactions. Mechanisms of nitric oxide-related antimicrobial activity.

J. Clin Invest., 99(12):2818-2825.

42. Chen CF, Wu KG, Hsu MC, Tang RB (2001) Prevalence and relationship between allergic diseases and infectious diseases. J. Microbiol Immunol Infect Wei mian yu gan ran za zhi., 34(1):57-62.
43. Hughes J, Jefferson JA (2008) Clinical Chemistry Made Easy E-Book. Elsevier

Health Sciences.

44. Burtis CA, Ashwood ER, Bruns DE (2012) Tietz Textbook of Clinical Chemistry and Molecular Diagnostics-e-Book. Elsevier Health Sciences.
45. Wynn TA (2003) IL-13 effector functions. Annu. Rev. Immunol., 21(1):425-456.