The Korean Journal of Parasitology
Vol. 35, No. 1, 47-54, March 1997

Effects of anti-IgE mAb on serum IgE, FcεRII/CD23 expression on splenic B cells and worm burden in mice infected with *Paragonimus westermani*

Myeong-Heon SHIN* and Hong-Ki MIN

Department of Parasitology, College of Medicine, Ewha Womans University, Seoul 158-056, Korea

Abstract: It is generally accepted that parasite-specific IgE plays a crucial role in host defense against helminthic parasites. However, the role of high levels of nonspecific IgE in helminthic infections is still controversial. To investigate the role of nonspecific IgE in primary infections with *P. westermani*, the effect of anti-IgE mAb treatment on serum IgE, FcεRII/CD23 expression and worm burden in *Paragonimus*-infected mice were examined. In mice treated with anti-IgE antibody, the total IgE levels were not detectable (1 μg/ml) throughout the experiment compared with untreated infected mice. The mean percentages of FcεRII/CD23 positive splenic B cells in anti-IgE treated mice (range: 20.3 - 30.5) were also decreased throughout the experiment compared with untreated infected mice (range: 35.7 - 44.4). Reduction of the total IgE and expression of FcεRII/CD23 on splenic B cells resulted in decreased worm burden six weeks post infection. These results suggest that high levels of nonspecific IgE in mice with primary infections of *P. westermani* play a harmful, rather than beneficial, role for the host, perhaps by interfering with CD23-dependent cellular pathways.

Key words: anti-IgE mAb, FcεRII/CD23, IgE, worm burden, *Paragonimus westermani*

INTRODUCTION

Helminth infections have been characterized by highly elevated plasma levels of total IgE (Radermerker *et al.*, 1974). It is generally recognized that the IgE antibodies play an essential role in host defense against helminthic parasites. The high schistosome-specific IgE levels recently have been associated with host protection (Hagan *et al.*, 1991). In addition, *in vitro* IgE antibodies directly damaged or killed the parasitic worms through macrophage and eosinophils mediated cytotoxicity (Auriault *et al.*, 1984; Capron *et al.*, 1984). The IgE dependent cellular cytotoxicity to parasitic worms was associated with the IgE binding activity of FcεRII/CD23 on macrophages and eosinophils (Capron *et al.*, 1986) and was significantly correlated with the levels of parasite-specific IgE (Pestel *et al.*, 1988). These data imply that cross-linking between parasite-specific IgE and parasite antigen in helminthiasis is very important in mediating CD23-dependent cellular cytotoxicity against parasites. However, since the parasite-specific IgE constitutes less than 10% of the total IgE in helminthic infections (Rousseaux-Prevost *et al.*, 1978), it is unknown whether *in vitro* cytotoxicity to parasitic worms occurs similarly *in vivo*.

• This study was supported in part by NON DIRECTED RESEARCH FUND, Korea Research Foundation, 1996 (#04 F 0110) and by the grant No. 95-85 from the Ewha Womans University Promotion Fund (1995).

*Corresponding author (e-mail: mhshin@mm.ewha.ac.kr).

Recently, it was hypothesized that high levels of nonspecific IgE may be protective for the parasite since IgE molecules would occupy the available FcεRIIs on CD23 bearing cells (Pritchard, 1993).

In murine paragonimiasis, the high levels of total IgE in sera were shown (Min *et al.*, 1993) to be similar to other helminthic infections. Also IgE plays a major role in antibody dependent macrophage-mediated cytotoxicity (ADCC) *in vitro* on metacercariae of *P. westermani* (Min *et al.*, 1990). However, little has been reported about the role of nonspecific IgE in protective immunity in mice infected with *P. westermani*. In this study, the effect of anti-IgE mAb treatment on IgE production, expression of FcεRII (CD23) on splenic B cells and worm burden in *P. westermani*-infected mice are reported.

MATERIALS AND METHODS

Parasites and experimental animals

Metacercariae of *P. westermani* were separated from crayfish, *Cambaroides similis*, collected at Wando-gun, Chollanam-do, Korea. Four to six week old BALB/c female mice were inoculated orally with 20 metacercariae. Age-matched mice were used as non-infected controls.

Administration of anti-IgE monoclonal antibody

The 4-6-week old mice were treated intraperitoneally with either 100 μg anti-IgE mAb (R35-72, rat IgG1, Pharmigen, CA) or PBS (control). Next day, all of the mice were inoculated orally with 20 metacercariae of *P. westermani*. Subsequently, the infected mice were retreated weekly for 6 weeks intraperitoneally with 50 μg anti-IgE mAb or PBS.

Sera

Blood was collected from the retro-venous plexus of mice. Sera were stored for IgE measurement at -20°C until it was assayed.

ELISA for total and parasite-specific IgE

Total IgE levels in sera were measured by capture ELISA similarly as previously described by Shin *et al.* (1991). In brief, 2 μg/ml of monoclonal rat anti-mouse IgE (Pharmigen, CA, R35-72) were coated on 96 well microtiter plates. Then the plates were washed 3 times, diluted test sera (1:500 in dilution buffer) and standard IgE (Pharmigen, CA) added to each well, and then incubated at 37°C for 1 hr. After incubation, the plates were washed, 2 μg/ml biotinylated monocloanl rat anti-mouse IgE (Pharmigen, R35-92) added to each well, and incubated again at 37°C for 45 min and then washed. Finally, avidin peroxidase (Pharmigen, 1:1,000 in dilution buffer) was added to each well and the plates were incubated at 37°C for 25 min followed by washing and the addition of the developing substrate 3,3′ 5,5′ -tetramethylbenzidine base (TMB, Gibco, NY). The amounts of total IgE (μg/ml) were calculated by reference to standard curves ($Y = 1.4 X + 0.115$, $r = 0.98$). The assay was sensitive to 1 μg/ml. In addition, the parasite-specific IgE titers were measured by conventional ELISA of using metacercarial excretory-secretory antigens. In brief, 50 μg/ml metacercarial excretory-secretory antigens were coated on 96 well microtiter plates. Then the plates were wahsed 3 times with PBS/0.5% Tween 20 and incubated for 2 hours at 37°C with PBS/3% BSA. 1:50 dilution of each serum added to each well and the plates were incubated for 1 hour at 37°C. After incubation, the plates were washed, 0.5 μg/ml biotinylated anti-mouse IgE mAb (Pharmigen, R35-92) added to each well, and incubated for 45 min at 37°C. Final steps were same as described above. Data were expressed as OD at 450 nm. All tests were done in duplicate.

Flow cytometry

Spleens were removed from anti-IgE mAb or PBS treated mice at 2, 3, 4 and 6 weeks after infection with *P. westermani*. Splenocytes were dispersed with a 1 ml syringe and washed with PBS. 1×10^6 cells were double stained with 4 μl of PE-conjugated rat anti-mouse CD45R/B220 (RA3-6B2, rat IgG_{2a}, Pharmigen, CA) and FITC-conjugated rat anti-mouse CD23 mAbs (B3B4, rat IgG_{2a}, Pharmigen, CA) for 30 min at 4°C. Splenocytes from non-infected mice were used for reference B cell expression

of CD23. Cells were washed and the florescence intensity measured by a flow cytometer (Becton Dickinson, CA). Splenic lymphocytes were gated to remove dead cells and debris using forward and side light scatter.

Quantitation of worm burden

Five mice treated with either anti-IgE mAb or PBS were sacrificed at 2, 3, 4 and 6 weeks post-infection. The total body was macerated in normal saline and then incubated for 2 hours at 37°C. The worms were recovered by stereomicroscopy and then counted.

Statistical analysis

The Student's t-test was used to determine the significance difference between treated and control groups.

RESULTS

Total IgE and *P. westermani*-specific IgE responses

Total serum IgE levels in anti-IgE treated mice were inhibited significantly ($P < 0.05$) to undetectable levels (< 1 μg/ml) during the 6-week course of infection compared with infected mice treated with PBS which demonstrated increased serum IgE levels from the third (13.7 μg/ml) to sixth (46.1 μg/ml) week after infection (Fig. 1). In comparison, the serum IgE levels of non-infected mice were < 1 μg/ml throughout the study. In addition, *P. westermani*-specific IgE titers (O.D.) for infected mice treated with anti-IgE were reduced (0.16) significantly ($P < 0.05$) at the sixth week of infection compared with infected mice treated with PBS which exhibited elevated specific IgE titers (0.34) (Fig. 2).

CD23 (FcεRII) expression on splenic B lymphocytes

The mean percentages of FcεRII/CD23 positive splenic B cells was decreased significantly ($P < 0.05$) during the experiment (range: 20.3 - 30.5) compared with infected mice treated with PBS (range: 35.7 - 44.4) (Table 1). Also, the flourescent intensity of FcεRII/CD23 in infected mice treated with anti-IgE was decreased compared with infected mice treated with PBS (Fig 3). The mean percentages of FcεRII/CD23 positive splenic B cells in non-infeced mice ranged from 26.1 to 33.6.

The number of worms recovered *in vivo*

The number of worms recovered from infected mice treated with anti-IgE (range: 14.0 ± 1.73 - 16.0 ± 2.00) were similar compared to infected mice treated with PBS (range: 14.5 ± 0.05 - 17.0 ± 1.41) for weeks 2-4 post-infection. However, the number of worms recovered in infected mice treated with anti-IgE (8.8 ± 2.22) were significantly decreased ($P < 0.05$) by 40% 6 weeks post infection

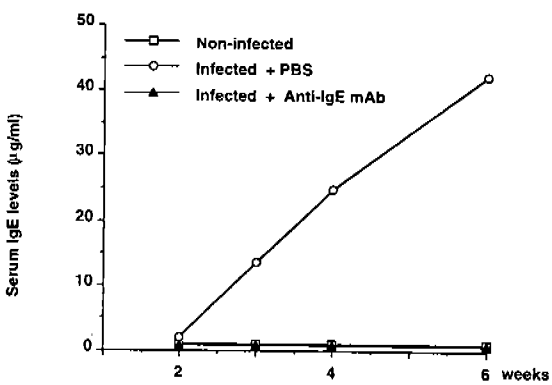

Fig. 1. Effect of anti-IgE mAb on total serum IgE levels in BALB/c mice infected with *P. westermani*. Total serum IgE levels were determined by ELISA using monoclonal rat anti-mouse IgE.

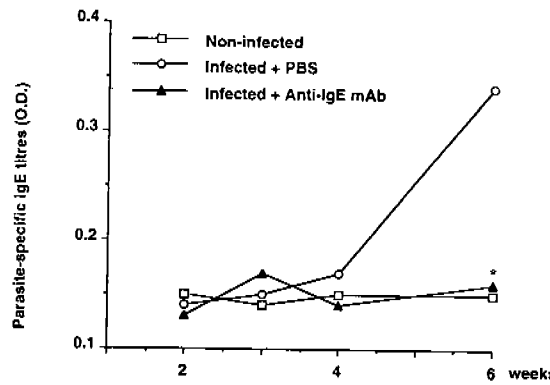

Fig. 2. Effect of anti-IgE mAb on parasite-specific IgE levels in BALB/c mice infected with *P. westermani*. Absorbance (O.D.) was measured at 450 nm. Asterisk (*) denotes significant difference ($P < 0.05$) between infected mice treated with PBS and infected mice treated with anti-IgE mAb.

Table 1. Effect of anti-IgE on the percent (%) of FcεRII/CD23 positive splenic B cells in mice infected with *Paragonimus westermani*

	2 w	3 w	4 w	6 w
Non-infected mice	26.1(3.18)[a]	33.6(2.91)	30.7(5.94)	31.1(3.77)
Infected mice treated with PBS	43.4(7.52)[b]	44.4(2.99)[b]	35.7(3.29)	38.9(4.96)
Infected mice treated with anti-IgE	30.5(1.28)[c]	20.3(0.25)[c]	25.4(2.62)[c]	29.7(3.45)[c]

[a]Each value represents the mean (SD), n = 5

[b]Significant ($P < 0.05$) increase compared with non-infected mice

[c]Significant ($P < 0.05$) decrease compared with infected mice treated with PBS

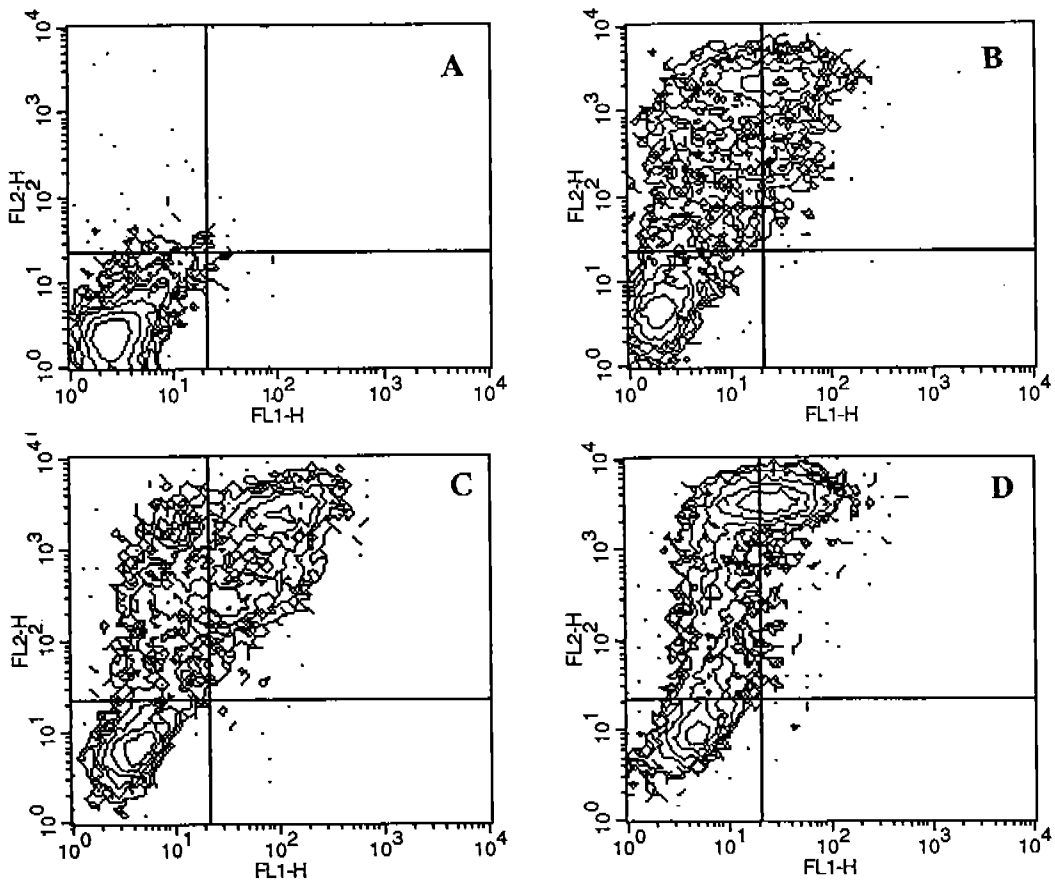

Fig. 3. Suppressive effect of anti-IgE on the expression of FcεRII/CD23 on splenic B cells in mice infected with *P. westermani*. FL1-H and FL2-H indicate green fluorescence (CD23) and red fluorescence (CD45R/B220), respectively. Results are shown as two color contour plots (**A**, autofluorescence; **B**, non-infected mice; **C**, infected mice treated with PBS; **D**, infected mice treated with anit-IgE mAb).

compared with infected mice treated with PBS (15.0 ± 2.00) (Table 2).

DISCUSSION

This study shows that anti-IgE mAb treatment decreases total serum IgE levels,

Table 2. Effect of anti-IgE on the number of worms recovered in mice infected with *Paragonimus westermani*

	2 w	3 w	4 w	6 w
Infected mice treated with PBS	15.7(3.16)[a]	14.5(0.05)	17.0(1.41)	15.0(2.00)
Infected mice treated with anti-IgE	16.0(2.00)	13.3(2.04)	14.0(1.73)	8.8(2.22)[b]

[a]Each value represents the mean (SD), n = 5

[b]Significant ($P < 0.05$) decrease compared with infected mice treated with PBS

expression of FcεRII/CD23 on splenic B cells and worm burden in mice infected with *Paragonimus westermani*. The total serum IgE levels in anti-IgE mAb treated mice were markedly less after 3 weeks post-infection compared with untreated infected mice which showed ordinarily high serum IgE levels 3-6 weeks post-infection. In addition, parasite-specific IgE titers in anti-IgE mAb treated mice also were decreased significantly ($P < 0.05$) after infection compared with the untreated infected mice. These results suggested that the high levels of nonspecific IgE are suppressed throughout the experiment. Also, the number of excysted metacercariae in infected mice treated with anti-IgE mAb were decreased compared with untreated infected mice. These findings were smilar to those of Amiri *et al.* (1994). *Schistosoma mansoni*-infected mice had undetectable IgE levels after treatment with a high dose of anti-IgE mAb and showed a significant reduction of worm burden and number of eggs produced. They also reported that partial reduction of serum IgE levels had little effect on either parasite burden or the number of eggs produced. This suggests that complete inhibition of the total IgE responses to undetectable levels, rather than a partial reduction in IgE levels, is beneficial for the host. In contrast, Kigoni *et al.* (1986) reported that suppression of IgE antibodies using polyclonal anti-IgE antiserum showed no harmful effect on primary infections of *S. mansoni* infected rats. However, since that study measured only worm-specific IgE responses, it is difficult to identify the exact role of nonspecific IgE in protective immunity.

Unlike tissue helminth infections, including *S. mansoni*, suppression of total IgE in intestinal helminthic infections did not have any effect on worm expulsion (Korenaga *et al.*, 1991). These discrepancies between tissue and intestinal helmithic infections were likely due to different protective mechanisms. There were no differences in the worm expulsion between normal and congenitally IgE-deficient mice infected with *Nippostrongylus brasiliensis* or *Trichinella spiralis* (Watanabe *et al.*, 1988). These results indicate that the IgE antibodies are not essential for worm expulsion in intestinal helminthic infections.

The parasite specific IgE responses seemed to coincide with the migratory phase of live worms in *Paragonimus ohirai*-infected rats (Ikeda & Fujita, 1980). The parasite specific and total serum IgE responses arised at the same time in rat fascioliasis (Pfister *et al.*, 1983). However, the parasite-specific IgE antibodies in this study were detectable three weeks after the increase of total serum IgE. This delayed detection of parasite specific IgE responses were prabably due to using metacercarial antigens instead of adult antigens in conventional ELISA. According to Reiner & Zahner (1986), the different patterns of a specific IgE responses against cercarial and adult antigens in conventional ELISA were observed in rat schistosomiasis. Using cercarial antigens, the specific IgE antibodies were increased 2-3 weeks after the rise in total serum IgE. It is suggested that larva antigens have lower antigenicity to detect the parasite specific IgE than adult antigens. However, it is not exclusively precluded low sensitivity of conventional ELISA. Using different methods for the determination of specific IgE, it is at least expected certain degree of overlapping of parasite specific and total serum IgE responses.

It also has been shown that FcεRII/CD23 on B cells is involved in IgE synthesis and antigen presentation to T cells (Kehry & Yamashita, 1989; Flores-Romo *et al.*, 1993). In this study, the percentages of FcεRII/CD23 positive

splenic B cells in mice infected with *P. westermani* significantly increased 2-3 weeks post-infection compared to untreated non-infected mice. The kinetic changes of the expression of FcεRII/CD23 in this study correlated with those of IL-4 production by splenocytes in *Paragonimus*-infected mice as previously reported by Shin & Min (1996). In contrast, this increased expression on splenic B cells was not shown in anti-IgE mAb treated mice throughout the study. These results were similar to previous reports (Haak-Frendscho *et al.*, 1994). They observed that the administration of anti-IgE antibody inhibits CD23 expression and IgE production *in vivo*. These results suggest that the expression of FcεRII/CD23 on B cells plays a central role in the production of IgE.

The mechanisms resulting in decreased worm burden is unknown. Recently, Pritchard (1993) proposed two mechanisms suggesting that the presence of excess non-parasite IgE could play a detrimental role, rather than beneficial, for the host. The first mechanism is likely due to decreased IgE-dependent cellular cytotoxicity by high levels of nonspecific IgE. Eosinophils and macrophages posses the type B isoforms of FcεRII/CD23 (Delespesse *et al.*, 1992). The specific linkage between parasite specific IgE and FcεRII/CD23 on eosinophils and macrophages is crucial to the process for arming the effector cells. Therefore, the high levels of nonspecific IgE prevents effector cells from being sufficiently armed to kill or damage the parasites by saturating its IgE receptors. However this theory has been remained disputable in murine models. According to Jones *et al.* (1994), IgE receptors (FcεRII/CD23) on eosinophils are deficient in *Toxocara*-infected mice, murine eosinophils do not kill helminth larvae by an IgE dependent mechanism. Further studies on IgE-dependent eosinophil cytotoxicity to helminthic worms in murine models should be needed. The second mechanism, which may be ill influenced by nonspecific IgE, is the cytophilic binding of IgE to type A isoforms of FcεRII/CD23 on B cells. These cells have been demonstrated to be involved in IgE-dependent antigen focussing and presentation to T cells (Pirron *et al.*, 1990; Fujiwara *et al.*, 1994). The complete suppression of high levels of nonspecific IgE in helminthic infection is likely to afford the B cells favorable condition, thereby activating the B cells to present specific antigens to T cells through specific ligation of FcεRII on B cells. Because IgE molecules stabilize membrane FcεRII/CD23 on cells (Kawabe *et al.*, 1988), cross linking of CD23 antigen by its natural ligand (IgE) prevents B lymphocyte poliferation and differentiation (Luo *et al.*, 1991).

Vouldoukis *et al.* (1995) observed that the killing of *Leishmania major* by human macrophages was mediated *in vitro* by nitric oxide induced after specific ligation of the FcεRII/CD23 surface antigen. Also, the immune macrophages showed an IgE-dependent schistosomulicidal activity *in vitro* in the presence of parasite-specific IgE-rich serum (Pestel *et al.*, 1988). While, the effector cells are capable of killing the parasites *in vitro*, it is not clear whether the cellular cytotoxicity occurs *in vivo* since the amonut of parasite-specific IgE was very slight among the total IgE. This may be the reason why many tissue helminths survive for a long time in primary infection. In this study, we only measured the low affinity receptors for IgE expressed on B cells. Therefore, the precise mechanism of IgE-dependent macrophage or eosinophil cytotoxicity to parasitic worms *in vivo* remains unknown. The association between high levels of nonspecific IgE and effector cells *in vivo* shoud be further investigated.

In conclusion, our experiment suggests that high levels of non-parasitic IgE in *Paragonimus*-infected mice play a harmful role for the host, perhaps by interfering with CD23-dependent cellular pathways.

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=초록=

폐흡충 감염 마우스에 있어 Anti-IgE 단일크론 항체 처치시 혈청내 총 IgE, 비장 B 세포표면의 FcεRII/CD23 발현 및 충체수에 미치는 영향

신명헌, 민흥기

이화여자대학교 의과대학 기생충학교실

윤충 감염시 증가되는 기생충 특이 IgE 항체는 항기생충 작용을 하는 것으로 알려져 있으나 총 IgE 항체의 90% 이상을 차지하는 비특이성 IgE 항체의 역할에 대해서는 잘 알려져 있지 않다. 이 실험에서는 폐흡충 감염 마우스에 anti-IgE 단일크론 항체를 처치한 후 혈청내 IgE, 비장 B 세포의 FcεRII/CD23의 발현 및 충체수를 관찰하여 비특이성 IgE가 숙주의 방어면역에 미치는 역할을 알아보았다. Anti-IgE를 처치한 폐흡충 감염 마우스의 혈청내 총 IgE 항체의 양은 실험 기간 동안 억제되어(< 1 μg/ml) PBS로 처치한 감염군에 비해 유의한 수준($P < 0.05$)으로 감소되었다. 또한 Anti-IgE를 처치한 폐흡충 감염 마우스에서의 FcεRII/CD23 양성인 비장 B 세포수 및 그 발현도 PBS로 처치한 감염군에 비해 실험 기간 동안 유의한 수준($P < 0.05$)으로 감소되었다. 충체 회수율에 있어서도 anti-IgE를 처치한 감염 마우스는 6주에 44%로 PBS로 처치한 감염군(75%)에 비해 유의한 수준($P < 0.05$)으로 감소하였다. 이상의 결과를 종합할 때 폐흡충 감염 마우스에서 높게 증가되는 비특이성 IgE는 숙주의 방어면역을 방해하여 오히려 충체를 보호하는 역할을 하는 것으로 생각된다.

(기생충학잡지 35(1): 47-54, 1997년 3월)