Published in final edited form as:

Ann Allergy Asthma Immunol. 2009 October ; 103(4): 342–347. doi:10.1016/S1081-1206(10)60535-2.

Differences in total and allergen-specific IgE during pregnancy compared to 1 month and 1 year postpartum

Lee M. Perry, MD¹, Dennis R. Ownby, MD¹, Ganesa R. Wegienka, PhD², Edward L. Peterson, PhD², Kimberly J. Woodcroft, PhD², L.M. Joseph, PhD², and Christine C. Johnson, PhD²

¹Medical College of Georgia, Section of Allergy and Immunology, 1120 15th Street BG 1019, Augusta, GA 30912

²Department of Biostatistics and Research Epidemiology, Henry Ford Hospital, 1 Ford Place, Detroit, MI 48202, USA

Abstract

Background—Pregnancy alters the function of many body systems, including the immune system. However, little is known regarding the affects of pregnancy on maternal immunoglobulin E (IgE) levels or atopy.

Objective—To determine whether pregnancy consistently influences serum levels of total- or allergen-specific IgE.

Methods—Blood samples were obtained from 470 women during the third trimester of pregnancy and 1 month post partum. A third sample was obtained from 103 of these women 1 year post partum. Samples were analyzed for total and specific IgE to 8 regionally common allergens using the Phadia UniCAP system. Sensitization was defined as an allergen-specific IgE 0.35 kU/L to any allergen.

Results—Total IgE increased significantly postpartum, both at 1 month (34.7 versus 39.8 IU/ml, $P = 0.001$) and at 1 year (38.0 versus 46.4 IU/ml, $P = 0.01$). Allergen-specific IgE decreased significantly at 1 month for cat, dog, cockroach, ragweed, timothy grass, *Alternaria* and egg ($P = 0.001$ – 0.012), but not for dust mite (*D. farinae*, $P = 0.569$). Similar patterns of change in total and

Corresponding Author: Dennis R. Ownby, M.D., Betty B. Wray Professor of Pediatrics and Professor of Medicine, Medical College of Georgia, 1120 15th Street, BG 1019, Augusta, GA 30912-3790, Phone: 706-721-3531, Fax: 706-721-2527, downby@mcg.edu.

Contribution of each author to the study:

1. Lee M. Perry, M.D. was the principal author of the submitted manuscript (leperry@mail.mcg.edu).
2. Dennis R. Ownby M.D. was involved in the design and conduct of the study. He supervised all IgE measurements presented (downby@mcg.edu).
3. Ganesa R. Wegienka Ph.D. was involved in the design and conduct of the WHEALS study, supervises WHEALS data collection, reviewed and revised the current manuscript (gwegien1@hfh.org).
4. Edward L. Peterson Ph.D. is a senior biostatistician who performed or checked all data analyses and who reviewed and revised the current manuscript (EPETERS1@hfh.org).
5. Kimberley J. Woodcroft Ph.D., MPH was involved in the design and conduct of the WHEALS study, especially the laboratory processing of all biologic samples and reviewed and revised the current manuscript (kwoodcr1@hfh.org).
6. Christine L. M. Joseph Ph.D., MPH is an epidemiologist involved in all aspects of the design and conduct of the WHEALS study and who reviewed and revised the current manuscript (CJOSEPH1@hfh.org).
7. Christine C. Johnson Ph.D., MPH is the senior epidemiologist who conceived and obtained funding for the WHEALS study and who reviewed and revised the current manuscript (CJOHNSO1@hfh.org).

specific IgE were seen at 1 year. However, on average, only 3.5% of subjects changed sensitization status to the individual allergens studied over the 1 year of observation.

Conclusions—Compared to intrapartum levels, total IgE levels increased significantly at one month and one year postpartum. Conversely, at the same time points, IgE levels specific for common allergens significantly declined to most but not all allergens. Few women changed their sensitization status over one year.

Keywords

total IgE; allergen-specific IgE; pregnancy

INTRODUCTION

Normal pregnancy causes profound changes in almost every female organ system in order to meet the physiologic needs of both the fetus and the placenta. For example, maternal cardiac output increases by 30 to 50 percent during normal pregnancy.^{1–3} In addition, maternal plasma volume increases by 10–15 percent at six to twelve weeks gestation, and then continues to expand until approximately 34 weeks gestation.^{4–6}

In order to prevent rejection of the developing fetus, significant immunologic and anatomic adaptations must occur within both the mother and the fetus.^{7–8} Fetal trophoblast cells protect the developing embryo from potentially harmful components of the maternal immune system. HLA class I expression on trophoblast cells is strictly regulated, most likely to prevent maternal immune cells from attacking foreign (paternal) HLA class I antigens.⁸ Trophoblast cells also express high levels of membrane cofactor protein (CD46) and decay accelerating factor, which prevent complement activation and possible destruction of fetal cells.⁹ Tolerance to fetal tissue is enhanced by placental production of various immunosuppressive molecules such as progesterone, prostaglandin E2, and anti-inflammatory cytokines (e.g. interleukin-10 and interleukin-4).^{8,10} In the uterus, T and B cell numbers decrease, while specialized natural killer (NK) cells and macrophages become more predominant.⁸ All together, these adaptations result in a maternal-fetal interface characterized by normal innate immune responses and diminished adaptive immunity.

The effect of pregnancy on asthma symptoms and severity has been evaluated in numerous studies. Pregnancy has a variable effect on asthma symptoms.^{11–15} It is generally accepted that asthma symptoms improve in one third of pregnant women, worsen in one third of women, and are unchanged in another third.^{11–14} While it is difficult to predict the effect of pregnancy on an individual woman's asthma, there is evidence to suggest that women with milder asthma are less likely to worsen during pregnancy than those with severe asthma.^{11, 14–16} In contrast, the question of whether pregnancy could affect maternal allergic status or IgE levels has not been well studied. Amino et al.¹⁷ measured serum levels of immunoglobulins at various times during pregnancy for eleven women and reported statistically significant decreases in IgG, IgA and IgM, but not for IgE, in the second and third trimesters of pregnancy. The authors of this study attributed the decrease in immunoglobulin concentration to a combination of hemodilution and immune suppression. They did not follow immunoglobulin levels after delivery. A more recent study evaluated the effect of pregnancy on cytokine production (IL-1 β , IL-6, IL-10, and IL-12) in response to stimulation of various arms of the immune system.¹⁸ The authors of this study incidentally observed that in allergic women (as defined by clinical history and skin prick testing), total IgE levels were significantly lower two years after pregnancy compared to intrapartum levels. However, Abraham et al.¹⁹ demonstrated a 54% prevalence of seroatopy (one or more allergen-specific IgE $\geq$ 0.35 kUa/L) in a large cohort of pregnant females (the

same cohort as the present study), which was similar to the prevalence of all women having at least one positive skin test response (49.2%) in the NHANES III study.²⁰ When these findings are considered together, it appears less likely that pregnancy significantly alters IgE production.

The objective of this study was to determine whether pregnancy influences serum levels of total or allergen-specific IgE using an ethnically mixed population-based cohort of women. We also examined whether these IgE levels were affected by ethnicity or the presence of self-reported allergic disease (asthma or allergic rhinitis).

METHODS

Data for this study were collected as part of the Wayne County Health, Environment, Allergy, and Asthma Longitudinal Study (WHEALS) being conducted in Wayne County, MI (Detroit and western suburbs), as previously described.¹⁹ Participants were women, ages 21–49 years, in the second trimester of pregnancy or later. Serum was obtained from all subjects during the third trimester of pregnancy, then at approximately one month and one year after delivery. Study recruitment continued over four years with approximately equal numbers of serum samples being obtained in each month of the year. The samples were analyzed for total IgE and for specific IgE to eight regionally common allergens: *Dermatophagoides farinae* (dust mite), cat, dog, *Blattella germanica* (German cockroach), *Ambrosia artemisiifolia* (ragweed), *Phleum pratense* (timothy grass), hens' egg, and *Alternaria alternata*. Measurements of allergen-specific IgE were performed using the Phadia UniCAP system (Phadia AB, Portage, Michigan). Seroatopy was defined as having at least one allergen-specific IgE ≥ 0.35 kUa/L to any given allergen. The specific IgE assays were able to measure IgE to a level of 0.1 kUa/L. Undetectable levels of specific IgE were assigned a value of 0.05 kUa/L for purposes of computing geometric mean IgE values. There were no undetectable total IgE values.

Statistical analysis was performed on data from 764 women comparing IgE levels in the third trimester and one month postpartum. For comparison between the third trimester and one year postpartum, a random sample of 106 subjects was examined. At the time of recruitment, each woman was interviewed regarding her asthma and allergy history. The women were subsequently classified according to the presence of asthma (self-reported, physician-diagnosed) in the past twelve months and allergic rhinitis (self-reported, physician-diagnosed) during their lifetime. Due to non-normality of total and specific-IgE distributions, geometric means were calculated. Comparisons from one time period to the next were done using nonparametric tests: the Wilcoxon matched-pairs signed-rank test and the Friedman test. Because of missing data the numbers of individuals shown in each table are not always identical. Approval for this study was obtained from both the Henry Ford Health System and the Medical College of Georgia human rights committees, and written informed consent was obtained from all participants.

RESULTS

Total IgE measurements were performed on the serum of 764 women while the number of specific IgE measurements varied slightly because of insufficient serum volume for testing all allergens. Third trimester serum samples were collected at an average of 1.3 months prior to delivery with an interquartile interval of 0.6–1.9 months. Samples for one month postpartum were collected at an average of 1.2 months after delivery with an interquartile interval of 0.9–1.6 months. The results of comparison between the third trimester and one month postpartum are shown in Table 1. At the population level, total IgE increased a small but statistically significant amount from late in pregnancy to one month after delivery (35.37

to 40.36 IU/mL, $P = 0.001$). In contrast to the total IgE results, analysis of allergen-specific IgE revealed statistically significant decreases at one month postpartum for all allergens ($P = 0.001$ – 0.018) except *D. farinae* ($P = 0.903$), cockroach ($P = 0.735$), and *Alternaria* ($P = 0.149$).

A random sample of 106 women were included in the comparison of total and allergen-specific IgE between the third trimester and one year postpartum (Table 2). Samples for one year postpartum were collected at an average of 13.8 months after delivery with an interquartile interval of 12.8–14.5 months. There was a statistically significant increase in total IgE over this time period ($P = 0.005$). However, all allergen-specific IgEs were statistically significantly decreased over the interval, $P = 0.001$ – 0.011 , except for IgE specific to timothy grass, $P = 0.070$.

Despite the statistically significant decreases in allergen-specific IgE to the eight common allergens tested, there was only a small percentage (2.1–7%) of subjects who changed sensitization status to an individual allergen (Table 3). Most of the changes in sensitization status were due to small changes in specific IgE, and may have been due to variation of the assay. Larger changes in specific IgE were less frequent—only 6 of 28 changes were of more than 1 kUa/L. These findings were not significantly affected by race (data available upon request). Interestingly, the women with self-reported histories of asthma (Table 4) and allergic rhinitis (Table 5) were less likely to demonstrate significant increases in total IgE and decreases in specific IgE.

DISCUSSION

To our knowledge, this is the first study examining the possible effect of pregnancy on total and allergen-specific IgE levels in a large, multiracial cohort of women who were not selected for allergic disease. Compared to intrapartum levels, our data demonstrate a statistically significant increase in total IgE levels at one month and one year after delivery. In contrast to the total IgE results, analysis of allergen-specific IgE revealed statistically significant decreases at both one month and one year after delivery for nearly all allergens. While these changes were statistically significant, they are unlikely to be clinically important since IgE sensitization status, either sensitized or not sensitized, changed in <5% of women for all allergens except cockroach (7%), and only 2 of the 106 women developed new sensitivities.

It is likely that the total IgE during the third trimester appears low in comparison to postpartum levels because of the hemodilution seen during pregnancy. During the third trimester, maternal plasma volume increases by 1100–1600 mL.^{4,6} This results in a total plasma volume of approximately 4700–5200 mL, which is 30–50% higher than that found in nonpregnant women. After delivery, plasma volume is still elevated by 10–15% three weeks after delivery, and decreases to normal nonpregnant levels at approximately six weeks postpartum. When these estimated plasma volume changes are applied to our data, the difference in total IgE before and one month after delivery is negligible. However, the decline in allergen-specific IgE concentrations after delivery seen in our data argues against plasma volume contraction as the potential mechanism for the increase in total IgE postpartum. We do not think that the decline in specific IgEs is a result of multiple testing or statistical error since the decline was consistently seen in all but one of the allergens. It may be that pregnancy causes a gradual decline in specific IgE over time, but we could not find any additional studies to support this speculation.

Amoudruz et al.¹⁸ investigated the possible effect of pregnancy on cytokine production in response to stimulation of the innate and adaptive immune systems. This study also

evaluated total IgE levels in allergic and nonallergic pregnant women during the third trimester, at delivery, and two years after delivery. The classification of “allergic” was based on clinical history and skin prick testing (skin test considered positive if the wheal diameter after 15 minutes was ≥ 3 mm). Interestingly, the total IgE of allergic women was significantly lower two years after delivery than during pregnancy. This is in contrast to the results of our study, in which total IgE increased at both one month and one year in comparison with intrapartum levels. The women in our study with a self-reported history of asthma and/or allergic rhinitis were less likely to demonstrate an increase in total IgE after delivery.

The possible impact of maternal IgE level on offspring has been studied. Liu et al.²¹ found that maternal total IgE correlates with elevated infant IgE levels and the subsequent development of infantile atopic dermatitis. The prominent effect of maternal atopy on cord blood IgE has also been well-described in the literature.^{22–23} Furthermore, Bertino et al. recently demonstrated a positive correlation between maternal and cord blood specific IgE to cow’s milk protein.²⁴ Interestingly, there are studies that suggest a decline in maternal IgE levels with increasing order of live offspring.²⁵ This assertion is strengthened by several studies which have demonstrated a decreased risk of childhood atopic disease with larger family size and higher birth order.²⁶

There are numerous strengths to our study. Our cohort is comprised of a large, diverse population of women. The women included were relatively unselected in that they were not recruited at an allergy clinic, health fair, or similar event. Another strength of our study is the geographic limitation of the participants, who resided in a group of contiguous ZIP codes in the western portion of Wayne County. Thus, their environmental exposures were probably similar, as were their exposures to seasonal changes.

Our study had several limitations. Only pregnant women were studied and followed up to a year after pregnancy. Due to the limited geographic area studied, it is unknown whether the results obtained apply to women in other regions of the country. It might have also been valuable to have been able to apply more consistent diagnostic criteria for asthma and allergic rhinitis than that provided by self-reported histories. Inclusion of a control group of non-pregnant women living in the same area would have allowed estimates of the normal variation in IgE levels. However, this was far beyond the scope of the parent study.

Finally, it is not known whether the changes in total and allergen-specific IgE over time were accompanied by changes in clinical status. If further studies are performed to assess whether pregnancy alters IgE levels, it would be helpful to relate such findings to clinical history.

In summary, our data demonstrate a statistically significant increase in total IgE levels at one month and one year after delivery when compared to the levels during pregnancy. Conversely, the average level of allergen-specific IgEs tested decreased significantly after pregnancy. Despite the variations observed in total and allergen-specific IgE after pregnancy, there was only a small percentage of subjects who changed sensitization status to a particular allergen. Thus, while these changes were statistically significant, they are unlikely to be clinically important. Further studies are needed to better describe the potential clinical implications of these findings.

Acknowledgments

Funding Sources include NIH grants: AI59415, AI50681, AI61774 and the Fund for Henry Ford Hospital.

REFERENCES

1. Katz R, Karliner JS, Resnik R. Effect of a natural volume overload state (pregnancy) on left ventricular performance in normal human subjects. *Circulation*. 1978; 58:434. [PubMed: 679433]
2. Capeless EL, Clapp JF. Cardiovascular changes in early phase of pregnancy. *Am J Obstet Gynecol*. 1989; 161:1449. [PubMed: 2603897]
3. Van Oppen AC, Stigter RH, Bruinse HW. Cardiac output in normal pregnancy: a critical review. *Obstet Gynecol*. 1996; 87:310. [PubMed: 8559545]
4. Lund CJ, Donovan JC. Blood volume during pregnancy: significance of plasma and red cell volumes. *Am J Obstet Gynecol*. 1967; 98:394. [PubMed: 5621454]
5. Bernstein IM, Ziegler W, Badger GJ. Plasma volume expansion in early pregnancy. *Obstet Gynecol*. 2001; 97:669. [PubMed: 11339913]
6. Pritchard JA. Changes in blood volume during pregnancy and delivery. *Anesthesiology*. 1965; 26:393. [PubMed: 14313451]
7. Poole JA, Claman HN. Immunology of pregnancy: implications for the mother. *Clin Rev Allergy Immunol*. 2004; 26:161. [PubMed: 15208462]
8. Hunt JS. Stranger in a strange land. *Immunol Rev*. 2006; 213:36. [PubMed: 16972895]
9. His BL, Hunt JS, Atkinson JP. Differential expression of complement regulatory proteins on subpopulations of human trophoblast cells. *J Reprod Immunol*. 1991; 19:209. [PubMed: 1713970]
10. Denison FC, Kelly RW, Calder AA, Riley SC. Cytokine secretion by human fetal membranes, decidua and placenta at term. *Hum Reprod*. 1998; 13:3560. [PubMed: 9886550]
11. Nelson-Piercy C. Asthma in pregnancy. *Thorax*. 2001; 56:325–328. [PubMed: 11254828]
12. Schatz M, Harden K, Forsythe A, et al. The course of asthma during pregnancy, postpartum, and with successive pregnancies: a prospective analysis. *J Allergy Clin Immunol*. 1988; 81:509–517. [PubMed: 3346481]
13. Stenius-Aarniala B, Piirila P, Teramo K. Asthma and pregnancy: a prospective study of 198 pregnancies. *Thorax*. 1988; 43:12–18. [PubMed: 2895502]
14. Schatz M. Interrelationships between asthma and pregnancy: a literature review. *J Allergy Clin Immunol*. 1999; 103:s330–s336. [PubMed: 9949333]
15. Schatz M, Dombrowski MP, Thom EA, et al. Asthma morbidity during pregnancy can be predicted by severity classification. *J Allergy Clin Immunol*. 2003; 112:283–288. [PubMed: 12897733]
16. Gluck JC, Gluck PA. The effects of pregnancy on asthma: a prospective study. *Allergy*. 1976; 37:164–168.
17. Amino N, Tanizawa O, Miyai K, et al. Changes of serum immunoglobulin IgG, IgA, IgM and IgE during pregnancy. *Obstet Gynecol*. 1978; 52:415–420. [PubMed: 714321]
18. Amoudruz P, Minang JT, Sundstrom CN, Lilja G, Troye-Blomberg M, Sverremark-Ekstrom E. Pregnancy, but not the allergic status, influences spontaneous and induced interleukin-1 β (IL-1 β), IL-6, IL-10, and IL-12 responses. *Immunology*. 2006; 119:18–26. [PubMed: 16764689]
19. Abraham CM, Ownby DR, Peterson EL, et al. The relationship between seroatopy and symptoms of either allergic rhinitis or asthma. *J Allergy Clin Immunol*. 2007; 119:1099–1104. [PubMed: 17416408]
20. Arbes SJ, Gergen PJ, Elliott L, Zeldin DC. Prevalence of positive skin test responses to 10 common allergens in the US population: Results from the Third National Health and Nutrition Examination Survey. *J Allergy Clin Immunol*. 2005; 116:377–383. [PubMed: 16083793]
21. Liu CA, Wang CL, Chuang H, Ou CY, Hsu TY, Yang KD. Prenatal prediction of infant atopy by maternal but not paternal total IgE levels. *J Allergy Clin Immunol*. 2003; 112:899–904. [PubMed: 14610477]
22. Kaan A, Dimich-Ward H, Manfreda J, et al. Cord blood IgE: its determinants and prediction of development of asthma and other allergic disorders at 12 months. *Ann Allergy Asthma Immunol*. 2000; 84:37–42. [PubMed: 10674563]
23. Magnusson CG. Cord serum IgE in relation to family history and as predictor of atopic disease in early infancy. *Allergy*. 1988; 43:241–251. [PubMed: 3389491]

24. Bertino E, Bisson C, Martano C, et al. Relationship between maternal- and fetal-specific IgE. *Pediatr Allergy Immunol.* 2006; 17:484–488. [PubMed: 17014621]
25. Karmaus W, Arshad SH, Sadeghnejad A, Twiselton R. Does maternal immunoglobulin E decrease with increasing order of live offspring? Investigation into maternal immune tolerance. *Clin Exp Allergy.* 2004; 34:853–859. [PubMed: 15196270]
26. Karmaus W, Arshad SH, Mattes J. Does the sibling effect have its origin in utero? Investigating birth order, cord blood immunoglobulin E concentration, and allergic sensitization at age 4 years. *Am J Epidemiol.* 2001; 154:909–915. [PubMed: 11700245]

TABLE 1

Geometric Mean Total (IU/mL) and Allergen-Specific IgE (kUa/L) During 3rd Trimester Compared to One Month Postpartum (PP)

Variable	Patients	3 rd trimester mean IgE (95% C.I.)	1-month PP mean IgE (95% C.I.)	<i>P</i> value*
Total IgE	764	35.37 (31.78, 39.36)	40.36 (36.27, 44.91)	0.001
Dust mite	740	0.089 (0.076, 0.104)	0.089 (0.076, 0.104)	0.903
Cat	739	0.066 (0.057, 0.077)	0.062 (0.053, 0.072)	0.001
Dog	741	0.082 (0.072, 0.093)	0.078 (0.068, 0.088)	0.010
Cockroach	739	0.055 (0.049, 0.062)	0.052 (0.046, 0.058)	0.735
Ragweed	739	0.176 (0.151, 0.205)	0.161 (0.138, 0.188)	0.001
Timothy grass	740	0.087 (0.073, 0.104)	0.085 (0.071, 0.102)	0.006
Hens' egg	741	0.054 (0.051, 0.057)	0.049 (0.046, 0.052)	0.018
Alternaria	737	0.116 (0.014, 0.132)	0.108 (0.094, 0.123)	0.149

* Wilcoxon matched-pairs signed-rank test

TABLE 2

Geometric Mean Total IgE (IU/mL) and Allergen-Specific IgE (kUa/L) During Third Trimester Compared to One Month and One Year Postpartum (PP)

Variable	Patients	3 rd trimester [*]	1-month PP [*]	1-year PP [*]	P value ^{**}
Total IgE	106	37.00	40.25	44.97	0.005
Dust mite	99	0.108	0.110	0.084	0.011
Cat	98	0.099	0.082	0.066	0.001
Dog	100	0.115	0.094	0.091	0.007
Cockroach	100	0.060	0.044	0.041	0.001
Ragweed	100	0.224	0.191	0.146	0.001
Timothy grass	99	0.088	0.081	0.069	0.070
Hens' egg	95	0.093	0.065	0.044	0.001
Alternaria	100	0.167	0.128	0.070	0.001

* Geometric mean values differ from those in Table 1 because they are from 95–106 women rather than 739–764 women.

** Friedman's test used to calculate P value comparing 3rd trimester to 1-year postpartum.

TABLE 3

Change in Specific IgE to Individual Allergens from Third Trimester to One Year Postpartum for Women Changing Sensitization Status*

Allergen	Positive to Negative IgE status**			Negative to Positive IgE status**		
	3 rd trimester specific IgE (kUa/L)	1 yr postpartum specific IgE (kUa/L)		3 rd trimester specific IgE (kUa/L)	1 yr postpartum specific IgE (kUa/L)	% changing status at 1 year [‡]
Dust mite	0.65	0.27		0.29	0.56	3.0
	4.44	0.10		-	-	
Cat	0.37	0.26		0.05	0.60	3.1
	-	-		0.19	0.44	
Dog	0.38	0.11		0.31	0.51	3.0
	-	-		0.33	0.46	
Cockroach	0.41	0.14		0.09	0.40	7.0
	0.41	0.28		-	-	
	0.44	0.29		-	-	
	0.46	0.28		-	-	
	0.61	0.31		-	-	
	8.26	0.06		-	-	
Ragweed	0.52	0.05		0.30	1.32	3.0
	0.57	0.35		-	-	
Timothy grass	7.11	0.01		0.15	1.86	3.0
	-	-		0.20	0.48	
Hens' egg	0.44	0.18		-	-	2.1
	0.54	0.04		-	-	
Alternaria	0.36	0.33		0.09	0.04	4.0
	0.37	0.33		-	-	
	4.59	0.01		-	-	
Mean ± SD	1.77 ± 2.54	0.19 ± 0.12		0.20 ± 0.10	0.70 ± 0.47	
Median (IQ range)	0.49 (0.41 – 0.65)	0.22 (0.06 – 0.28)		0.20 (0.09 – 0.31)	0.50 (0.44 – 0.60)	

* Numbers of women tested for each allergen are the same as shown in Table 2.

** Positive to negative or negative to positive allergen-specific IgE status based on an allergen-specific IgE level of 0.35 kU_a/L. The numbers in each column represent the actual values of specific IgE to the listed allergens at the specified times for individual women whose status changed.

[‡] Represents the percentage of women that changed sensitization status to the specified allergen over one year. The percentage change over the year is the sum of those changing from positive to negative and from negative to positive.

TABLE 4

Geometric Mean Total IgE (IU/mL) and Allergen-Specific IgE (kUa/L) During Third Trimester Compared to One Month Postpartum (PP) Stratified by the Presence or Absence of Asthma

Variable	N	3 rd trimester	1-mo PP	<i>P</i> value **
<u>Women With Asthma*</u>				
Total IgE	56	60.04	65.43	0.184
Dust mite	54	0.178	0.168	0.667
Cat	53	0.299	0.254	0.808
Dog	54	0.322	0.260	0.157
Cockroach	54	0.070	0.069	0.333
Ragweed	54	0.271	0.229	0.043
Timothy Grass	54	0.152	0.161	0.258
Hens' egg	53	0.057	0.051	0.619
Alternaria	53	0.271	0.278	0.563
<u>Women Without Asthma</u>				
Total IgE	527	29.84	35.55	0.001
Dust mite	510	0.071	0.073	0.583
Cat	509	0.053	0.049	0.018
Dog	510	0.068	0.066	0.609
Cockroach	510	0.050	0.049	0.772
Ragweed	508	0.173	0.158	0.001
Timothy grass	510	0.086	0.086	0.115
Hens' egg	511	0.053	0.048	0.022
Alternaria	509	0.109	0.099	0.040

* Self-reported history of having a physician diagnosis of asthma within the past year.

** Wilcoxon matched-pairs signed-rank test within strata.

TABLE 5

Geometric Mean Total IgE (IU/mL) and Allergen-Specific IgE (kUa/L) During Third Trimester Compared to One Month Postpartum (PP) Stratified by the Presence or Absence of Allergic Rhinitis*

Variable	N	3 rd trimester	1-mo PP	P value [†]
<u>Women With Allergic Rhinitis**</u>				
Total IgE	119	57.40	68.40	0.001
Dust mite	116	0.119	0.111	0.666
Cat	115	0.208	0.193	0.084
Dog	115	0.193	0.191	0.171
Cockroach	114	0.063	0.066	0.659
Ragweed	115	0.490	0.454	0.001
Timothy Grass	116	0.268	0.256	0.225
Hens' egg	114	0.057	0.051	0.025
Alternaria	113	0.217	0.189	0.193
<u>Women Without Allergic Rhinitis</u>				
Total IgE	537	30.27	34.95	0.001
Dust mite	518	0.079	0.082	0.945
Cat	516	0.054	0.050	0.049
Dog	520	0.070	0.067	0.378
Cockroach	519	0.052	0.049	0.098
Ragweed	518	0.149	0.135	0.039
Timothy grass	518	0.073	0.072	0.012
Hens' egg	521	0.053	0.048	0.489
Alternaria	518	0.113	0.105	0.426

* Differences between number of patients in Table 4 and Table 5 are due to the definitions used for asthma and allergic rhinitis, and that some women had missing data.

** Self-reported history of having a physician diagnosis of allergic rhinitis during their lifetime.

[†] Wilcoxon matched-pairs signed-rank test within strata.