

The effects of alcohol consumption and smoking on allergy risk in Korean adults

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ABSTRACT

Background: The immune-modulating effects of alcohol and smoking are well known through a great body of research. Furthermore, many studies have been performed on the influence of alcohol and smoking on the development of allergic disease. However, previous studies have reported conflicting results with associations between alcohol intake or smoking and risk of allergy. The purpose of this study was to evaluate the effects of alcohol consumption and smoking on total serum immunoglobulin E (IgE) and prevalence of sensitization to *Dermatophagoides farinae* in a representative sample of Korean adults.

Methods: IgE levels and IgE levels specific to *D. farinae* were measured. Alcohol, smoking habit, and other influencing variables were obtained from the 2010 Korean National Health and Nutrition Examination.

Results: Smoking (smoked five packs or more in lifetime) was independently associated with increased risk of sensitization to *D. farinae* (odds ratio [OR], 1.439; 95% CI, 1.075–1.927). Frequent alcohol drinking (more than four times a week) also showed similar association when compared with subjects who drank less than once per month (OR, 2.052; 95% CI, 1.257–3.350). IgE levels were also increased along with increased frequency of alcohol drinking. However, smoking habit was not related to IgE levels.

Conclusion: These findings suggest that history of smoking and frequent alcohol consumption are significantly associated with increased prevalence of sensitization to *D. farinae*. In addition, frequency of alcohol drinking was also related to a moderate increase in IgE levels in Korean adults.

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Allergic diseases, such as allergic rhinitis, allergic asthma, and atopic dermatitis, are inflammatory conditions caused by increased Th2 immune response, and immunoglobulin E (IgE) plays an important role in allergic processes. The prevalence of allergic disease and associated social economic burden has continued to increase in recent years. Genetic and environmental factors, such as westernized life style, urbanization, and overhygiene, have been attributed to an increase in prevalence of allergic diseases.^{1,2} Because of this, many studies have evaluated influencing factors that develop allergic disease.

Alcohol and smoking are well-known factors related to nonallergic health problems, such as cardiovascular diseases or cancerous conditions. Moreover, immune-modulating adverse effects of alcohol and smoking have been proved.^{3,4} There are some reports suggesting an association between alcohol intake or smoking and specific allergic diseases. Karvonen *et al.* and Lerbaek *et al.* reported that alcohol intake is related with eczematous dermatitis.^{5,6} González-Quintela showed that alcohol consumption increased sensitization rates to pollen.⁷ Thyssen *et al.* reported that the prevalence of nickel sensitization was higher in previous and current smokers than in nonsmokers.⁸ However, other studies have reported conflicting results. Mortaz showed that smoking reduced allergic activation of mouse mast cells and also suppressed cytokine release, such as TNF- α and IL-6, but not leukotrienes.⁹ Personal and parental smoking may reduce the risk of allergic sensitization in people with a family history of atopy; however, this association is not shown in subjects whose parents did not have a history of atopic disease.¹⁰ Additionally, there is a report that maternal alcohol intake reduced the IgE level in cord blood.¹¹

Although many studies have been investigated the effects of alcohol and smoking on allergic sensitization in Western countries, genetic and racial differences in response to alcohol and smoking can

exist. Adverse reaction of alcohol consumption can be more prominent in the Asian population, among whom the capacity to metabolize acetaldehyde is reduced.¹² The risk of attention-deficit and hyperactivity disorder increases as blood cotinine levels increase in children whose parents had tobacco history, and this effect varies across racial groups.¹³

However, studies on the association between alcohol intake or smoking and allergic sensitization continue to be lacking. We evaluated the cross-sectional association between alcohol consumption and smoking on the sensitization to *Dermatophagoides farinae* in Korean adults.

METHODS

Study Population

The data from the 2010 Korean National Health and Nutrition Examination Survey conducted by the Ministry of Health and Welfare in Korea were used for this research. The overall survey consisted of a health interview survey, a health examination survey, and a nutrition survey. It was a cross-sectional nationwide study for noninstitutionalized civilians using a stratified, multistage probability sampling design.

Detailed information on the design of Korean National Health and Nutrition Examination Survey has been reported in a previous study.¹⁴ For the present study, we obtained the data for adults between the ages of 20 and 60 years and excluded subjects with incomplete laboratory and survey data. Subsequent to this, the data for 1469 subjects (760 men and 709 women) were used for this analysis. The Institutional Review Board at the Jeju National University Hospital (Jeju, South Korea) approved this study. Written informed consent was obtained from all participants.

Information on alcohol and tobacco consumption was obtained from a self-registered questionnaire. Frequency of alcohol consumption was classified into four categories: <1 a month, 2–4 times a month, 2–3 times a week, and >4 times a week, according to the average number of drinks during the past 1 year. The amount of alcohol intake was classified into five categories: 1–2 glasses, 3–4 glasses, 5–6 glasses, 7–8 glasses, and >10 glasses, according to the average amount of drinks in one drinking session. Smoking habit was divided into nonsmoker (never smoked or smoked <5 packs in lifetime) and smoker (smoked ≥ 5 packs in lifetime). Demographic

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variables, such as age, sex, and body mass index (BMI), were also included. Age was grouped as 20–29 years, 30–39 years, 40–49 years, and 50–60 years. BMI was calculated using the following formula ($\text{BMI} = \text{weight [kg]} / \text{height}^2 [\text{m}^2]$) and categorized into underweight ($<20 \text{ kg/m}^2$), normal (20 kg/m^2 – 24.99 kg/m^2), overweight (25 kg/m^2 – 29.99 kg/m^2), and obese ($\geq 30 \text{ kg/m}^2$). Economic status was classified into quartiles, according to household income: lowest ($<\text{US\$}918.3$), medium–lowest ($\text{US\$}918.3$ – 1836.5), medium–highest ($\text{US\$}1826.6$ – 3213.9), and highest ($>\text{US\$}3214.0$).

Blood samples were collected after fasting for at least 8 hours. Samples were immediately processed, refrigerated, and transported in cold storage to NeoDin Medical Institute (Seoul, South Korea). The samples were analyzed within 24 hours. Total serum IgE (TIgE) and specific IgE for *D. farinae* were measured using a 1470 WIZARD γ -Counter analyzer (PerkinElmer, Turku, Finland) with ImmunoCAP100 (Phadia, Uppsala, Sweden). *D. farinae* sensitization was defined as specific IgE to *D. farinae* level of $\geq 0.35 \text{ kU/L}$.

Statistical Analysis

TIgE levels were expressed as median and interquartile range because this variable did not show normal distribution after Kolomogorov-Smirnov test (Table 1). The *p* value was assessed by Mann-Whitney test or Kruskal-Wallis test, as appropriate. Number of subjects sensitized to *D. farinae* are shown in Table 2, and logistic regression analysis was used to calculate the crude odd ratios (ORs). To evaluate the independent effects of variables to TIgE levels, multivariate linear regression model was used (Table 3). For this analysis, TIgE levels were transformed into their natural logarithm for normal distribution. In addition, logistic regression analysis was performed to evaluate the association between possible influencing factors and *D. farinae* sensitization. All data were analyzed with the Statistical Products and Service Solutions package for Windows (Release 17.0; SPSS, Inc., Chicago, IL). A value of *p* < 0.05 was considered statistically significant.

RESULTS

The prevalence of smoking was higher in men than women (76.6% versus 10.3%), and the prevalence of subjects drinking alcohol less than one time per month was higher for women than for men (68.6% versus 30.6%). The prevalence of smokers tended to decrease with age, but this difference was not statistically significant. However, the prevalence of subjects drinking alcohol less than one time per month increased with age. The prevalence of smoking and drinking frequency was higher in the overweight and obese groups than in the underweight and normal groups.

TIgE levels ranged from 1.9 to 5000.1 kU/L. The median value was 79.9 kU/L. Table 1 showed the distribution of TIgE levels according to gender, age group, BMI, household income, smoking, and alcohol consumption. Smokers had higher TIgE levels than nonsmokers, and TIgE levels were increased with increasing amount and frequency of alcohol drinking. With regard to other variables, TIgE levels were higher in men than in women, and the 50- to 60-year age group showed higher levels than other age groups. TIgE levels were increased with increasing BMI and decreased with increasing household income.

The overall prevalence of sensitization to *D. farinae* was 39.7% (585/1471). The prevalence of sensitization to *D. farinae* was higher in men (OR, 2.053; 95% CI, 1.658–2.541). Smoking habit and frequency and amount of drinking were also associated with increased prevalence of sensitization, with statistical significance (Table 2). Sensitization to *D. farinae* was lowest for the 40- to 49-year age group. The 20- to 29-year age group (OR, 1.951; 95% CI, 1.443–2.636) presented significantly higher prevalence compared with the reference group (40- to 59-year old group). However, BMI and household income were not associated with sensitization to *D. farinae*.

Table 1 Distribution of TIgE values according to various variables

Variables	TIgE (kU/L)	<i>p</i> Value
Gender		
Males (<i>n</i> = 760)	127.0 (43.9–361.7)	<0.001
Females (<i>n</i> = 709)	53.7 (22.8–131.5)	
Age group (yr)		
20–29 (<i>n</i> = 361)	79.5 (28.9–207.5)	0.014
30–39 (<i>n</i> = 369)	69.0 (28.9–204.5)	
40–49 (<i>n</i> = 357)	71.8 (31.5–208.5)	
50–60 (<i>n</i> = 382)	105.0 (33.2–336.0)	
Smoking		
Smoke (<i>n</i> = 656)	126.0 (43.2–374.5)	<0.001
Nonsmoke (<i>n</i> = 813)	58.1 (25.0–149.0)	
Frequency of drink		
<1 time/mo (<i>n</i> = 718)	56.3 (23.4–141.0)	<0.001
2–4 times/mo (<i>n</i> = 405)	94.3 (33.0–261.5)	
2–3 times/wk (<i>n</i> = 245)	128.0 (41.6–360.5)	
>4 times/wk (<i>n</i> = 101)	283.0 (88.7–568.0)	
Amount of drinking		
none (<i>n</i> = 251)	67.6 (23.9–159.0)	<0.001
1–4 Glasses (<i>n</i> = 605)	59.3 (27.5–181.5)	
5–9 Glasses (<i>n</i> = 398)	96.5 (37.7–323.5)	
>10 Glasses (<i>n</i> = 215)	147.0 (44.9–394.0)	
BMI		
Underweight (<i>n</i> = 200)	58.0 (21.1–152.0)	<0.001
Normal (<i>n</i> = 819)	75.4 (31.0–216.7)	
Overweight (<i>n</i> = 406)	101.5 (33.3–326.0)	
Obese (<i>n</i> = 44)	121.0 (49.2–644.5)	
Economic class		
Lowest (<i>n</i> = 149)	113.0 (42.5–335.7)	<0.036
Medium–low (<i>n</i> = 389)	86.5 (30.4–251.5)	
Medium–high (<i>n</i> = 442)	69.5 (29.6–206.0)	
Highest (<i>n</i> = 489)	77.5 (29.5–238.5)	
Sensitization to <i>Dermatophagoides farinae</i>		
Negative (<i>n</i> = 885)	42.6 (19.4–97.1)	<0.001
Positive (<i>n</i> = 584)	268.5 (95.7–597.7)	

Total IgE values are presented with median and interquartile range (25th–75th percentiles).

The *p* value was assessed by Mann-Whitney test or Kruskal-Wallis test as appropriate.

BMI = body mass index; TIgE = total immunoglobulin E.

To evaluate independent influences of smoking and alcohol intake, multivariate linear regression analysis was performed using logarithmic transformed total IgE (LnTIgE). All possible variables (gender, smoking, alcohol consumption, BMI, and household income) were entered for analysis. The result showed that sensitization to *D. farinae* was mainly associated with LnTIgE levels. The frequency of alcohol drinking showed an association with increased LnTIgE. In addition, male gender, age between 50 and 60 years, increased frequency of alcohol drinking, and household income were related to LnTIgE levels. However, smoking history did not have any significant relationship to LnTIgE levels.

Table 4 presents the result of multivariate logistic regression analysis. In model A, some variables (smoking, frequency, and amount of alcohol drinking) were analyzed after adjusting for gender, age, BMI and household income, whereas all variables were entered in model B. Smoking was positively related with higher prevalence of sensitization to *D. farinae*, which was similarly shown in both models A and B (OR, 1.587, and 95% CI, 1.194–2.108; OR 1.43, and 95% CI, 1.075–1.927, respectively). Moreover, frequent drinking (more than four times a week) was related to increased risk of sensitization to *D.*

Table 2 Crude OR for the association between *Dermatophagoides farinae* sensitization and various confounding variables by logistic regression analysis

	<i>Dermatophagoides farinae</i> -Specific IgE (n)		OR (95% CI)*	p Value*
	<0.35 kU/L	≥0.35 kU/L		
Sex				
Male	396	364	2.053 (1.658–2.541)	<0.001
Female	489	220	Reference	
Age (yr)				
20–29	182	179	1.951 (1.443–2.636)	<0.001
30–39	226	143	1.264 (0.933–1.711)	0.13
40–49	237	120	reference	
50–60	240	142	1.173 (0.868–1.587)	0.299
Smoking				
Smoker	334	322	2.023 (1.637–2.502)	<0.001
Nonsmoker	551	262	Reference	
Frequency of drinking				
<1 time/mo	478	240	Reference	
2–4 times/mo	233	172	1.467 (1.142–1.885)	0.003
2–3 times/wk	132	113	1.701 (1.267–2.285)	<0.001
>4 times/wk	42	59	2.792 (1.826–4.270)	<0.001
Amount of drinking				
None	171	80	Reference	
1–4 glasses	399	206	1.096 (0.802–1.499)	0.565
5–9 glasses	215	183	1.807 (1.300–2.513)	<0.001
>10 glasses	100	115	2.442 (1.676–3.558)	<0.001
BMI				
Underweight	128	72	0.883 (0.639–1.219)	0.449
Normal	501	318	Reference	
Overweight	239	167	1.119 (0.878–1.427)	0.363
Obese	21	23	1.730 (0.942–3.177)	0.077
Economic class				
Lowest	85	64	Reference	
Medium–low	216	173	1.067 (0.729–1.563)	0.737
Medium–high	286	156	0.722 (0.495–1.055)	0.092
Highest	298	191	0.848 (0.584–1.230)	0.385

*A value of $p < 0.05$; presented in boldface type.

BMI = body mass index; IgE = immunoglobulin E; OR = odds ratio.

farinae in both models (OR, 2.391, and 95% CI, 1.528–3.743; OR, 2.052, and 95% CI, 1.257–3.350). Amount of drinking was associated with higher prevalence of sensitization to *D. farinae* in model A, but this relationship was not evident in model B. In addition, the 20- to 29-year age group had significantly higher prevalence compared with other age groups in both models. However, there was no difference among the three remaining groups. Male gender (adjusted OR, 1.406; 95% CI, 1.042–1.896) was also positively associated with sensitization to *D. farinae* in both models. BMI and household income were not related to the prevalence of sensitization to *D. farinae*.

DISCUSSION

In Korean adults, we found that sensitization to *D. farinae* was significantly associated with an increase in smoking history (more than five packs a year) and frequency of alcohol intake (more than four times a week). Frequent drinking was also independently related to increased TIgE levels. Smokers had higher TIgE levels compared with nonsmokers. However, smoking did not show an association to increased TIgE levels after adjusting for confounding variables. These

Table 3 Independent association between logarithmic transformation of TIgE and variables by multivariate linear regression analysis

Variables	Coefficient	SE	p Value
Gender			
Female	reference		
Male	0.378	0.086	<0.001
Age (yr)			
20–29	–0.058	0.087	0.507
30–39	Reference		
40–49	0.102	0.087	0.241
50–59	0.361	0.088	<0.001
Smoking			
Nonsmoker	Reference		
Smoker	0.106	0.084	0.206
Frequency of drink			
<1 time/mo	Reference		
2–4 times/mo	0.305	0.083	<0.001
2–3 times/wk	0.477	0.101	<0.001
>4 times/wk	0.735	0.139	<0.001
Amount of drinking			
None	Reference		
1–4 glasses	–0.148	0.093	0.111
5–9 glasses	–0.226	0.117	0.054
> 10 glasses	–0.249	0.138	0.071
BMI			
Underweight	Reference		
Normal	–0.013	0.094	0.89
Overweight	0.132	0.071	0.063
Obese	0.327	0.178	0.068
Economic class			
Lowest	Reference		
Medium–low	–0.25	0.112	0.025
Medium–high	–0.201	0.11	0.068
Highest	–0.28	0.108	0.01
Sensitization to <i>Dermatophagoides farinae</i>			
Negative	Reference		
Positive	1.621	0.063	<0.001

All variables were entered. The value of $p < 0.05$ is presented in boldface type.

TIgE = total immunoglobulin E; BMI = body mass index; SE = standard error.

results suggested that alcohol consumption and smoking might be related with increased risk of allergic sensitization.

There have been many studies regarding immune-modulating effects of alcohol and smoking. Most studies suggested immunosuppressive effects of alcohol. Ethanol is known to reduce cytokine production, lymphocyte activity, and mast cell degranulation.^{15–17} Recently, some studies have reported that alcohol ingestion could cause Th1/Th2 imbalance, which might predispose a person to allergic diseases.^{18,19} In contrast, other studies have reported conflicting results with associations among alcohol and risk of allergic sensitization and TIgE level.^{2,5,20} Thyssen *et al.* have suggested that alcohol consumption is not associated with nickel sensitization and contact allergic sensitization.⁸

Our results showed that frequent alcohol drinking might increase TIgE levels and risk of allergic sensitization, especially to *D. farinae*. Most studies supported that alcohol drinking could increase TIgE levels and risk of allergic disease.

In studies on the effect of smoking to allergic sensitization, discordant findings were more commonly reported. Some reports showed

Table 4 Adjusted ORs for the association between sensitization to *Dermatophagoides farinae* and confounding variables

	Model A		Model B	
	OR (95% CI)	p Value	OR (95% CI)	p Value
Smoking				
Nonsmoker	Reference		Reference	
Smoker	1.587 (1.194–2.108)	0.001	1.439 (1.075–1.927)	0.015
Frequency of drinking				
<1 time/mo	Reference		Reference	
2–4 times/mo	1.143 (0.875–1.492)	0.328	1.083 (0.802–1.461)	0.604
2–3 times/wk	1.330 (0.970–1.824)	0.077	1.187 (0.830–1.699)	0.347
>4 times/wk	2.391 (1.528–3.743)	<0.001	2.052 (1.257–3.350)	0.004
Amount of drinking				
None	Reference		Reference	
1–4 glasses	1.006 (0.730–1.386)	0.972	0.930 (0.661–1.307)	0.675
5–9 glasses	1.286 (0.895–1.847)	0.174	1.051 (0.690–1.600)	0.818
>10 glasses	1.570 (1.032–2.389)	0.035	1.147 (0.703–1.871)	0.583

Model A, adjusted for gender, age group, BMI, and family income; model B, all variables were entered including gender, age group, BMI, and household income.

The values of $p < 0.05$ are presented in boldface type.

BMI = body mass index; OR = odds ratio.

that smoking might be a risk factor for allergic sensitization, but others have indicated a protective role of smoking.^{8,10,21–24}

These discordant results were usually caused by study design and variables included in analyses. Jarvis *et al.* showed that cat and grass sensitization was less prevalent among smokers, but smokers were at increased risk of sensitization to house-dust mites, which was also observed in our study.²⁵

The mechanism by which alcohol consumption and smoking is associated with TIgE production is unknown. It was presumed that alcohol consumption induces a shift toward Th2 cell responses and increases the production of Th2 cytokines involved in IgE synthesis, which represents elevated TIgE levels in alcoholic subjects.³ It was also speculated that nicotine from smoking modulates immune systems through effects on Th2 and B cells, resulting in the production of cytokines and IL-4 by the Th2 cells and a switch from IgG1 to IgE production by B cells.^{26,27}

On one hand, TIgE levels were significantly higher in the 50- to 60-year age group compared with other age groups in our study. However, there were no differences among other age groups. On the other hand, sensitization to *D. farinae* was more prevalent in the 20- to 29-year age group compared with older groups.

High TIgE levels were related to various conditions, such as parasite infection, hematologic diseases, and rheumatologic diseases. There were also previous results on the relationship between TIgE levels and age. Previous reports showed that TIgE level is the highest in teenagers between the ages of 10 and 15 years and decreased afterward.^{28,29} However, our results showed different distribution of TIgE levels compared with the results from previous studies. TIgE levels did not decrease with advancing age. On the contrary, the levels were increased in the 50- to 60-year age group. This difference was also shown in previous studies, which could be caused by ethnic differences.^{30,31}

Some studies have investigated the relationship between gender and TIgE levels. Kerkhof *et al.* reported that TIgE levels were independently higher in male subjects. Our results also support this previous finding.³² Similarly, Litonjua *et al.* reported that low household income, high serum cotinine level, and high BMI were related to increased TIgE levels.³¹ With regard to economic status, our data showed that TIgE levels were decreased along with increases in household income. This result was not influenced even after adjusting for other variables, including sensitization to *D. farinae*, and this correlates with previous findings by Lewis *et al.*³³

There are several weaknesses to our study. First, this survey did not estimate the exact frequency or amount of tobacco and alcohol consumption. We used smoking history, frequency of drinking, and amount of drinking as variables. More detailed assessment on the amount of smoking and alcohol consumption was needed. Second, we only tested *D. farinae*-specific IgE. Although *D. farinae* is one of most common allergens, IgE levels for other allergens should be included. Third, secondhand smoking was not evaluated in this analysis, but variables related to secondhand smoking should be included for more detailed investigation. Fourth, our study did not collect information on individual allergic diseases or symptoms. Allergic sensitization and higher TIgE did not imply that a subject had allergic disease. Therefore, clinical severity of allergic diseases should be considered in future studies. Finally, other possible variables, such as family history of allergy, should be included in future studies.

Nevertheless, our study suggests that smoking history and frequent alcohol consumption are significantly associated with increases in the prevalence of sensitization to *D. farinae*. Frequency of alcohol drinking over four times a week was also related with increases in TIgE levels in Korean adults.

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REFERENCES

1. Barnes PJ. Pathophysiology of allergic inflammation. *Immunol Rev* 242:31–50, 2011.
2. Linneberg A, Hertzum I, Husemoen LL, et al. Association between alcohol consumption and aeroallergen sensitization in Danish adults. *Clin Exp Allergy* 36:714–721, 2006.
3. Gonzalez-Quintela A, Vidal C, and Gude F. Alcohol, IgE and allergy. *Addict Biol* 9:195–204, 2004.
4. Hällgren R, and Lundin L. Increased total serum IgE in alcoholics. *Acta Med Scand* 213:99–103, 1983.
5. Karvonen J, Poikolainen K, Reunala T, and Juvakoski T. Alcohol and smoking: Risk factors for infectious eczematoid dermatitis? *Acta Derm Venereol* 72:208–210, 1992.
6. Lerbaek A, Kyvik KO, Ravn H, et al. Incidence of hand eczema in a population-based twin cohort: Genetic and environmental risk factors. *Br J Dermatol* 157:552–557, 2007.

7. Gonzalez-Quintela A, Garrido M, Gude F, et al. Sensitization to cross-reactive carbohydrate determinants in relation to alcohol consumption. *Clin Exp Allergy* 38:152–160, 2008.
8. Thyssen JP, Johansen JD, Menne T, et al. Effect of tobacco smoking and alcohol consumption on the prevalence of nickel sensitization and contact sensitization. *Acta Derm Venereol* 90:27–33, 2010.
9. Mortaz E, Folkerts G, Engels F, et al. Cigarette smoke suppresses in vitro allergic activation of mouse mast cells. *Clin Exp Allergy* 39:679–687, 2009.
10. Hancox RJ, Welch D, Poulton R, et al. Cigarette smoking and allergic sensitization: A 32-year population-based cohort study. *J Allergy Clin Immunol* 121:38–42, 2008.
11. Grzybowski M, Ownby DR, Johnson CC, and Peterson EL. Maternal alcohol ingestion and blood cord IgE concentrations. *Pediatr Allergy Immunol Pulmonol* 9:193–198, 1995.
12. Vally H, and Thompson PJ. Allergic and asthmatic reactions to alcoholic drinks. *Addict Biol* 8:3–11, 2003.
13. Xu X, Cook RL, Ilacqua VA, et al. Racial differences in the effects of postnatal environmental tobacco smoke on neurodevelopment. *Pediatrics* 126:705–711, 2010.
14. Lee KW, Park BJ, Kang HT, and Lee YJ. Alcohol-drinking patterns and metabolic syndrome risk: The 2007 Korean National Health and Nutrition Examination Survey. *Alcohol* 45:499–505, 2011.
15. Jayasinghe R, Gianutsos G, and Hubbard AK. Ethanol-induced suppression of cell-mediated immunity in the mouse. *Alcohol Clin Exp Res* 16:331–335, 1992.
16. Roselle GA, and Mendenhall CL. Ethanol-induced alterations in lymphocyte function in the guinea pig. *Alcohol Clin Exp Res* 8:62–67, 1984.
17. Xie J, Kolls J, Bagby G, and Greenberg SS. Independent suppression of nitric oxide and TNF alpha in the lung of conscious rats by ethanol. *FASEB J* 9:253–261, 1995.
18. Cohen DA. Alcohol abuse as a possible cofactor in the progression of acquired immunodeficiency syndrome: Do TH-1 and TH-2 helper T-cell subsets play a role? In *Alcohol, Drugs of Abuse, and Immune Functions*. Boca Raton, FL: Watson RR (Eds). CRC Press, 213–228, 1995.
19. Peterson JD, Herzenberg LA, Vasquez K, and Waltenbaugh C. Glutathione levels in antigen-presenting cells modulate Th1 versus Th2 response patterns. *Proc Natl Acad Sci U S A* 95:3071–3076, 1998.
20. González-Quintela A, Gude F, Boquete O, et al. Association of alcohol consumption with total serum immunoglobulin E levels and allergic sensitization in an adult population-based survey. *Clin Exp Allergy* 33:199–205, 2003.
21. Krämer U, Lemmen CH, Behrendt H, et al. The effect of environmental tobacco smoke on eczema and allergic sensitization in children. *Br J Dermatol* 150:111–118, 2004.
22. Adisesh A, Gruszka L, Robinson E, et al. Smoking status and immunoglobulin E seropositivity to workplace allergens. *Occup Med (Lond)* 61:62–64, 2011.
23. Kulig M, Luck W, Lau S, et al. Effect of pre- and postnatal tobacco smoke exposure on specific sensitization to food and inhalant allergens during the first 3 years of life. Multicenter Allergy Study Group, Germany. *Allergy* 54:220–228, 1999.
24. Kuyucu S, Saraçlar Y, Tuncer A, et al. Determinants of atopic sensitization in Turkish school children: Effects of pre- and post-natal events and maternal atopy. *Pediatr Allergy Immunol* 15:62–71, 2004.
25. Jarvis D, Chinn S, Luczynska C, and Burney P. The association of smoking with sensitization to common environmental allergens: Results from the European Community Respiratory Health Survey. *J Allergy Clin Immunol* 104:934–940, 1999.
26. Lin SY, Reh DD, Clipp S, et al. Allergic rhinitis and secondhand tobacco smoke: A population-based study. *Am J Rhinol Allergy* 25:e66–e71, 2011.
27. Lutz PM, Keltz EA, Brown TD, et al. Environmental cigarette smoke exposure modulates IgE levels of Pb-exposed children. *Toxicology* 291:43–50, 2012.
28. Barbee RA, Brown WG, Kaltenborn W, and Halonen M. Allergen skin-test reactivity in a community population sample: Correlation with age, histamine skin reactions and total serum immunoglobulin E. *J Allergy Clin Immunol* 68:15–19, 1981.
29. Nye L, Merrett TG, Landon J, and White RJ. A detailed investigation of circulating IgE levels in a normal population. *Clin Allergy* 5:13–24, 1975.
30. Gergen PJ, Arbes SJ Jr, Calatroni A, et al. Total IgE levels and asthma prevalence in the US population: Results from the National Health and Nutrition Examination Survey 2005–2006. *J Allergy Clin Immunol* 124:447–453, 2009.
31. Litonjua AA, Celedón JC, Hausmann J, et al. Variation in total and specific IgE: Effects of ethnicity and socioeconomic status. *J Allergy Clin Immunol* 115:751–757, 2005.
32. Kerkhof M, Droste JH, de Monchy JG, et al. Distribution of total serum IgE and specific IgE to common aeroallergens by sex and age, and their relationship to each other in a random sample of the Dutch general population aged 20–70 years. Dutch ECRHS Group, European Community Respiratory Health Study. *Allergy* 51:770–776, 1996.
33. Lewis SA, Weiss ST, Platts-Mills TA, et al. Association of specific allergen sensitization with socioeconomic factors and allergic disease in a population of Boston women. *J Allergy Clin Immunol* 107:615–622, 2001. □

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