

The Association Between Alcohol Consumption and Biomarkers of Alcohol Exposure With Total Serum Immunoglobulin E Levels

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Background: A number of studies have shown that self-reported alcohol intake is positively associated with total serum immunoglobulin E (IgE) levels. The purpose of the present study was to investigate the association of self-reported alcohol consumption and different biomarkers of alcohol exposure to total serum IgE levels in a general adult population.

Methods: A total of 3,443 subjects aged 20 to 79 years from the population-based cross-sectional Study of Health in Pomerania (SHIP) were included in the analyses. Information on alcohol consumption and serum carbohydrate-deficient transferrin (CDT), gamma-glutamyl transferase (GGT), aspartate-amino transferase (ASAT), and alanine-amino transferase (ALAT) levels were measured. Multivariable linear regression models were performed separately in atopic and nonatopic subjects.

Results: In nonatopic subjects positive associations between self-reported alcohol consumption as well as all considered biomarkers of alcohol exposure and total serum IgE levels were found. Further the results also suggested positive associations between alcohol consumption as well as alcohol biomarkers and total serum IgE level in atopic subjects, even though not all tests for linear trend reached statistical significance.

Conclusions: In conclusion, biomarkers of alcohol exposure were positively associated with total serum IgE levels supporting that the positive association between self-reported alcohol intake and IgE levels observed in previous studies is real and not due to misclassification of alcohol intake or confounding by other factors that may be linked to both alcohol intake and total serum IgE levels.

Key Words: Alcohol, Total Serum IgE, Epidemiology, Atopy, Study of Health in Pomerania.

IMMUNOGLOBULIN E (IgE) play an important role in the etiology of allergic diseases. Atopy is the familial or personal predisposition to produce specific IgE antibodies against common allergens in the environment (Johansson et al., 2004) and the most important determinant of IgE levels in developed countries. Besides atopy, a number of other factors may affect the synthesis of IgE such as parasitic infections, especially worm infections, smoking, liver diseases, age, and gender. A number of studies have provided evidence that alcohol intake is positively associated with total serum IgE levels (Gonzalez-Quintela et al., 2002, 2004). Total serum IgE levels are increased in alcoholics (Dominguez-Santalla et al.,

2001; Gonzalez-Quintela et al., 1999; Hällgren and Lundin, 1983; Raithel et al., 2003; Van Epps et al., 1976; Vidal et al., 1994) and moderate alcohol consumers (Criqui et al., 1990; Gonzalez-Quintela et al., 2003; Linneberg et al., 2003; Vidal et al., 2002) and decrease after alcohol abstinence in alcoholics (Gonzalez-Quintela et al., 1999; Hällgren and Lundin, 1983). In addition, a birth cohort study in 2,631 infants found a significant association between maternal alcohol consumption during pregnancy and elevated total serum IgE levels in the cord blood of newborn children (Bjerke et al., 1994).

Over the past decades, the accuracy of the assessment of alcohol consumption has been the subject of much discussion. In population-based studies interviews or questionnaires have been predominantly used for the assessment of alcohol consumption. There are also methods, e.g., the Alcohol Use Disorders Identification TEST (AUDIT) (Reinert and Allen, 2007), for the detection of high risk drinking behavior. However, the validity of these methods based on self-reports has been questioned, in particular under-reporting of alcohol intake is an issue of concern (Del Boca and Darkes, 2003; Midanik, 1982). The aforementioned studies used only self-reported alcohol consumption as a measure of alcohol exposure. One further issue of concern is that the observed association between self-reported alcohol consumption and

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total serum IgE could be due to factors that may be linked to both alcohol intake and total serum IgE.

Beside self-reports, a broad range of laboratory measurements including blood ethanol, serum carbohydrate-deficient transferrin (CDT), serum gamma-glutamyl transferase (GGT), aspartate-amino transferase (ASAT), alanine-amino transferase (ALAT) may be used as biomarkers of recent as well as long-term exposure to alcohol (Niemela, 2007; Sommers et al., 2003). Possible associations between these laboratory measures and total serum IgE levels have not been reported. The purpose of the present study was to investigate the association of self-reported alcohol consumption and different biomarkers of alcohol exposure with total serum IgE levels in a general adult population.

MATERIAL AND METHODS

Study Population

The Study of Health in Pomerania (SHIP) is a cross-sectional population-based survey of the adult population that was conducted in the northeast of Germany (John et al., 2001). From the entire population of 212,157 inhabitants living in the area a sample was selected from the population registration offices, where all German inhabitants are registered. A two-stage cluster sampling method was adopted from the WHO MONICA Project Augsburg, Germany (Keil et al., 1988). A representative sample of 7,008 men and women, aged 20 to 79 years and 292 individuals of each gender in each of the twelve 5-year age strata, was drawn. Data collection started in October 1997 and was finished in May 2001. A total of 4,310 individuals (68.8% of eligible subjects) participated in this study. All participants were investigated in 1 of 2 health examination centers established for the purpose of the study. All participants gave written informed consent. The study confirmed to the principles of the Declaration of Helsinki as reflected by an a priori approval of the Ethics Committee of the University of Greifswald.

Of the 4,310 participants, 844 individuals with missing data on total serum IgE were excluded. However, no differences ($p = 0.16$) in alcohol intake could be observed between subjects with (0 g/d: 34%; 0 to <10 g/d: 31%; 10 to <20 g/d: 13%; 20 to <40 g/d: 13%; ≥ 40 g/d: 8%) and without (0 g/d: 39%; 0 to <10 g/d: 28%; 10 to <20 g/d: 12%; 20 to <40 g/d: 13%; ≥ 40 g/d: 8%) data for total serum IgE. Further 23 subjects with no information on alcohol consumption or atopy status were excluded from the analyses. Altogether the final study population comprised 3,443 subjects (54.9% of eligible subjects) who were included in the present analyses.

Interview Data

A computer-aided personal interview was used to gain information on medical history, behavioral, and socio-demographic characteristics. Alcohol consumption was assessed using the drink-specific quantity–frequency (Q–F) measure: number of days with alcohol consumption and average daily alcohol consumption for such a day over the past month. Average alcohol consumption (in grams per day) was calculated by multiplying frequency and amount of alcohol from beer, wine, and spirits, respectively, using a standard ethanol content of 4.8% (by volume) in beer, 11% (by volume) in wine and 33% (by volume) in spirits to conversion (Bühner et al., 2002). The resulting Q–F of alcohol consumption was categorized into 5 groups: 0 g/d, 0 to <10 g/d, 10 to <20 g/d, 20 to <40 g/d, and ≥ 40 g/d. The smoking status was assessed by self-report. Subjects were categorized into current, former, and never smokers. Hay fever and house dust mite (HDM) allergy were defined according to self-reported answers to the following 3 questions: (1) “Do you suffer

from time to time or permanently from hay fever?”, (2) “Do you have any allergies?” and (3) “Which allergies of the following list, do you have: (3a) pollen allergy; (3b) house dust mite allergy?”. Hay fever was defined as positive answer to question (1) or to questions (2) and (3a). HDM allergy was defined as positive answers to question (2) and (3b). Asthma was assessed by the following 3 questions: (1) “Have you had wheezing or whistling in the chest during the past 12 months?”, (2) “Have you had this wheezing or whistling when you didn’t have a cold?” and (3) “Do you take any anti-asthmatic medication at present, e.g., tablets, spray or inhalations?” Asthma was defined as positive answers to both questions (1) and (2) or to question (3) alone. Chronic bronchitis was defined as productive cough at least 3 consecutive months within the past 12 months. Subjects with chronic bronchitis were classified as nonasthmatics. Atopy was present if subjects reported a positive history for either hay fever, HDM allergy, or asthma.

Blood Samples

Serum ALAT, ASAT, and GGT levels were measured photometrically (Hitachi 717 and Hitachi 704 system; Roche Diagnostics GmbH, Mannheim, Germany). Serum CDT was analyzed nephelometrically (%CDT_{Tri}-TIA, AXIS Biochemicals, Oslo, on a BN II Dade Behring). A combination of CDT and GGT was calculated as $CDT + GGT = [\log_e(GGT)] + [\log_e(CDT)]$ (Anttila et al., 2003). GGT, CDT, ASAT, ALAT and CDT + GGT were categorized into 4 groups according to quartiles of distribution. Total serum IgE levels were measured by the Latex IgE test on the BN II Nephelometer (Dade Behring Marburg GmbH, Marburg, Germany).

Statistical Analyses

Continuous data are expressed as median (25th; 75th quartile). Nominal data are expressed as percentage and total number. For bivariate analyses the Kruskal–Wallis test (continuous data) or chi-square test (nominal data) were used to compare atopic and nonatopic subjects. Multivariable linear regression models were performed to estimate the independent associations of alcohol consumption and biomarkers of alcohol exposure to total serum IgE levels. Biomarkers of alcohol exposure were included as categorized variables. Due to the strong link between atopy and total serum IgE levels and on findings which showed different effects of alcohol consumption on serum total IgE levels in atopic and nonatopic subjects (Linneberg et al., 2003) we performed separate analyses in atopic and nonatopic subjects. Total serum IgE levels were $\log_e$ transformed to obtain normal distribution. The full models were adjusted for age, sex, and smoking. Further tests for linear trend were calculated by fitting alcohol intake, GGT, CDT, ASAT, ALAT and CDT + GGT as continuous covariates in the linear regression models. A value of $p < 0.05$ was considered statistically significant. Statistical analyses were performed with SAS 9.1 (SAS Institute Inc., Cary, NC).

RESULTS

General Characteristics

In the study population, 14.1% ($n = 484$) of the subjects reported to have either hay fever, HDM allergy, or asthma and were classified as atopic subjects. The prevalence of hay fever, HDM allergy, and asthma was 10.3% ($n = 355$), 3.3% ($n = 114$), and 3.9% ($n = 135$), respectively. Subjects with atopy were on average 10 years younger than subjects without atopy (Table 1). Furthermore, atopic subjects consumed more alcohol than nonatopic subjects. In details, in the atopic group a lower proportion of subjects reported to be abstainers

Table 1. General Characteristics in Our Study Population by Atopy

	All Subjects (N = 3,443)	Subjects		<i>p</i> *
		Without Atopy (N = 2,959)	With Atopy (N = 484)	
Men, % (n)	47.9 (1649)	48.2 (1427)	45.9 (222)	0.34
Age, years	35 (49; 62)	50 (36; 63)	40 (30; 57)	<0.01
Smoking, % (n)				
Never smoker	35.6 (1225)	35.2 (1042)	37.8 (183)	0.51
Former smoker	33.0 (1135)	33.1 (978)	32.4 (157)	
Current smoker	31.4 (1083)	31.7 (939)	29.8 (144)	
Alcohol consumption, % (n)				
0 g/d	34.5 (1187)	35.0 (1035)	31.4 (152)	0.06
0 to <10g/d	30.9 (1063)	30.9 (921)	29.4 (142)	
10 to <20g/d	12.7 (438)	12.1 (359)	16.3 (79)	
20 to <40g/d	13.5 (466)	13.3 (392)	15.3 (74)	
≥40g/d	8.4 (289)	8.5 (252)	7.6 (37)	
Alcohol consumption, g/d	5.0 (0; 17.5)	5.0 (0; 17.5)	6.2 (0; 18.7)	0.03
Serum total IgE, IU/ml	36 (17; 103)	33 (15; 91)	63 (25; 186)	<0.01
GGT, IU/l	20 (13; 32)	20 (13; 32)	18 (13; 32)	0.10
ASAT, IU/l	20 (16; 24)	20 (16; 24)	19 (16; 24)	0.61
ALAT, IU/l	23 (17; 32)	23 (17; 32)	23 (16; 33)	0.94
CDT, %	3.8 (4.5; 5.5)	4.5 (3.8; 5.5)	4.6 (3.8; 5.5)	0.44

Continuous data are given as median (25th; 75th quartile); nominal data are given as percentages (total number). *Statistical comparisons were performed with χ^2 test (nominal data) or Kruskal–Wallis test (interval data).

IgE, total immunoglobulin E; GGT, gamma-glutamyl transferase; ASAT, aspartate-amino transferase; ALAT, alanine-amino transferase; CDT, carbohydrate-deficient transferrin.

or to have a low (0 to 10 g/d) as well as high alcohol consumption (≥40 g/d). However, atopic subjects had more often an alcohol intake between 10 and 40 g/d. Further, the total serum IgE levels were higher in atopic subjects. No differences between atopic and nonatopic subjects were apparent with regard to GGT, ASAT, ALAT, and CDT levels.

All 4 markers of alcohol consumption were positively associated with daily alcohol intake (Fig. 1). In abstainers, the GGT, ASAT, ALAT, and CDT levels were 17 IU/l (12; 24 IU/l), 19 IU/l (16; 22 IU/l), 21 IU/l (16; 28 IU/l) and 3.6% (4.3; 5.5%), and increased to 43 IU/l (26; 73 IU/l), 25 IU/l (21; 33 IU/l), 35 IU/l (24; 52 IU/l), and 5.4% (4.3; 6.8%) in alcohol consumers with intake of more than 40 g/d, respectively.

Alcohol Consumption and Total Serum IgE

Descriptive analyses showed an increase in total serum IgE levels with increasing alcohol intake. Among nonatopic abstainers, the total serum IgE level was 30 IU/ml (13 IU/ml; 82 IU/ml), and increased to 44 IU/ml (21 IU/ml; 129 IU/ml) in consumers with daily alcohol intake ≥40 g/d. In atopic subjects a clearer pattern of increasing IgE levels was found. Whereas abstainers had a median total serum IgE level of 52 IU/ml (19 IU/ml; 131 IU/ml), in subjects with high alcohol consumption the total serum IgE level increased to 137 IU/ml (64 IU/ml; 301 IU/ml). Furthermore, multivariable analyses adjusted for sex, age, and smoking also suggested a positive association between alcohol consumption and total serum IgE level in atopic and nonatopic subjects (Table 2), even though the test for linear trend barely missed statistically significance in

atopic subjects ($p = 0.06$). The estimated adjusted mean levels of total serum IgE depending on alcohol consumption are given in Fig. 2.

Alcohol Biomarker Levels and Total Serum IgE

Multivariable analyses (Table 2) supported positive associations between all considered markers for alcohol consumption and total serum IgE level (Table 2), even though some tests for linear trend barely missed significance in subjects with atopy. The estimated adjusted mean level of total serum IgE by biomarker levels are shown in Fig. 2.

Further Determinants of Increased Total Serum IgE

Moreover, in both atopic and nonatopic subjects men had higher levels of total serum IgE [model including alcohol consumption: atopics: coefficient 0.43 (Stderr 0.14); nonatopic 0.44 (0.06)] than women in all tested models. A positive association between smoking and total serum IgE was only seen in nonatopic subjects. Former smokers [0.13 (0.06)] as well as current smokers [0.46 (0.14)] had higher levels of total serum IgE.

For Sensitivity analyses we excluded all subjects ($N = 184$) with liver diseases defined as self-reported liver cirrhosis, atrophy of the liver or ASAT, ALAT or GGT levels > population mean + 2 standard deviation. However, the main results of positive associations between alcohol consumption as well as biomarkers of alcohol intake and total serum IgE levels remained reasonably similar after exclusion of these subjects (data not shown).

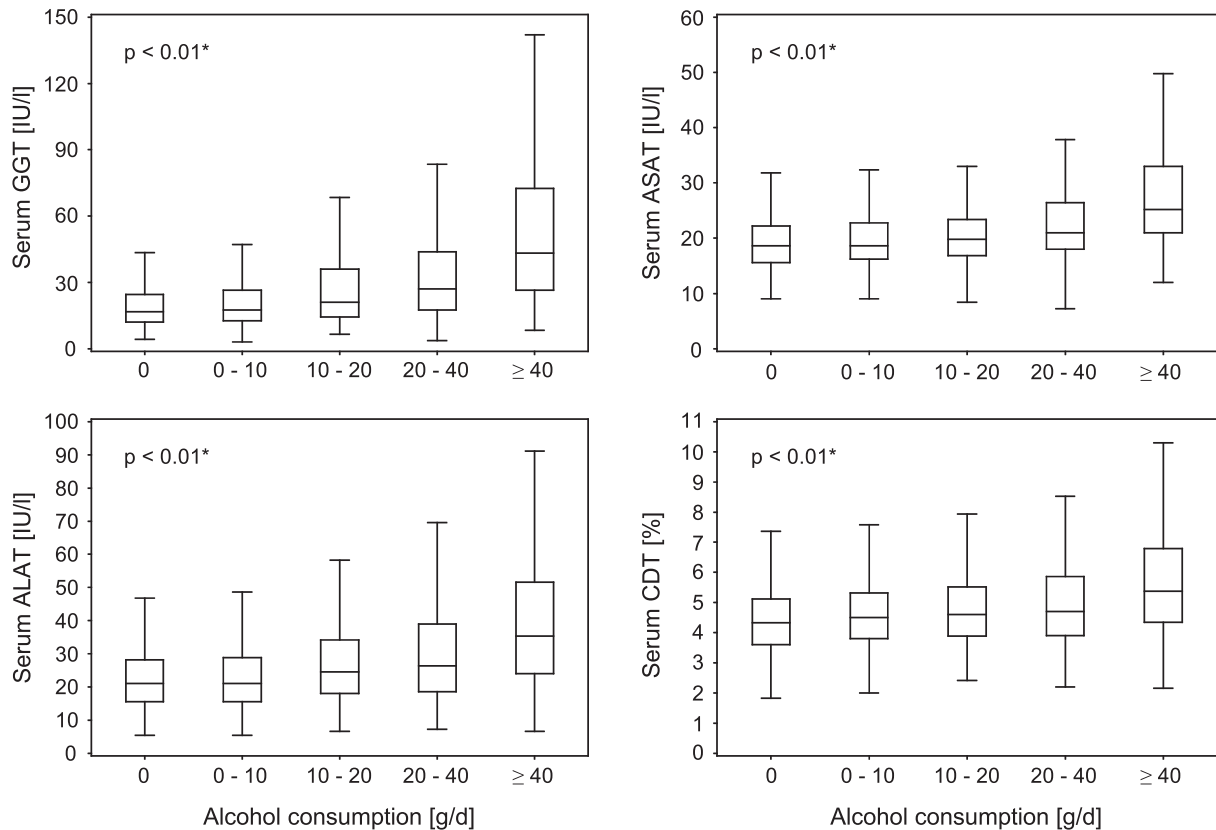


Fig. 1. Serum gamma-glutamyl transferase (GGT), aspartate-amino transferase (ASAT), alanine-amino transferase (ALAT), and carbohydrate-deficient transferrin (CDT) levels according to daily alcohol consumption. Box-plots are diagrammed with the median (horizontal line within the box) and the interquartile range, corresponding to 25th to 75th quartile (lower and upper horizontal line of the box). *Statistical comparisons were performed with the Kruskal–Wallis test.

DISCUSSION

The present analyses suggest positive associations of alcohol consumption and different biomarkers of alcohol intake with total serum IgE in both atopic and nonatopic subjects. However, in atopic subjects positive associations between alcohol consumption, GGT, ALAT or CDT and total serum IgE barely missed statistical significance. To the best of our knowledge, this is the first report on the associations between biomarkers for alcohol consumption and total serum IgE.

Our results are in line with 2 previous cross-sectional population-based studies which reported a positive association between self-reported alcohol intake and total serum IgE (Gonzalez-Quintela et al., 2003; Linneberg et al., 2003). In a Danish population (Linneberg et al., 2003), however, alcohol consumption was positively related to total serum IgE levels only in nonatopic subjects, whereas a negative association was seen in atopic subjects. Also studies in alcoholics (Dominguez-Santalla et al., 2001; Gonzalez-Quintela et al., 1999; Hällgren and Lundin, 1983; Raithel et al., 2003; Van Epps et al., 1976; Vidal et al., 1994) and different patients populations (Criqui et al., 1990; Raithel et al., 2003; Van Epps et al., 1976; Vidal et al., 2002) have provided evidence that alcohol

consumption is related to increased total serum IgE levels. The finding of a positive association between biomarkers of alcohol exposure and serum total IgE suggests that the observed association between self-reported alcohol consumption and total serum IgE is not due to misclassification of alcohol exposure or confounding by factors associated with both alcohol consumption and increased IgE levels.

The most important influencing factors for IgE levels are parasite infections and atopy. As reported previously (Dominguez-Santalla et al., 2001), it is not likely that the association between alcohol consumption and serum IgE levels observed in different countries and settings can be explained by confounding by parasite infection. The investigation of *Toxocara* antibodies in alcoholics and controls showed that the serum IgE increase was independent of *Toxocara* seropositivity (Nash, 2000). However, two Brazilian studies found a higher frequency of intestinal infestation by *Strongyloides stercoralis* in alcoholic compared to nonalcoholic subjects (de Oliveira et al., 2002; Zago-Gomes et al., 2002). The frequencies of other intestinal nematodes were comparable (Zago-Gomes et al., 2002).

Because atopy is a main influencing factor of IgE antibodies levels, we performed separate analyses for atopic and nonatopic persons, respectively. However, the association of

Table 2. Associations Between Serum Total Immunoglobulin E Levels and Alcohol Consumption as Well as Selected Biomarkers in Atopic and Nonatopic Subjects by Multivariate Linear Regression Analyses

	Subjects Without Atopy (N = 2,959)		Subjects With Atopy (N = 484)	
	β Coefficient* (95% CI)	p-trend (linear)	β Coefficient* (95% CI)	p-trend (linear)
Alcohol consumption, g/d				
0	Reference	0.02	Reference	0.06
0 to <10	-0.05 (-0.17; 0.06)		0.19 (-0.13; 0.52)	
10 to <20	0.13 (-0.03; 0.29)		0.02 (-0.37; 0.41)	
20 to <40	0.05 (-0.11; 0.22)		0.08 (-0.33; 0.48)	
≥40	0.19 (-0.01; 0.39)		0.49 (-0.05; 1.03)	
GGT, IU/l**				
≤13.2	Reference	<0.01	Reference	0.06
13.3 to 19.2	0.01 (-0.13; 0.15)		-0.27 (-0.62; 0.08)	
19.3 to 32.3	0.05 (-0.09; 0.19)		0.05 (-0.32; 0.42)	
≥32.4	0.25 (0.10; 0.40)		0.05 (-0.34; 0.44)	
ASAT, IU/l				
≤16.3	Reference	<0.01	Reference	0.03
16.4 to 19.7	0.06 (-0.07; 0.20)		-0.09 (-0.45; 0.26)	
19.8 to 23.9	0.15 (0.01; 0.29)		0.06 (-0.31; 0.43)	
≥24.0	0.18 (0.04; 0.33)		0.36 (-0.02; 0.74)	
ALAT, IU/l				
≤16.3	Reference	<0.01	Reference	0.09
16.4 to 22.2	0.09 (-0.05; 0.23)		0.03 (-0.33; 0.39)	
22.3 to 31.8	0.09 (-0.05; 0.24)		0.26 (-0.11; 0.63)	
≥31.9	0.21 (0.06; 0.36)		0.35 (-0.04; 0.75)	
CDT, %				
≤3.79	Reference	<0.01	Reference	0.11
3.79 to 4.52	-0.05 (-0.19; 0.09)		0.21 (-0.15; 0.56)	
4.52 to 5.46	0.07 (-0.07; 0.20)		0.13 (-0.22; 0.48)	
≥5.46	0.11 (-0.02; 0.25)		0.38 (0.02; 0.73)	
CDT + GGT				
≤3.98	Reference	<0.01	Reference	<0.01
3.98 to 4.36	-0.03 (-0.16; 0.11)		-0.01 (-0.36; 0.35)	
4.36 to 4.86	0.05 (-0.09; 0.19)		0.15 (-0.20; 0.50)	
≥4.86	0.29 (0.14; 0.44)		0.31 (-0.08; 0.70)	

CI, confidence interval; GGT, gamma-glutamyl transferase; ASAT, aspartate-amino transferase; ALAT, alanine-amino transferase; CDT, carbohydrate-deficient transferrin.

*Estimates are adjusted for age, sex, and smoking.

**GGT, ASAT, ALAT, CDT, and CDT+GGT were categorized into 4 groups according to the quartiles of distribution.

alcohol consumption and biomarkers with total serum IgE levels was apparent in both atopic and nonatopic subjects, although in the first group not all biomarkers reached statistical significance possibly due to lower power in this group.

Accurate biomarkers of alcohol consumption are needed. Our results suggest that total serum IgE may be a novel biomarker of alcohol exposure. However, due to strong influence of atopy, total serum IgE is likely to be of low specificity in developed populations where the prevalence of atopy is high.

The mechanisms of alcohol-induced increase in total serum IgE are not fully elucidated (Gonzalez-Quintela et al., 2002). The available evidence points to ethanol or one of its metabolites as the substance responsible for the effects of alcoholic drink consumption on the immune system and increased IgE production (Gonzalez-Quintela et al., 2002, 2004). There is substantial evidence that alcohol impairs the T helper lymphocyte type 1 (Th1) immune responses possibly favouring a relative Th2 dominance. Furthermore, the cytokine balance may be skewed in a Th2 direction (Gonzalez-Quintela et al., 2002, 2004). Recently, an unifying explanation for alcohol effects on immune function has been presented (Heinz and

Waltenbaugh, 2007). Thus, this hypothesis proposes that alcohol exerts its effects in particular at the level of dendritic cells and antigen presentation inducing interleukin-25 (IL-25) production to enhance Th2 effector function by increasing IL-13. IL-13 is a Th2 cytokine that is known to promote IgE class switching.

It is well-known that alcohol may increase the permeability of the gastrointestinal mucosa thereby enhancing intestinal absorption of endotoxin (lipopolysaccharide), a cell wall component of gram negative bacteria. Thus, an alternative explanation for the alcohol-induced increase in IgE production could be that endotoxin may stimulate Th2 deviation and B-cell IgE antibody production through its binding to Toll-like receptors on innate immune cells and regulatory T cells. Evidence in favour of this hypothesis was generated by Campos et al., who observed a significant interaction between alcohol consumption and a polymorphism (-159C/T) of the CD14 promoter gene encoding for the CD14 molecule of Toll-like receptors (Campos et al., 2006). The carriers of the CD14/-159 allele show higher levels of total serum IgE and appeared to be particularly susceptible to the effects of alcohol on IgE levels (Campos et al., 2006).

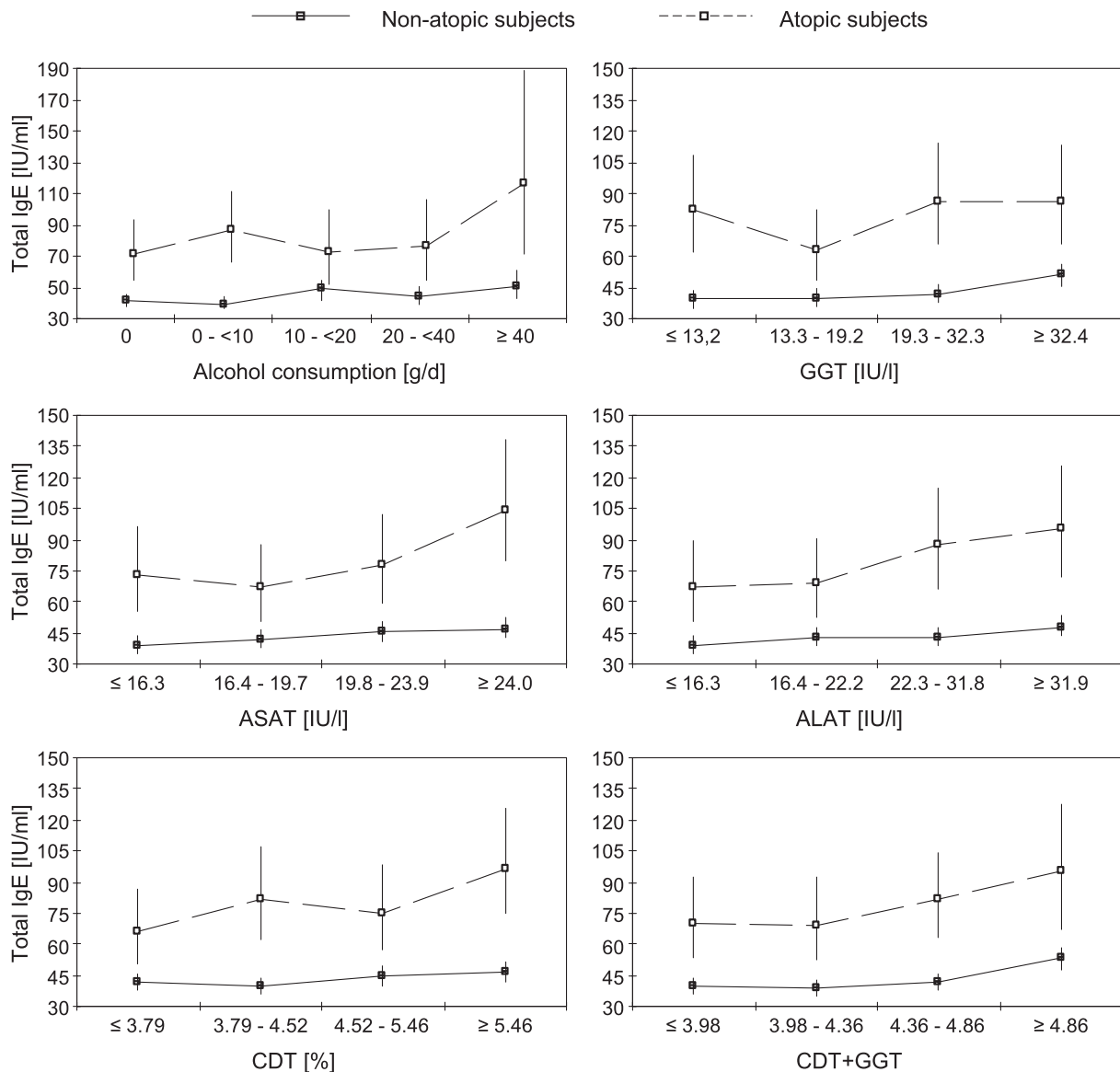


Fig. 2. Estimated mean level of total serum immunoglobulin E (IgE) level with 95% confidence interval by alcohol consumption and gamma-glutamyl transferase (GGT), aspartate-amino transferase (ASAT), alanine-amino transferase (ALAT), carbohydrate-deficient transferrin (CDT), and CDT + GGT combination level. Multivariate linear regression model were adjusted for age, sex, and smoking.

Our multivariable analyses obtained 2 further determinants of increased total serum IgE levels. First, in concordance with previous studies (Criqui et al., 1990; Jarvis et al., 1999; Linneberg et al., 2003; Omenaas et al., 1994; Vidal et al., 2002; Wuthrich et al., 1996) our analyses revealed that men had higher levels of total serum IgE than women. Second, former and current smoking was also positively associated with total serum IgE levels. Several previous studies (Gonzalez-Quintela et al., 2003; Jarvis et al., 1999; Linneberg et al., 2003; Wuthrich et al., 1996), but not all (Dominguez-Santalla et al., 2001; Vidal et al., 2002), have found a positive association between smoking and IgE levels. However, some of these studies (Criqui et al., 1990; Jarvis et al., 1999; Omenaas et al., 1994; Wuthrich et al., 1996) did not take into account the possible confounding effects of alcohol consumption.

One of the strengths of this study was the accurate determination of alcohol consumption by the drink-specific Q-F measure. Moreover, we performed our analyses using data of a large population-based sample. There are limitations of our study that should be considered. Measurements on serum total IgE levels were only available for 3,466 of the 4,310 investigated subjects in SHIP. Furthermore, the atopy status was only assessed by questionnaire without any objective measurements, and some misclassifications of atopy status are likely to have occurred. However, the prevalence data of hay fever and asthma from our study are consistent with other reported prevalence proportions for adults in Germany (Hermann-Kunz, 1999a; Janson et al., 2001) thereby emphasizing the good validity of our study. The prevalence of HDM allergy in our study was 3.9% and thus lower than the

reported prevalence of 11 to 15% from the MONICA study (Filipiak et al., 2001). However, it should be considered that the present study was performed in a population in the former East German where the prevalence of HDM allergy is the lowest in Germany (Hermann-Kunz, 1999b). The accordance of two of the above-mentioned studies (Hermann-Kunz, 1999a; Janson et al., 2001) with our results supports the representativeness of our study.

In conclusion, biomarkers of alcohol exposure were positively associated with total serum IgE levels supporting that the positive association between self-reported alcohol intake and IgE levels observed in previous studies is real and not due to misclassification of alcohol intake or confounding by other factors that may be linked to both alcohol intake and total serum IgE levels.

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