Chapter 5

Risk Factors for Allergy in Secondary School Girls

Ramla Hamed Ahmed Alobaidi,
Amina Hamed Ahmed Alobaidi and
Abdulghani M. Alsamarai

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/59376

1. Introduction

Ambient air is identified by the World Health Organization as a high public health priority, since air pollution associated with increase in mortality and morbidity of disease [1,2]. Respiratory disorders are the most important health problems in Iraq [3] The reported studies indicated a high prevalence rate of allergic diseases in Iraqi population.[4] In addition, respiratory infection in children represent one of the major infectious problems in Iraq [5]. The community health research that were performed by Tikrit University College of Medicine [TUCOM] have estimated that more than a fourth of Iraqi children with asthma report weekly wheeze and cough. Two-thirds of report school absences and one-third report frequent sleep disturbances due to asthma. [5]

Respiratory and asthma symptoms are public health challenges in the area of secondary school student's health and are the leading cause of school absenteeism in children, and result in missed workdays and lost productivity in adults as well [6]. The evidence strongly suggests that poor indoor air quality in schools can impact on the respiratory health of students. Children are at greater risk of the development of respiratory diseases in poor environmental conditions because their immune system is still developing [7-9].

Even though poor indoor air quality (IAQ) may have a role in exacerbation of allergic disorders [10] the socio-economic status may also have a key role in the development and progress of respiratory symptoms and asthma, especially in school students [11-13]. The influence of socio-economic status could be explained by current and past individual exposures to lifestyle and environmental factors [13].

Globally allergic diseases form a major health problem with increased incidence with time and thus are with socioeconomic impact on individuals and community [14]. Health care delivery for individuals with allergic diseases is insufficient and/or inadequate. Although, in the last decades there is progress in allergic diseases research, still there is a gap in information related to explore the underlying causes, therapies and eventual prevention [15-17]. Therefore, 'Global Allergy Forum'. participants concluded that *there are numerous unmet clinical needs and millions of patients are undertreated or not treated with the most appropriate methods.*[14]. In many countries, including Iraq, health care delivery accessibility to and affordability of effective therapeutic approaches are not provided. The development of novel effective therapies for allergic diseases is slow as compared to other medicine fields [14].

A new integrative approach is needed to understand how a complex network of immunological, genetic, and environmental factors leads to a complex allergic phenotype [15]. *There is a tremendous lack of knowledge regarding many unsolved issues, Apart from true lack of information, there is a tremendous gap between actual existing knowledge and its effective application for the millions of people in need [14].* Kirkuk, an area that characterised with high air pollution levels [18]. Since action should be taken at various levels and through existing doctors, scientists, and lay organizations to solve these problems, thus this study was performed to clarify the problem levels in the above context. Informed consent taken from all girls included in the study and the study protocol was approved by the ethical committee.

Objective: To:

Clarify the role of environmental and personal factors as risk factors for development of asthma and allergy in secondary school students.

2. Materials & methods

2.1. Site of the study

Kirkuk, an Iraqi governorate that is located North-Western to Baghdad, with 1.200 000 inhabitants. An area that is characterised by production of oil and its products. The governorate is with air pollution, which may be of health hazard in some areas of the governorate [18].

2.2. Study population

The study was carried out in two sites of Kirkuk Governorate. The first one was a secondary school in Kirkuk city center, while the others (3 schools) located in Kirkuk rural areas. A total of 594 girls included in the study, their age range from 12-22 years, with a mean age of 16.4 years. Of them, 387 (65.2%) were from urban and 207 (34.8%) were from rural areas.

2.3. Assessment of symptoms

The occurrence of symptoms and demographic characteristics was recorded by a self administered questionnaire given directly to each student. The questionnaire requested information

http://dx.doi.org/10.5772/59376

on personal factors, health status, physical activities, environmental exposures at present and during childhood, and information on type of residence, type of ownership, house age, size of the dwelling, number of subjects living in the house, type of ventilation, type of cooling, type of heating, presence of animals in the house, presence of tree, grass, spider and wool in houses, presence of cockroach and wall painting.

2.4. Statistical methods

Chi square test was determined using SPSS (version 16) to clarify the significance of differences. Significance of differences in means were calculated by Students t test. In all statistical analyses, two tailed tests and 5% level of significance were used. The influence of different factors on the prevalence of asthma or allergy was analysed by both linear and logistic regression using SPSS statistical package. Odd ratios with 95% confidence interval were calculated from the logistic regression models.

3. Results

3.1. Asthma for whole study population

For all survey data (combined urban and rural), there was agreement between the two analysis methods in demonstrating significant association between asthma development and risk factors such as oil drinking; oil heating; fan cooling; child respiratory tract infection; child exposure to agricultural dust and work; family history of asthma; child playing; menses irregularity; depression; food allergy; heartburn; IBS; stress; house presence of wool, grass and tree; family history of atopy; and presence of water cycle within house. However, there was agreement between the two methods in relation to negative significant association between crowding index; house ownership; air condition heating; electricity heating and asthma development. Table 1

Variable	Regression		Logistic regression	
	B	P value	B	P value
Oil drinking	0.272	0.000	1.544	0.000
BMI	**0.012**	**0.015**	-0.057	0.068
Crowding index	-0.050	0.000	-0.274	0.002
House ownership	-0.297	0.000	-1.626	0.000
Oil heating	0.119	0.007	0.900	0.000
Gas heating	0.017	0.803	-0.014	0.973
Air condition heating	-0.146	0.000	-0.731	0.003
Electricity heating	-0.139	0.000	-0.483	0.037

Variable	Regression		Logistic regression	
	B	P value	B	P value
Fan cooling	0.164	0.002	1.003	0.007
Air condition cooling	**-0.085**	**0.035**	-0.418	0.067
Water cooling	-0.022	0.615	-0.141	0.555
Animal exposure	0.077	0.123	0.423	0.079
Child exposure to smoking	0.066	0.177	0.348	0.145
Child respiratory tract infection	0.186	0.000	0.988	0.000
Child exposure to cockroach	-0.022	0.566	0.061	0.766
Child residence	**0.111**	**0.023**	0.452	0.069
Child hitting	-0.062	0.157	-0.343	0.181
Child exposure agriculture dust	0.171	0.001	1.012	0.001
Child agriculture work	0.149	0.002	0.891	0.003
Child physical activity	0.055	0.139	0.283	0.145
Family history of asthma	0.169	0.003	1.160	0.003
Family history allergic rhinitis	-0.033	0.436	-0.187	0.426
Family history atopic dermatitis	0.039	0.384	0.342	0.161
Aspirin use	-0.049	0.218	**-0.627**	**0.010**
School stress	0.038	0.429	0.011	0.966
Child playing	0.204	0.000	0.65	0.000
Cold sore	**-0.309**	**0.000**	-20.571	0.996
Menses irregularities	0.187	0.000	1.107	0.000
Hirsutism	0.048	0.494	**0.930**	**0.004**
Anxiety	0.053	0.053	**0.660**	**0.004**
Depression	0.130	0.000	0.828	0.001
Psychological problem	0.312	0.000	-21.671	0.998
Social problem	0.064	0.200	0.876	0.061
Food allergy	0.758	0.000	5.396	0.000
Heart burn	0.241	0.000	2.426	0.000
Irritable	0.001	0.965	0.007	0.985
IBS	0.275	0.000	2.325	0.019
Stress	0.383	0.000	3.655	0.000
House animal	**0.138**	**0.001**	0.160	0.644
House cockroach	-0.009	0.807	-0.829	0.061

Variable	Regression		Logistic regression	
	B	P value	B	P value
House wool	0.195	0.000	1.144	0.000
House spider	0.063	0.102	**0.803**	**0.001**
House grass	0.126	0.000	0.864	0.000
House tree	0.220	0.000	1.496	0.000
Family history of atopy	0.150	0.000	0.812	0.001
Water cycle	0.201	0.000	0.871	0.000
Breast feeding	**-0.129**	**0.008**	-0.371	0.122
Illiterate father	-0.032	0.508	-0.199	0.462
Illiterate mother	-0.028	0.543	-0.058	0.816
House painting	-0.004	0.911	-0.126	0.568

Table 1. Comparison between regression and logistic regression for study population in relation to asthma.

3.2. Allergy for whole study population

For allergy development (any allergy) in study population, both models demonstrated agreement of significant positive association with risk factors such as: animal exposure; family history of allergic rhinitis and atopic dermatitis; school stress; child playing; anxiety; depression; psychological problem; irritable personality; house presence of animal, wool, spider and grass; family history of atopy and presence of water cycle within house. However, both models agreed as that allergy development was with negative significant association with BMI; crowding index; house ownership; breast feeding and mother illiteracy. Table 2

Variable	Regression		Logistic regression	
	B	P value	B	P value
Oil drinking	**0.456**	**0.000**	21.851	0.996
BMI	-0.030	0.000	-0.248	0.000
Crowding index	-0.008	0.455	**-0.151**	**0.043**
House ownership	-0.322	0.000	-2.763	0.000
Oil heating	0.042	0.382	**0.741**	**0.009**
Gas heating	**0.301**	**0.000**	21.136	0.997
Air condition heating	0.066	0.124	**0.925**	**0.001**
Electricity heating	-0.054	0.194	**-0.937**	**0.001**
Fan cooling	**0.184**	**0.001**	0.428	0.114
Air condition cooling	0.060	0.153	**0.451**	**0.047**

Variable	Regression		Logistic regression	
	B	P value	B	P value
Water cooling	0.007	0.880	-0.148	0.558
Animal exposure	0.293	0.000	1.734	0.000
Child exposure to smoking	**0.100**	**0.042**	0.414	0.190
Child respiratory tract infection	**0.330**	**0.000**	20.361	0.996
Child exposure to cockroach	**0.107**	**0.005**	0.123	0.512
Child residence	-0.039	0.415	-0.334	0.176
Child hitting	0.034	0.434	0.119	0.606
Child exposure agriculture dust	-0.047	0.370	-0.060	0.823
Child agriculture work	-0.059	0.224	-0.215	0.398
Child physical activity	-0.052	0.162	0.006	0.975
Family history of asthma	0.031	0.571	0.554	0.177
Family history allergic rhinitis	0.291	0.000	2.208	0.000
Family history atopic dermatitis	0.126	0.004	0.675	0.023
Aspirin use	**0.103**	**0.014**	0.402	0.092
School stress	0.209	0.000	1.594	0.000
Child playing	0.223	0.000	1.792	0.000
Cold sore	0.056	0.356	1.097	0.015
Menses irregularities	0.057	0.204	-0.320	0.233
Hirsutism	-0.102	0.166	-0.288	0.370
Anxiety	0.137	0.000	1.586	0.000
Depression	0.228	0.000	3.121	0.000
Psychological problem	0.220	0.002	3.176	0.000
Social problem	-0.078	0.261	0.867	0.188
Food allergy	0.268	0.000	20.296	0.995
Heart burn	**0.223**	**0.000**	19.711	0.996
Irritable	0.162	0.000	1.849	0.000
IBS	-0.073	0.367	17.829	0.998
Stress	**0.217**	**0.007**	18.841	0.998
House animal	0.121	0.002	0.576	0.036
House cockroach	**0.074**	**0.048**	0.485	0.066
House wool	0.129	0.000	0.905	0.000
House spider	0.194	0.000	1.780	0.000

Variable	Regression		Logistic regression	
	B	P value	B	P value
House grass	0.210	0.000	1.342	0.000
House tree	-0.009	0.783	0.045	0.835
Family history of atopy	0.166	0.000	1.023	0.000
Water cycle	0.186	0.000	0.858	0.000
Breast feeding	-0.250	0.000	-1.798	0.000
Illiterate father	0.049	0.322	0.486	0.074
Illiterate mother	-0.091	0.048	-0.711	0.005
House painting	0.005	0.909	0.061	0.768

Table 2. Comparison between regression and logistic regression for study population in relation to allergy

3.3. Asthma for urban study population

When analysis performed for urban population, the two models demonstrated agreement on significant positive association between asthma development and risk factors such as: oil drinking; child respiratory tract infection; child hitting; child exposure to agricultural dust band work; child physical activity; aspirin use; child playing and heartburn. However, agreement between the two models demonstrated a significant negative association between asthma development and risk factors such as: house ownership; air condition heating; electricity heating; air condition cooling; presence of house cockroach, wool, and tree; and family history of atopy. Table 3

Variable	Regression		Logistic regression	
	B	P value	B	P value
Oil drinking	0.555	0.000	2.421	0.000
BMI	0.000	0.948	-0.027	0.532
Crowding index	0.017	0.393	**-0.280**	**0.048**
House ownership	-0.297	0.000	-1.259	0.000
Oil heating	-0.024	0.645	-0.440	0.154
Gas heating	0.079	0.402	1.175	0.057
Air condition heating	-0.283	0.000	-1.732	0.000
Electricity heating	-0.146	0.004	-1.243	0.000
Fan cooling	0.226	0.163	18.081	0.999
Air condition cooling	-0.258	0.000	-1.946	0.000
Water cooling	-0.048	0.380	-0.063	0.844

Variable	Regression		Logistic regression	
	B	P value	B	P value
Animal exposure	0.040	0.575	-0.293	0.452
Child exposure to smoking	0.081	0.179	0.456	0.202
Child respiratory tract infection	0.272	0.000	1.176	0.001
Child exposure to cockroach	-0.032	0.519	0.111	0.677
Child residence	**0.118**	**0.042**	0.510	0.109
Child hitting	0.160	0.007	1.662	0.000
Child exposure agriculture dust	0.376	0.000	1.855	0.001
Child agriculture work	0.365	0.003	3.953	0.000
Child physical activity	0.149	0.005	1.338	0.000
Family history of asthma	**0.249**	**0.001**	-21.781	0.997
Family history allergic rhinitis	-0.124	0.018	0.737	0.027
Family history atopic dermatitis	0.102	0.084	0.469	0.230
Aspirin use	0.274	0.000	1.510	0.000
School stress	0.020	0.751	-0.072	0.850
Child playing	0.169	0.003	1.639	0.000
Cold sore	**-0.526**	**0.000**	-22.213	0.998
Menses irregularities	**0.129**	**0.009**	0.259	0.382
Hirsutism	0.199	0.075	22.857	0.997
Anxiety	**0.140**	**0.000**	0.370	0.223
Depression	0.064	0.089	0.472	0.096
Psychological problem	**-0.325**	**0.000**	-21.881	0.997
Social problem	0.017	0.773	**1.007**	**0.038**
Food allergy	0.791	0.000	37.158	0.994
Heart burn	0.205	0.000	1.168	0.001
Irritable	0.073	0.102	**1.270**	**0.000**
IBS	0.304	0.000	**-17.598**	**0.996**
Stress	0.377	0.000	**21.047**	**0.998**
House animal	0.261	0.000	1.294	0.000
House cockroach	0.123	0.009	-0.769	0.004
House wool	0.200	0.000	1.211	0.000
House spider	-0.017	0.733	-0.144	0.606
House grass	-0.056	0.256	-0.374	0.166

http://dx.doi.org/10.5772/59376

Variable	Regression		Logistic regression	
	B	P value	B	P value
House tree	0.312	0.000	1.727	0.000
Family history of atopy	0.298	0.000	1.626	0.000
Water cycle	0.083	0.145	0.060	0.816
Breast feeding	**-0.151**	**0.012**	-0.297	0.264
Illiterate father	**-0.165**	**0.026**	-0.619	0.083
Illiterate mother	-0.049	0.466	-0.076	0.806
House painting	-0.066	0.278	-0.400	0.152

Table 3. Comparison between regression and logistic regression for urban population in relation to asthma.

3.4. Allergy for urban study population

The two models agreement on positive significant association was achieved between allergy development in urban population and risk factors such as: oil heating; animal exposure; child exposure to cockroach; irritable personality; presence of house grass and family history of atopy. However, agreement between the two models was achieved on significant negative association between allergy development and risk factors such as: house ownership; electricity heating; and child residence. Table 4

Variable	Regression		Logistic regression	
	B	P value	B	P value
Oil drinking	**0.323**	**0.000**	20.752	0.997
BMI	-0.010	0.105	-0.142	0.014
Crowding index	**0.054**	**0.001**	0.160	0.402
House ownership	-0.218	0.000	-2.452	0.000
Oil heating	0.192	0.000	1.265	0.000
Gas heating	**0.185**	**0.019**	18.463	0.998
Air condition heating	0.085	0.265	**0.986**	**0.006**
Electricity heating	-0.132	0.002	-1.312	0.000
Fan cooling	-0.222	0.095	-18.883	0.999
Air condition cooling	-0.084	0.302	-0.139	0.659
Water cooling	**0.111**	**0.023**	-0.506	0.167
Animal exposure	0.180	0.001	0.850	0.042
Child exposure to smoking	-0.033	0.475	-0.557	0.154

Variable	Regression		Logistic regression	
	B	P value	B	P value
Child respiratory tract infection	**0.289**	**0.000**	20.430	0.996
Child exposure to cockroach	0.139	0.000	1.0656	0.001
Child residence	-0.226	0.000	-0.851	0.005
Child hitting	**-0.073**	**0.116**	2.545	0.000
Child exposure agriculture dust	-0.055	0.495	22.336	0.997
Child agriculture work	0.074	0.442	21.654	0.998
Child physical activity	**-0.161**	**0.000**	-0.274	0.433
Family history of asthma	0.067	0.247	15.993	0.996
Family history allergic rhinitis	0.131	0.212	**1.345**	**0.003**
Family history atopic dermatitis	0.196	0.288	20.620	0.995
Aspirin use	0.025	0.577	**1.439**	**0.000**
School stress	-0.008	0.874	0.805	0.097
Child playing	**0.230**	**0.000**	19.881	0.996
Cold sore	0.093	0.236	-0.267	1.000
Menses irregularities	-0.029	0.485	-0.322	0.240
Hirsutism	**0.463**	**0.000**	19.847	0.998
Anxiety	-0.018	0.666	**1.342**	**0.002**
Depression	**0.172**	**0.000**	36.112	0.992
Psychological problem	**-0.273**	**0.000**	-19.861	0.995
Social problem	**-0.296**	**0.000**	-18.040	0.994
Food allergy	**0.193**	**0.000**	20.454	0.996
Heart burn	**0.258**	**0.000**	34.915	0.993
Irritable	0.162	0.002	2.428	0.000
IBS	0.039	0.677	17.404	0.998
Stress	0.011	0.906	-1.902	1.000
House animal	0.091	0.054	**2.067**	**0.002**
House cockroach	0.008	0.839	**-1.249**	**0.003**
House wool	0.032	0.439	-0.663	0.202
House spider	**0.243**	**0.000**	21.189	0.995
House grass	0.183	0.000	2.041	0.000
House tree	0.006	0.875	0.210	0.540
Family history of atopy	0.194	0.000	1.506	0.000

Variable	Regression		Logistic regression	
	B	**P value**	**B**	**P value**
Water cycle	-0.040	0.399	-0.195	0.599
Breast feeding	-0.066	0.187	**-0.931**	**0.030**
Illiterate father	-0.046	0.455	-0.285	0.517
Illiterate mother	0.003	0.964	-0.300	0.482
House painting	-0.007	0.897	0.488	0.209

Table 4. Comparison between regression and logistic regression for urban population in relation to allergy.

3.5. Asthma for rural study population

In rural population, the two models demonstrated agreement on significant negative association between asthma and risk factors such as: BMI; and crowding index. Table 123. In addition, agreement on significant association was achieved between asthma development and hirsutism. Table 5

Variable	Regression		Logistic regression	
	B	**P value**	**B**	**P value**
Oil drinking	-0.064	0.265	-19.440	0.998
BMI	-0.021	0.003	-0.881	0.011
Crowding index	-0.037	0.001	-1.118	0.002
Oil heating	**0.786**	**0.000**	-60.025	0.997
Gas heating	-0.143	0.107	-19.411	0.999
Air condition heating	-0.286	0.082	-38.822	0.999
Electricity heating	**-0.143**	**0.013**	-19.411	0.998
Fan cooling	**0.143**	**0.002**	19.411	0.997
Air condition cooling	0.143	0.273	19.431	0.999
Water cooling	**0.143**	**0.032**	19.411	0.998
Animal exposure	**1.154**	**0.000**	42.406	0.998
Child exposure to smoking	-0.032	0.486	-17.840	0.998
Child respiratory tract infection	**1.082**	**0.000**	-41.887	0.998
Child exposure to cockroach	**0.142**	**0.000**	-18.718	0.998
Child hitting	**0.222**	**0.000**	0.908	0.151
Child exposure agriculture dust	**0.064**	**0.033**	17.147	0.996
Child agriculture work	0.052	0.116	5.250	1.000

Variable	Regression		Logistic regression	
	B	P value	B	P value
Child physical activity	**-0.184**	**0.000**	12.134	1.000
Family history of asthma	-0.094	0.211	-1.864	1.000
Family history allergic rhinitis	-0.054	0.394	-23.981	1.000
Family history atopic dermatitis	**0.107**	**0.044**	-7.652	1.000
Aspirin use	**0.472**	**0.000**	13.042	1.000
School stress	**0.218**	**0.000**	-18.989	0.998
Child playing	-0.017	0.776	-0.163	1.000
Cold sore	**-0.248**	**0.000**	-19.438	0.998
Hirsutism	0.367	0.000	2.794	0.000
Anxiety	-0.036	0.351	54.328	0.991
Depression	**0.179**	**0.000**	-17.328	0.995
Food allergy	**0.786**	**0.000**	41.324	0.996
Heart burn	**0.250**	**0.000**	20.121	0.999
Irritable	**-0.214**	**0.000**	-20.211	0.998
IBS	**0.393**	**0.000**	-20.211	0.999
Stress	**0.250**	**0.000**	ND	
House animal	0.003	0.940	-37.041	0.997
House cockroach	-0.052	0.389	-19.617	0.996
House wool	**0.238**	**0.000**	0.368	1.000
House spider	**0.497**	**0.000**	58.734	0.994
House grass	**0.389**	**0.000**	58.715	0.995
House tree	**0.176**	**0.000**	-1.643	1.000
Family history of atopy	**0.172**	**0.000**	18.587	0.994
Breast feeding	**0.208**	**0.002**	57.968	0.995
Illiterate father	**0.280**	**0.000**	56.884	0.993
Illiterate mother	-0.078	0.097	-53.158	0.998
House painting	**0.108**	**0.001**	14.570	0.993

Table 5. Comparison between regression and logistic regression for rural population in relation to asthma.

3.6. Allergy for rural study population

Agreement on significant negative association was achieved between allergy development and BMI. However, agreement between the two models on significant association between allergy development and risk factors such as: family history of asthma; aspirin use; anxiety; presence of house wool and spider. Table 6

Variable	Regression		Logistic regression	
	B	P value	B	P value
Oil drinking	**0.631**	**0.000**	23.963	0.997
BMI	-0.051	0.000	-0.485	0.000
Crowding index	**-0.039**	**0.029**	-0.155	0.097
Oil heating	**0.476**	**0.047**	-1.598	0.085
Gas heating	**0.429**	**0.007**	21.141	0.999
Air condition heating	-0.476	0.106	-0.528	1.000
Electricity heating	0.095	0.353	**0.944**	**0.028**
Fan cooling	0.071	0.388	-0.069	0.834
Air condition cooling	-0.095	0.684	-0.944	1.000
Water cooling	**0.571**	**0.000**	21.264	0.998
Animal exposure	**0.580**	**0.002**	21.608	0.999
Child exposure to smoking	**0.292**	**0.017**	20.680	0.998
Child respiratory tract infection	-0.110	0.555	-0.592	1.000
Child exposure to cockroach	-0.009	0.915	0.065	0.857
Child hitting	**0.154**	**0.049**	0.446	0.212
Child exposure agriculture dust	0.057	0.469	-0.613	0.163
Child agriculture work	0.058	0.511	1.488	0.001
Child physical activity	0.001	0.984	0.680	0.045
Family history of asthma	0.326	0.009	1.535	0.018
Family history allergic rhinitis	**0.671**	**0.000**	38.009	0.997
Family history atopic dermatitis	-0.158	0.072	-0.066	0.905
Aspirin use	0.470	0.000	1.298	0.006
School stress	**0.548**	**0.000**	21.173	0.998
Child playing	-0.072	0.499	-19.038	0.998
Cold sore	**-0.308**	**0.003**	-0.238	0.695
Hirsutism	-0.062	0.486		
Anxiety	0.305	0.001	2.397	0.000
Depression	**0.321**	**0.001**	37.191	0.996
Food allergy	**0.214**	**0.035**	20.571	0.998
Heart burn	0.250	0.115	1.421	1.000
Irritable	**0.214**	**0.035**	20.571	0.998
IBS	-0.107	0.616	20.571	0.999

Variable	Regression		Logistic regression	
	B	P value	B	P value
Stress	0.150	0.115	1.421	1.000
House animal	-0.074	0.430	0.560	0.207
House cockroach	0.175	0.200	20.656	0.998
House wool	0.425	0.000	1.907	0.000
House spider	0.314	0.013	1.132	0.040
House grass	-0.017	0.901	-0.367	0.541
House tree	-0.064	0.355	-0.452	0.159
Family history of atopy	0.070	0.474	**0.868**	**0.021**
Breast feeding	**-0.309**	**0.037**	-20.495	0.998
Illiterate father	0.269	0.004		
Illiterate mother	**-0.269**	**0.012**	-0.267	0.424
House painting	0.045	0.552	-0.119	0.717

Table 6. Comparison between regression and logistic regression for rural population in relation to allergy

4. Discussion

Rates of asthma morbidity and mortality are increasing [4] and this increase contributed to environmental exposure. Asthma is a complex multifactorial disease in which allergic factors and non-allergic triggers interact, resulting in bronchial obstruction and inflammation [19]. Asthma is the leading chronic disease of children in industrial countries; however, the disease is also common in children in developing countries [19], and may be extended to involve adolescent. The pathogenesis and underlying causes of childhood asthma is not fully understood, however, early life environmental exposure and life style may be implicated in the etiology of asthma [20,21]. Sensitization induced by allergens is essential step for the development of asthma, however, asthma exacerbation correlated to outdoor and indoor allergens, while indoor allergens influence disease prevalence and severity [22] However, timing of such environmental exposure during early development may also be important in allergic sensitization and later asthma development [23]. Early exposure to endotoxin from farm environments has been associated with reduced childhood asthma risk [24], however, endotoxin exposure later in life may increase asthma occurrence especially in agricultural settings [25].

In the present study, influenza and common cold cause allergic disease exacerbation in 46.8% of secondary school girls. In addition, stress was the predominant (66%) factor that exacerbates allergy in secondary school girls, followed by outdoor air pollution (55.3%), animal exposure (36.2%) and house dust (34%). This finding agreed with literature that implicate viral infections, rather than bacterial infections as exacerbating factor for asthma [26,27]. However, with increasing age asthma exacerbation was mainly associated with other factors such as exercise due to decline in trigger role of respiratory infections with age in children [28].

Exposure to various constituents including tobacco smoke, wood smoke, air-born allergens, dust mites, mould, and other indoor pollutants is known or suspected to trigger wheezing or exacerbate asthma in children [27]. The level of exposure to these compounds differs in regional Iraq from the situation in developed societies, as children spend more time outdoors with increasing age. Despite the increased exposure to asthma triggers, there are few population-based data examining whether exposure to environmental factors may be associated with asthma in Iraqi adolescent.

Exposure to chemical substances and pesticides exacerbate asthma attack in 53.2% of cases with allergy. Taking these together with air pollution suggest that allergy exacerbated in all cases with these factors, indicated the importance of pollution in the control of allergic diseases. Furthermore, these findings clarify that Kirkuk governorate is an area with high pollution, which warranted application of pollution control program. There is no population based study for adolescent girls in Iraq to compare with. However, there was a population based study in children [4].

Alsamarai et al [4] found that exposure to wood, oil smoke, cats, dogs, herbicides or pesticides, and animal and farm environments were associated with an increased risk of asthma among children in Samara city, Iraq. The findings suggest that the aetiology of childhood asthma is complex and may include both early life environmental exposure and early allergic sensitization. Combustion of wood liberates nitrogen dioxide, carbon monoxide, sulfur dioxide and particulate matter, all of which have been associated with increased respiratory illness [29].

Exposure to oil smoke has been shown to significantly increase the risk of asthma [30], while particles from wood combustion significantly reduced lung function in elementary school children [31]. The present study indicated that oil heating was a significant risk factor for asthma development in adolescent girls. In contrast, both air condition and electricity heating were with negative impact on asthma development in adolescent girls. The results of this study are consistent with previous observations showing that early transient wheezing and/or increased airway reactivity in children and exposure to products of combustion may be important in the pathophysiology of asthma [20,32,33]. The girls exposure to animal exacerbate allergic diseases. However, animal exposure was not shows a significant association with asthma development for whole data and when sub divided into urban and rural community. Although, animal exposure was an important risk factors for allergy development in urban, rural and whole study population. Alsamarai et al [4] observed associations between exposure to cats and dogs and childhood asthma which are consistent with other studies [20,34-37], but contrast with other studies which found pets were protective [38,39]. Presence of cats, dogs, sheep and / or cattle with the house were with significant association with asthma development in secondary school girls in Kirkuk. A review of 32 articles suggested anon-significant increase in asthma risk of 11% was associated with the presence of pets in the first two years of life [40]. However, it is difficult to explore the association between exposure to pets and childhood asthma, even in prospective studies, because of issues of temporality and possible confounders associated with keeping pets [20].

A positive association has been reported between asthma among adults and the use of herbicides and pesticides [41,42], although data on pesticide exposure and childhood asthma are limited [20]. In the present study, exposure to either pesticides or herbicides was associated with an increased risk of asthma in adolescent girls. These results are consistent with report concerning primary school children in Iraq [4] and other geographical areas [20,43]. Several studies have suggested a reduced risk of asthma with exposure to a farming environment in early life [44]. It has been suggested that exposure to a farming environment causes higher levels of exposure to bacterial endotoxin, eventually leading to the production of several cytokines that shift the balance towards the Th1- over Th2- mediated immunity, thereby reducing asthma risk [24]. In the present study and previously reported one in Iraq [4], such an inverse association with farm exposure was not evident, as there was a significantly increased risk of asthma in adolescent girls and children with farm-related exposure. In contrast, previous studies have reported that growing up in a farming environment is associated with an increased risk of asthma and that endotoxin exposure may increase asthma risk [20]. The discrepancy between studies may be due to differences in farming practice, crops, lifestyle and other "rural" factors that differ between this Iraqi environment and that in Europe and other regions from which previous reports originated. A further difference in Iraq may be the proximity of stables to the home and time spent in stables [24]; in this population stables were mostly attached to the family home and sometimes located within the house.

The protective effect of breastfeeding on the development of asthma has raised substantial interest, but the scientific evidence relating to the effect of breastfeeding is controversial [45]. The epidemiological studies have provided controversial results showing negative association consistent with a protective effect, whereas some studies have reported either no association or a positive association between the duration of breast-feeding and the risk of asthma [46-48]. The present study indicated that breastfeeding is with a protective effect on development of asthma and allergy in secondary school girls. In contrast, breastfeeding is a risk factor for asthma development in Iraqi children [4]. Both methodological issues and the complexity of the phenomenon may be responsible for these contrasting results [49]. Differences in several factors, including; the age at which various diseases were experienced, hereditary factors as well as environmental factors may influence the association between breast-feeding and the development of asthma, thus explaining the conflicting results reported to date. The finding of the present study may differ from that reported for developed countries because of variations in the duration of breast-feeding; generally about two years in Iraq. In addition, there is the potential for incorporation of local environmental pollutants into breast milk.

The duration of breast-feeding varies substantially in the reported studies, which becomes critical when fitting the variable if the relation is non-linear as previously suggested [49]. The duration of follow up and the age of onset of asthma are also important, as if breast-feeding could delay the onset of asthma, the prevalence of current asthma would be lower among breast-fed than non-breastfed young children, but similar in later life [19]. There is evidence that hereditary asthma or atopic disease [49] and exposure to environmental factors can modify the relation between the duration of breast-feeding and the risk of asthma. [4]. The controversial results referred to above may relate to the non-linear relation between the duration of breastfeeding and the risk of asthma [49].

The finding in this study is of a significant association between food allergy and asthma in adolescent girls is consistent with that reported by others in children [50]. Similarly, the association between a family history of atopy and asthma and developing asthma, with the association higher for asthma than for atopy was consistent with findings of others [4,51,52]. These study findings strengthen earlier reports suggesting that genetics might play an important role in the development of asthma in childhood [53], with parental asthma being the strongest determinant of asthma. The current study also adds to the literature suggesting that exposure to environmental tobacco smoke increases the risk of adolescent and childhood asthma [4,53].

Reported studies suggest that home environment may act as a risk factor for triggering of asthmatic attach and/or asthma development [54-56], in addition violence may be an asthma attack risk factor [57]. The present study indicated that child hitting by their parents was a significant risk factor for asthma development in urban and rural population when analyzed separately. In addition, stress was a significant risk factor for asthma development in Kirkuk adolescent girls.

Inflammatory mediators released as an outcome of stress and subsequently potentiate allergen induced responses [57,58]. Asthma may be prevented by primary and secondary approaches, however, the physicians mostly relies on performing secondary prevention approach. Our present study indicated that the predominant exacerbating factors are stress, pollution and animal exposure, all can be controlled through a healthcare and social programs and health education.

Studies in literature indicated an association between indoor and outdoor air pollutants and the evidences of such association were variable between the studies [59-66].

The present study indicated that smoking was responsible for exacerbation of allergic diseases in 19.1% of adolescent girls. However, child exposure to tobacco smoke is not a significant risk factor for development of asthma and other allergic diseases in adolescent girls. But when the data is collected together, linear regression analyses and not logistic regression analyses, shows a significant association between tobacco smoke exposure during childhood and development of allergic diseases [any one] in adolescent girls. In a previous study reported for Iraq, family history of smoking was associated with asthma (OR=1.52, 95% CI 1.17-1.97; P=0.001) [4].

Other studies suggested the association between asthma development and exacerbation and exposure to tobacco smoke [67-86].

The Institute of Medicine concluded that cockroach allergens are causally related to asthma attacks. [63] Our present study indicated that exposure to cockroach form 12.8% as exacerbating factor of asthma in adolescent girls. In addition, cockroach exposure during childhood was with significant association to development of allergy in Kirkuk population (Linear regression), urban population (Linear and Logistic regression), and rural community (Linear regression). Furthermore, present house presence of cockroach was significant risk factor for development of allergy in Kirkuk population (Linear regression), urban community (Logistic regression), but not for rural community. This could be explained on the basis that the density of cockroach is more in urban than in rural communities. In Kirkuk urban community, present house presence of cockroach was with highly significant association with asthma development (Linear and Logistic regression) in adolescent girls.

Our present study indicated that mold was responsible for 17% as exacerbating factor for asthma in adolescent girls a finding that was consistent to that reported by others [63,66]. By using both Linear regression and Logistic regression models, asthma development in adolescent girls in Kirkuk, Iraq, was with positive association with risk factors that include: oil drinking during childhood, oil heating, fan cooling, child respiratory tract infection, child exposure to agricultural dust and work, family history of asthma, child activity, depression, food allergy, heartburn, IBS, stress, presence of house wool, presence of grass and tree within house, family history of atopy, and presence of water cycle within house. However, when the data of urban and rural communities were analyzed separately, asthma development in urban community was associated with risk factors such as child respiratory tract infections, child hitting by his parents, child exposure to agriculture dust and work, child activity, aspirin use, heart burn, house presence of animal and cockroach, family history of atopy, and ho, family history of atopy, and house presence of wool and tree. The pattern for risk factors for asthma development in adolescent girls rural community was different, indicating that there are differences in risk factors influence between urban and rural population.

Several risk factors have been identified as protective against asthma. The present study indicated an inverse association between crowding index and development of asthma in adolescent girls (Linear regression), urban population (Logistic regression), and rural population (Linear and logistic regression). The same pattern was demonstrated for allergic diseases pooled together. *Ball et al* [56] *showed that exposure of young children to older children at home or to other children in child care settings protects against the development of asthma and frequent wheezing later in childhood. They hypothesized that within the first 6 months of an infant's life, the immune response of children without atopy shifts from one associated predominantly with type 2 helper T cells, such as that in adults with atopic illnesses, toward one based more on cytokines derived from type 1 helper T cells, such as that in adults without atopy.* [56,87] This could explain the prominent association between crowding index and asthma development in rural community in our study, since early exposure of young children to old one are more common in rural community. Riedler et al [88] study suggest that early-life long time exposure to stables and farm milk induces a strong protective effect against development of asthma, hay fever, and atopic sensitization. An interesting findings of this study was that house ownership, air condition heating, child residence in rural area, electricity heating, and breast feeding were acting as protective factors for development of asthma and allergic diseases in adolescent girls living.

The present study findings and the reported studies have documented that a decrease in allergic impact of environmental exposure can be achieved by application of specific interventions and subsequently may control asthma attack. However, many children and their families, particularly children who live in poverty and rely on emergency departments as their primary source of health care, and the decline in healthcare delivery in Iraq after the American invasion, may not be receiving adequate counseling about how to avoid environmental exposures. Furthermore, performing a campaigns of educational programs for parents and

http://dx.doi.org/10.5772/59376

individual with asthma about environmental controls may play an important role in asthma prevention, control and management [89-91].

To prevent unnecessary exposures to outdoor air pollution, clinicians may provide appropriate guidance to asthmatic subjects and their parents regarding exercise during periods of high pollution. With proper management, many environmental exposures can be decreased. [92]

Some researchers have shown links between exposure to allergens, pollutants and respiratory symptoms, while in contrast some other researchers have demonstrated that better hygiene and clean indoor environment may contribute to the increased prevalence of allergic diseases and respiratory symptoms. The present study will enhance our understanding and knowledge with regard to the two different hypotheses related to asthma and respiratory symptoms.

The study is significant for several reasons: (1)- Address the influence of different variables on prevalence of respiratory symptoms among secondary school students in Iraq. (2)- Assess the extent to which personal, environmental, socio-economic factors and indoor air pollution will affect the prevalence of respiratory symptoms in school students. (3)- Enhance our knowledge and understanding about the two contrasting theories; the hygiene theory and the theory that higher exposure to air pollutants and allergens is related to asthma and respiratory symptoms. (4)- Summarize the preventive measures to reduce exposure to air pollution and allergens in school environments located in different and also efforts in improving indoor air quality of schools thus reducing the absenteeism and respiratory symptoms in students.(5). Clarify the air pollution impact on health of Kirkuk community.

Author details

Ramla Hamed Ahmed Alobaidi[1], Amina Hamed Ahmed Alobaidi[2] and Abdulghani M. Alsamarai[3*]

*Address all correspondence to: galsamarrai@yahoo.com

1 Al Mansour Constructive Company, Kirkuk, Iraq

2 Tikrit University College of Medicine, Tikrit, Iraq

3 Tikrit Teaching Hospital, Tikrit, Iraq

References

[1] Bernstein, JA, Neil Alexis, Hyacinth Bacchus, Leonard Bernstein, Pat Fritz, Elliot Horner, Ning Li, Stephany Mason, Andre Nel, John Oullette, Kari Reijula, Tina Repo-

nen, James Seltzer, Alisa Smith, Susan M. Tarlo. The health effects of nonindustrial indoor air pollution. J Allergy Clin Immunol 2008;121:585-91.

[2] Khare M. Air pollution, monitoring, modeling and health. InTech Janeza Trdine 9, 51000 Rijeka, Croatia. 2012

[3] Alsamarai AGM, Alwan AM, Ahmad AH, Salih MA, Salih JA, Aldabagh MA, Alturaihi S, Abdulaziz ZH, Salih AA, Salih SK and Murbat MM. The relationship between asthma and allergic rhinitis in the Iraqi population. Allergology International 2009;58: 549-555.

[4] Alsamarai AGM, Salih MA, Alobaidi AHA, Alwan AM, Abdulaziz ZH. Risk factors for asthma in Iraqi children. Jour Rural Tropical Public Health 2009; 8:45-52.

[5] Alsamarai AGM. Infectious diseses fellowship program: proposed model for establishment in Iraqi Universities. Ann Iraqi Sci 2008;1:319-324.

[6] Mendell MJ, Heath GA. Do indoor pollutants and thermal conditions in schools influence student performance? A critical review of the literature. Indoor Air 2005;15:27-52.

[7] Rumchev, K., J. Spickett, et al. Association of domestic exposure to volatile organic compounds with asthma in young children. Thorax. 2004;59: 746-751

[8] Zhao Z, Zhang Z, Wang Z, Ferm M, Liang Y, Norbck D. Asthmatic symptoms among pupils in relation to winter indoor and outdoor air pollution in schools in Taiyuan, China. Environ Health Perspect 2008;116:90–97.

[9] McGwin G, Lienert J, Kennedy JR. Formaldehyde exposure and asthma in children: a systematic review. 2009. http://www.scielosp.org/pdf/csc/v16n9/a20v16n9.pdf

[10] Parker, J. Reducing asthma triggers in schools: Recommendations for Effective Policies, Regulations, and Legislation. NEW SOLUTIONS: A Journal of Environmental and Occupational Health Policy 2006;16(1): 87 - 105.

[11] Basagaña, X., J. Sunyer, Manolis Kogevinas, Jan-Paul Zock, Enric Duran-Tauleria, Deborah Jarvis, Peter Burney, and Josep Maria Anto on Behalf of the European Community Respiratory Health Survey. Socioeconomic Status and Asthma Prevalence in Young Adults. American Journal of Epidemiology 2004;160:178-188.

[12] Rona, R. J. Asthma and poverty. Thorax 2000;55: 239-244

[13] Weitzman, M., A. Sobol, et al. Racial, social, and environmental risks for childhood asthma. Am J Dis Child 1990;144(11): 1189-1194.

[14] Ring J. Davos Decleration: Allergy as a global problem. Allergy 2012;67:141143.

[15] Bousquet J, Anto J, Auffray C, Akdis M, Cambon-Thomsen A, Keil T, et al. MeDALL (Mechanisms of the Development of ALLergy): an integrated approach from phenotypes to systems medicine. Allergy 2011;66:596–604.

[16] Pawankar R, Canonica GW, Holgate ST, Lockey RF eds. World Allergy Organization (WAO) White Book on Allergy. WAO, Milwaukee, 2011; 1–216.

[17] Heinzerling L, Burbach G, van Cauwenberge P, Papageorgiou P, Carlsen KH, Lødrup Carlsen KC et al. Establishing a standardized quality management system for the European Health Network GA2LEN. Allergy 2010;65:743–752.

[18] Alsamarai AGM, et al. Development of air pollution index for Kirkuk Governorate. Ann Iraqi Sci 2008;1:231-235

[19] Busse WW, Holgate ST. Asthma and Rhinitis. 2nd ed., Vol: 1; Oxford, Blackwell Science 245-841.2003.

[20] Salam MT, Li Yu-Fen, Langholz B, Gilliland FD. Early life environmental risk factors for asthma: findings from children's health study. Environmental Health Perspective 2004;112: 760-5.

[21] Johnson CC, Ownby DR, Zoratti EM, Alford SH, Williams LK, Joseph CL. Environmental epidemiology of pediatric asthma and allergy. Epidemiology Review 2002; 24:154-75.

[22] Adkinson NF, Yunginger JW, Busse WW, Bochner BS, Holgate ST, Simon FE. Middletons Allergy: Principles and Practice. 6th ed. Vol: Two, USA, Mosby; 1175-208.2000.

[23] Melen E, Wickman M, Nordvall SL, van Hage-Hamsten M, Lindfors A. Influence of early and current environmental exposure factors on sensitization and outcome of asthma in pre-school children. Allergy 2001;56: 646-52.

[24] Braun-Fahrlander C. The role of the farm environment and animal contact for the development of asthma and allergies. Clinical Experimental Allergy 2001;31:1799-803.

[25] Schwartz DA. Does inhalation of endotoxin cause asthma? American Journal Respiratory Critical Care Medicine 2001;163: 305-6.

[26] Johnston SL. Mechanisms of asthma exacerbation. Clinical Experimental Allergy 1998;28(suppl): 181-6.

[27] Weiss ST. Environmental risk factors in childhood asthma. Clinical Experimental Allergy 1998;28(suppl): 29-34.

[28] Sarafino EP, Pterson ME, Murphy EL. Age and impacts of triggers in childhood asthma. Journal Asthma 1998;35: 213-7.

[29] Larson TV, Koenig JQ. Wood smoke: emissions and no cancer respiratory effects. Annals Review Public Health 1994;15:133-56.

[30] Chen P, Yu R, Hou X, Tan P, Xie H, Kong L, et al. Epidemiological survey on bronchial asthma in Liaoning province. ZhoghuaJie He Hu Xi ZaZhi 2002;25: 603-6.

[31] Koenig JQ, Larson TV, Hanley QS, Rebolledo V, Dumler K, Checkoway H, et al. Pulmonary function changes in children associated with fine particulate matter. Environmental Research 1993;63: 26-38.

[32] Belanger K, Beckett W, Triche E, Bracken MB, Holford T, Ren P, et al. Symptoms of wheeze and persistent cough in the first year of life: associations with indoor allergens, air contaminants, and maternal history of asthma. American Journal of Epidemiology 2003;158: 195-202.

[33] Sotir M, Yeatts K, Shy C. Presence of asthma risk factors and environmental exposures related to upper respiratory infection- triggered wheezing in middle school age children. Environmental Health Perspectives 2003;111:657-62.

[34] Nafstad P, Magnus P, Gaarder Pl, Jaakkola JJ. Exposure to pets and atopy-related diseases in the first 4 years of life. Allergy 2001;56: 307-12.

[35] Ronmark E, Perzanowski M, Platts-Mills T, lundback B. Incidence rates and risk factors for asthma among schoolchildren: a 2-years follow-up report from the obstructive lung diseases in Northern Sweden (OLIN) studies. Respiratory Medicine 2002;96: 1006-13.

[36] McConnell R, Berhane K, Gilliland F, Islam T, Gauderman WJ, London SJ, et al. Indoor risk factors for asthma in a prospective study of adolescents. Epidemiology 2002;13: 288-95.

[37] Zheng T, Niu S, Lu B, Fan X, Sun F, Wang J, et al. Childhood asthma in Beijing, China: a population-based case-control study. American Journal Epidemiology 2002;156: 977-83.

[38] Hesselmar B, Aberg N, Aberg B, Eriksson B, Bjorksten B. Does early exposure to cat or dog protect against later allergy development? Clinical Experimental Allergy 1999;29: 611-7.

[39] Remes ST, Castro-Rodriguez JA, Holberg CJ, Martinez FD, Wright AL. Dog exposure in infancy decreases the subsequent risk of frequent wheeze but not of atopy. Journal of Allergy Clinical Immunology 2001;108: 509-15.

[40] Apelberg BJ, Aoki Y, Jaakkola JJ. Systematic review: exposure to pets and risk of asthma and asthmalike symptoms. Journal of Allergy and Clinical Immunology 2001;107:455-60.

[41] . Bener A, Lestringant GG, Beshwari MM, Pasha MA. Respiratory symptoms, skin disorders and serum IgE levels in farm workers. Allergy Immunology (Paris) 2001;31: 52-6.

[42] Hoppin JA, Umbach DM, London SJ, Alavanja MC, Sandler DP. Chemicals predictors of wheeze among farmer pesticide applicators in the Agricultural Health Study. American Journal Respiratory Critical Care Medicine 2002;165: 683-9.

[43] Karmaus W, Kuehr J, Kruse H. Infections and atopic disorders in childhood and organochlorine exposure. Archives of Environmental Health 2001;56: 485-92.

[44] Von Ehrenstein OS, Von Mutius E, Illi S, Baunmann L, Bohm O, Von Kries R. Reduced risk of hay fever and asthma among children of farmers. Clinical Experimental Allergy 2000;30: 187-93.

[45] Friedman NJ. Zeiger RS. The role of breastfeeding in development of allergies and asthma. Journal of Allergy Clinical Immunology 2005;115: 1238-48.

[46] Chulada PC, Arbes SJ, Dunson D, Zeldin DC. Breastfeeding and the prevalence of asthma and wheezing in children: analysis from the third national health and nutrition examination survey, 1988-1994. Journal of Allergy Clinical Immunology 2003;111: 328-36.

[47] Nafstad P, Jaakola JJK. Breast feeding, passive smoking and asthma and wheeze in children. Journal of Allergy Clinical Immunology 2003;112: 807-8.

[48] Oddy WH, Sherriff JL, DE Klerk NH, et al.The relation of breastfeeding and body mass index to asthma and atopy in children: a prospective cohort study to age 6 years. American Journal of Public Health 2004; 94: 1531-7.

[49] Fredriksson P, Jaakola N, Jaakola JJK. Breastfeeding and childhood asthma: a six year population based cohort study. B M C Pediatrics 2007; 7:39-46.

[50] Simpson AB, Glutting J, Yousef E. Food allergy and asthma morbidity in children. Pediatric Pulmonology 2007;42: 489-95.

[51] Bjerg A, Hedman L, Perzanowski MS, Platt-Mills T, Lundback B, Ronmark E. Family history of asthma and atopy: in depth analyses of the impact on asthma and wheeze in 7 to 8 year old children. Pediatrics 2007;120:741-8.

[52] Palvo F, Toledo EC, Menin AMCR, Jorge PPO, Godoy MF, Sole D. Risk factors of childhood asthma in Sao Jose do Rio Preto, Sao Paulo, Brazil. Journal Tropical Pediatrics 2008;54: 253-7.

[53] Jaakola JJK, Nfstad P, Magnus P. Environmental tobacco smoke, parental atopy and childhood asthma. Environmental Health Perspective2001; 109: 579-82.

[54] Cullinan P, Taylor AJ. Asthma in children: environmental factors. BMJ. 1994;308:1585–1586

[55] Holt PG, Macaubas C, Stumbles PA, Sly PD. The role of allergy in the development of asthma. Nature. 1999;42(6760 suppl):B12–B17

[56] Ball TM, Castro-Rodriguez JA, Griffith KA, Holberg CJ, Martinez FD, Wright AL. Siblings, day-care attendance, and the risk of asthma and wheezing during childhood. N Engl J Med. 2000;343:538–543

[57] Wright RJ, Steinbach SF. Violence: an unrecognized environmental exposure that may contribute to greater asthma morbidity in high risk inner-city populations. Environ Health Perspect. 2001;109:1085–1089

[58] Wright RJ, Rodriguez M, Cohen S. Review of psychosocial stress and asthma: an integrated biopsychosocial approach. Thorax. 1998;53:1066–1074

[59] Figley KD, Elrod RH. Endemic asthma due to castor bean dust. JAMA. 1928;90:79–82

[60] Sunyer J, Anto JM, Rodrigo MJ, Morrell F. Case-control study of serum immunoglobulin-E antibodies reactive with soybean in epidemic asthma. Lancet. 1989;1:179–182

[61] Anto JM, Sunyer J, Rodriguez-Roisin R, Suarez-Cervera M, Vazquez L. Community outbreaks of asthma associated with inhalation of soybean dust. N Engl J Med. 1989;320:1097–1102

[62] Anto JM, Sunyer J, Reed CE, et al. Preventing asthma epidemics due to soybeans by dust-control measures. N Engl J Med. 1993;329:1760–1763

[63] Institute of Medicine, Committee on the Assessment of Asthma and Indoor Air. Clearing the Air: Asthma and Indoor Air Exposures. Washington, DC: National Academy Press; 2000

[64] Etzel RA. Indoor air pollution and childhood asthma: effective environmental interventions. Environ Health Perspect. 1995;103(suppl 6): 55–58

[65] Platts-Mills TA. Allergen-specific treatment for asthma: III. Am Rev Respir Dis. 1993;148:553–555

[66] Pope AM, Patterson R, Burge H, et al, eds. Indoor Allergens: Assessing and Controlling Adverse Health Effects. Institute of Medicine, Committee on the Health Effects of Indoor Allergens. Washington, DC: National Academy Press; 1993

[67] Matthews TJ. Smoking during pregnancy in the 1990s. Natl Vital Stat Rep. 2001;49:1–14

[68] Tager IB, Hanrahan JP, Tosteson TD, et al. Lung function, pre- and post-natal smoke exposure, and wheezing in the first year of life. Am Rev Respir Dis. 1993;147:811–817

[69] Pirkle JL, Flegal KM, Bernert JT, Brady DJ, Etzel RA, Maurer KR. Exposure of the U.S. population to environmental tobacco smoke: the Third National Health and Nutrition Examination Survey, 1988–1991. JAMA. 1996;275:1233–1240

[70] American Academy of Pediatrics, Committee on Environmental Hazards. Involuntary smoking—a hazard to children. Pediatrics. 1986;77: 755–757

[71] Murray AB, Morrison BJ. The effect of cigarette smoke from the mother on bronchial responsiveness and severity of symptoms in children with asthma. J Allergy Clin Immunol. 1986;77:575–581

[72] Evans D, Levison J, Feldman CH, et al. The impact of passive smoking on emergency room visits of urban children with asthma. Am Rev Respir Dis. 1987;135:567–572

[73] Burchfiel CM, Higgins MW, Keller JB, Howatt WF, Butler WJ, Higgins IT. Passive smoking in childhood: respiratory conditions and pulmonary function in Tecumseh, Michigan. Am Rev Respir Dis. 1986;133:966–973

[74] Chilmonczyk BA, Salmun LM, Megathlin KN, et al. Association between exposure to environmental tobacco smoke and exacerbations of asthma in children. N Engl J Med. 1993;328:1665–1669

[75] Ehrlich R, Kattan M, Godbold J, et al. Childhood asthma and passive smoking. Urinary cotinine as a biomarker of exposure. Am Rev Respir Dis. 1992;145:594–599

[76] Holberg CJ, Wright AL, Martinez FD, Morgan WJ, Taussig LM. Child day care, smoking by caregivers, and lower respiratory tract illness in the first 3 years of life. Group Health Medical Associates. Pediatrics. 1993;91:885–892

[77] Krzyzanowski M, Quackenboss JJ, Lebowitz MD. Chronic respiratory effects of indoor formaldehyde exposure. Environ Res. 1990;52:117–125

[78] Martinez FD, Cline M, Burrows B. Increased incidence of asthma in children of smoking mothers. Pediatrics. 1992;89:21–26

[79] Murray AB, Morrison BJ. Passive smoking by asthmatics: its greater effect on boys than girls and on older than on younger children. Pediatrics. 1989;84:451–459

[80] O’Connor GT, Weiss ST, Tager IB, Speizer FE. The effect of passive smoking on pulmonary function and nonspecific bronchial responsiveness in a population-based sample of children and young adults. Am Rev Respir Dis. 1987;135:800–804

[81] Oldigs M, Jorres R, Magnussen H. Acute effects of passive smoking on lung function and airway responsiveness in asthmatic children. Pediatr Pulmonol. 1991;10:123–131

[82] Rylander E, Pershagen G, Eriksson M, Bermann G. Parental smoking, urinary cotinine, and wheezing bronchitis in children. Epidemiology.1995;6:289–293

[83] Sherman CB, Tosteson TD, Tager IB, Speizer FE, Weiss ST. Early childhood predictors of asthma. Am J Epidemiol. 1990;132:83–95

[84] Weitzman M, Gortmaker S, Walker DK, Sobol A. Maternal smoking and childhood asthma. Pediatrics. 1990;85:505–511

[85] Murray AB, Morrison BJ. The decrease in severity of asthma in children of parents who smoke since the parents have been exposing them to less cigarette smoke. J Allergy Clin Immunol. 1993;91:102–110

[86] Centers for Disease Control and Prevention. Strategies for reducing exposure to environmental tobacco smoke, increasing tobacco-use cessation, and reducing initiation

in communities and health-care systems.MMWR Morb Mortal Wkly Rep. 2000;49(RR-12):1

[87] Prescott SL, Macaubas C, Smallcombe T, Holt BJ, Sly PD, Holt PG. Development of allergen-specific T-cell memory in atopic and normal children. Lancet. 1999;353:196–200

[88] Riedler J, Braun-Fahrlander C, Eder W, et al. Exposure to farming in early life and development of asthma and allergy: a cross-sectional survey. Lancet. 2001;358:1129–1133

[89] Jones AP. Asthma and the home environment. J Asthma. 2000;37:103–124

[90] Environmental controls and lung disease. Report of the ATS Workshop on Environmental Controls and Lung Disease. Santa Fe, New Mexico, March 24–25, 1988. Am Rev Respir Dis. 1990;142:915–939

[91] Ingram JM, Heymann PW. Environmental controls in the management of asthma. Immunol Allergy Clin North Am. 1993;13:785–801

[92] Etzel RA. Environmental Exposures Influence the Development and Exacerbation of Asthma. Pediatrics 2003;112:233–239.