Yonsei Medical Journal
Vol. 44, No. 2, pp. 313 - 322, 2003

Effect of Bedding Control on Amount of House Dust Mite Allergens, Asthma Symptoms, and Peak Expiratory Flow Rate

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This quasi-experimental study was designed to investigate the effect of bedding control on the amount of house dust mite (HDM) allergens, asthma symptoms, and peak expiratory flow rate (PEFR) in asthmatics sensitive to HDMs. The subjects in the study were drawn from patients receiving treatment at the allergy clinics of three university-affiliated hospitals in Seoul. Forty-two patients without prior practice of the bedding control used in this study were selected. They commonly showed bronchial asthma caused by HDMs, and exhibited strong positive points (more than 3 points) in skin prick test (D. farinae, D. pteronyssinus), and positive response in both fluoro-allergosorbent test (FAST), and PC20 methacholine test. Of the subjects, alternatively, 22 were assigned to the experimental group and 20 to control group.

Bedding control consisted of the use of outer cotton covers, boiling them for 10 minutes fortnightly, and disinfecting bedding by sunlight fortnightly. The experimental group was under bedding control for 4 weeks. The data were collected from October 2000 to January 2001.

The results were as follows:

1. After bedding control, the total amount of HDM allergens decreased significantly in the experimental group. However there was no significant difference in the decrease of the amount of HDM allergens between the two groups.

2. Of the asthma symptoms, there was significant difference only in the decrease of the frequency of dyspnea, and in the increase of sleeping disturbance between the two groups after bedding control.

3. After bedding control, PEFR increased in the experimental group whereas it decreased in the control group. However, neither change was significant.

The above findings indicate that bedding control improved several asthma symptoms in asthmatics sensitive to HDMs. Accordingly, we suggest that bedding control is adopted as a useful nursing intervention in the field.

Key Words: Bedding control, house dust mite allergens, asthma symptoms, peak expiratory flow rate

Received November 12, 2002
Accepted December 30, 2002

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INTRODUCTION

The incidence of allergic diseases is increasing throughout the world, presumably due to changes in living and working environments caused by economic development and industrialization. Among these allergic diseases, the incidence of asthma has been increas ing drastically over the past 50 years.[1,2]

Bronchial asthma is a chronic infection in the bronchus in which the infection is related with various cells such as mast cells and eosinophils, inflammatory mediating substances, cytokines, and neurogenic mechanisms. In this chronic respiratory disease, a hypersensitive reaction of the airway and airway spasm is induced so that treatment becomes difficult, and even death can result when the condition becomes serious.[3]

Bronchial asthma is described as either intrinsic or extrinsic according to its causes. Extrinsic allergic bronchial asthma is due to external factors such as HDMs, dust, pollen, animal dander, feathers, food, and synthetic drugs. More than 60% of bronchial asthma in adults in Korea is due to HDM antigen.[4]

The main HDM antigen causing asthmatic reactions are group I and group II antigen. When the level of group I antigen is over 2,000 ng/g,

sensitization and airway hyperresponsiveness occurs against the antigen, at over 10,000 ng/g, the risk of acute asthma symptoms increases.[5]

The most important treatment for allergic diseases such as asthma is to control the number of HDMs by creating an environment adverse to their growth.[6,7]

The number of HDMs and the level of HDM antigen are affected by regional and seasonal changes, and environmental characteristics within the house.[8] HDMs proliferate explosively with little sunlight at temperatures around 25℃ and relative humidity between 75-85%. The level of HDM antigen is high in bedding and carpets where these conditions are met.[9] It was reported that the level of HDM antigen with air in the bedroom is about 10 times higher when people are sleeping compared with air in the living room during daytime.[10] Therefore, patients easily inspire HDM antigen in bedrooms where people spend the most time in a day.[11]

Although there are many methods of controlling the environment to decrease the number of HDMs and level of HDM antigen, a significant difference is seen in the extent to which these methods are fulfilled due to various factors such as economic burden which introduce procedural or methodical difficulties when the patient or family members are trying to control the environment.[12] Thus, increasing the degree of accomplished of a preventive method is possible through the development of an environment-controlling method that patients and their family members can easily utilize. Therefore, the present study was conducted to determine the effects of the traditional bedding control method, which is inexpensive and familiar to Korean people, on the level of HDM antigen, asthmatic symptoms, and peak expiratory flow rate (PEFR) based on the results of previous studies.

MATERIALS AND METHODS

Research design

This quasi-experimental study was designed to investigate the effect of bedding control on the amount of HDM allergens, asthma symptoms and PEFR in HDM sensitive asthmatics (Fig. 1).

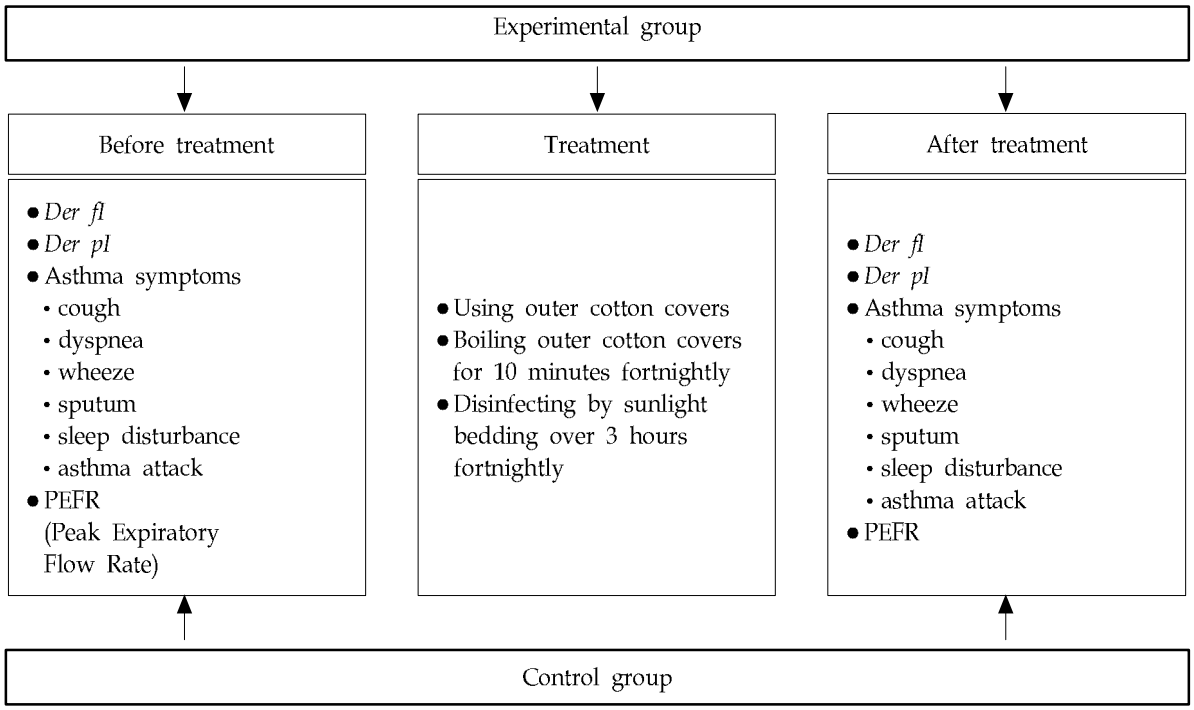

Fig. 1. Research design.

The independent variable in this study was bedding control and dependent variables were the amount of HDM allergens, asthma symptoms, and PEFR.

Only routine medical instruction was given to the control group, while four weeks of bedding control was provided to the experimental group by a researcher in addition to routine medical care.

The bedding control used in the present study consisted of boiling the outer cotton covers for 10 min and disinfecting the bedding by sunlight for more than 3 h every 2 weeks. This method was chosen as it is the traditional Korean method of treating bedding, and hence easily performed by the subjects; this method would be potentially effective for removing heat-resistant group II antigen.

Subjects and samples

The subjects in the study were drawn from patients receiving treatment at the allergy clinics of three university-affiliated hospitals in Seoul. Forty-two patients without prior practice of the bedding control used in this study who agreed to participate were selected. The subjects commonly showed bronchial asthma caused by HDMs, and exhibited strong positive points (more than 3 points) in skin prick test (*D. farinae, D. pteronyssinus*), and positive response in both fluoro-allergosorbent test (FAST), and PC20 methacholine test. Forty-two of the initial 60 subjects completed the study; 18 (30%) dropped out, 3 due to work, 1 due to disease, and 14 from a lack of desire to participate. Of the remaining subjects, 22 and 20 were randomly assigned to the experimental and control groups, respectively. No significant difference was initially present between the two groups in general characteristic factors, environmental factors, the amount of HDM allergens, asthma symptoms except the frequency of dyspnea, or PEFR, which might have affected the prognosis of asthma (Table 1-5). Dust samples were collected from each subject's bedding and bedroom floor by a researcher before and after bedding control. The same researcher collected dust for 2 min/square meter, in the morning

Table 1. Homogeneity Test for Subject Characteristics Between Experimental and Control Groups (n=42)

Characteristics	Exp. (n=22)	Cont. (n=20)	Total	χ^2	p
	N (%)	N (%)	N (%)		
Gender					
Male	14 (63.6)	9 (45.0)	23 (54.8)	1.47	0.226
Female	8 (36.4)	11 (55.0)	19 (45.2)		
Age (yrs)					
< 30	9 (40.9)	9 (45.0)	18 (42.9)	0.07	0.789
≥ 30	13 (59.1)	11 (55.0)	24 (57.1)		
Family history					
Yes	8 (36.4)	7 (35.0)	15 (35.7)	0.01	0.927
No	14 (63.6)	13 (65.0)	27 (64.3)		
Immunotherapy					
Yes	8 (36.4)	7 (35.0)	15 (35.7)	0.01	0.927
No	14 (63.6)	13 (65.0)	27 (64.3)		
Duration of asthma (yrs)					
< 3	15 (68.2)	10 (50.0)	25 (59.5)	1.44	0.377
≥ 3	7 (31.8)	10 (50.0)	17 (40.5)		
Total	22 (100.0)	20 (100.0)	42 (100.0)		

Exp., Experimental group; Cont., Control group.

Table 2. Homogeneity Test for Environmental Subject Characteristics Between Experimental and Control Groups (n=42)

Characteristics	Exp.(n=22)	Cont. (n=20)	Total	χ^2	p
	N(%)	N(%)	N(%)		
Type of residence					
Apartment	13 (59.1)	9 (45.0)	22 (52.4)	0.83	0.361
House	9 (40.9)	11 (55.0)	20 (47.6)		
Mean temperature in bedroom (℃)					
< 25	17 (77.3)	17 (85.0)	34 (81.0)	0.41	0.7
≥ 25	5 (22.7)	3 (15.0)	8 (19.0)		
Mean relative humidity in bedroom (%)					
< 45	10 (45.5)	10 (50.0)	20 (47.6)	0.09	0.768
≥ 45	12 (54.5)	10 (50.0)	22 (52.4)		
Living with pet					
Yes	5 (22.7)	10 (50.0)	15 (35.7)	3.39	0.065
No	17 (77.3)	10 (50.0)	27 (64.3)		
Bed in bedroom					
Yes	19 (86.4)	12 (60.0)	31 (73.8)	3.77	0.081
No	3 (13.6)	8 (40.0)	11 (26.2)		
Days after bedding washing					
< 15	12 (54.5)	7 (35.0)	19 (45.2)	1.62	0.204
≥ 15	10 (45.5)	13 (65.0)	23 (54.8)		
Total	22 (100.0)	20 (100.0)	42 (100.0)		

Exp., Experimental group; Cont., Control group.

Table 3. Homogeneity Test for Subject Distribution According to the Amount of House Dust Mite Allergens between Experimental and Control Groups (n=42)

Group	HDMA	Before	χ^2	p
	(ng/g)	N(%)		
Exp.	< 10000	5 (22.7)		
	≧ 10000	17 (77.3)	0.77	0.379
Cont.	< 10000	7 (35.0)		
	≧ 10000	13 (65.0)		

Exp., Experimental group; Cont., Control group.
HDMA, House Dust Mite Allergens.

before cleaning the room, with an electric vacuum cleaner (V-C601AQ, LG Electric Co, Seoul, Korea). The filters were changed according to bedding and bedroom floor. The filters were stored in sealed plastic bags at -20℃ before measurement of HDM allergens. Bedding control consisted of the use of outer cotton covers, boiling them for 10 minutes fortnightly and disinfecting bedding by exposure to sunlight fortnightly. The experimental group was under bedding control for a 4 week period. The data were collected from October 2000 to January 2001.

Measurements

The HDM antigen sample was obtained from the bedroom of each subject by collecting dust from the bedding and bedroom floor and the amount of the collected sample was measured using a Chapman kit, up to the maximum of 48 mg. The sample was combined with 1.2 ml of borate buffered saline (pH 8.5) to a sample amount of up to 24 mg and 2.4 ml of borate-buffered saline to a sample amount of 25 - 48 mg. These samples solutions were shaken at room temperature for 18 h to extract the antigen. The supernatant was collected after shaking and the

Table 4. Homogeneity Test for Frequency of Asthmatic Symptoms between Experimental and Control Groups (freq/week)

Group	Before		Z[b]
	Mean ± SD	(Range)	
Cough			
Exp.	41.14 ± 81.68	(290.00)	0.52
Cont.	38.95 ± 48.29	(140.00)	
Dyspnea			
Exp.	2.55 ± 5.19	(22.00)	1.97*
Cont.	0.85 ± 3.57	(16.00)	
Wheeze			
Exp.	2.23 ± 4.87	(17.00)	1.1
Cont.	3.40 ± 11.48	(50.00)	
Sputum			
Exp.	20.81 ± 39.09	(180.00)	0.56
Cont.	16.35 ± 28.27	(99.00)	
Sleep disturbance			
Exp.	1.86 ± 7.43	(35.00)	1.01
Cont.	1.15 ± 4.69	(21.00)	
Asthma attack			
Exp.	0.32 ± 1.49	(7.00)	0.1
Cont.	0.95 ± 4.25	(19.00)	

*$p<.05$, Exp., Experimental group; Cont., Control group.

amount of the major antigen, i.e., *Der fl* and *Der pI*, was determined according to ELISA. The total amount of antigen, derived by adding the amount of *Der fl* and *Der pI*, was used to calculate the antigen amount per g in the unit of ng/g. The symptoms used to indicate asthma included 6 symptoms frequently seen in asthma patients, i.e., cough, dyspnea, wheezing, sputum, sleep disturbance and asthmatic attacks. The subjects were asked the level of asthma symptoms for the 7 days starting on day 1 of study treatment and for the last 7 days of the study. The sum of the frequency of symptoms that occurred over each 7-day period was calculated. PEFR was measured using a peak flow meter over the same two 7-day periods. The subjects recorded their own measurement thrice each morning and thrice again 12 hours later; the largest value obtained was used in each case. PEFR is the percentile obtained by dividing the average obtained each morning and night for 7 days by the expected value based on the Morris normset (Table 6)

Table 5. Homogeneity Test for PEFR between Experimental and Control Groups (%)

Group	Before		Z[b]
	Mean ± SD	(Range)	
Morning PEFR			
Exp.	86.45 ± 14.89	(65.00)	1.525
Cont.	92.45 ± 13.92	(58.00)	
Evening PEFR			
Exp.	88.09 ± 13.88	(55.00)	1.701
Cont.	93.50 ± 12.42	(57.00)	

Exp., Experimental group; Cont., Control group.

Table 6. B. Morris Normset

	Gender	Age	Formula
PEF (lps)	Female	< 21	.1244H+.157A-3.916
	Female	21 - 99	.1244H-.025A-.7350
	Male	< 26	.1980H+.166A-8.060
	Male	26 - 99	.2387H-.035A-.5993

PEF (lps), Peak Expiratory Flow; H, Height in inches; A, Age in years.

Data analysis

For data analyses, Wilcoxon rank sum test, Wilcoxon signed rank test and χ^2-test were adopted using the SPSS program.

RESULTS

The amount of HDM allergens

The total amount of HDM allergens

After bedding control, the total amount of HDM allergens of bedding and bedroom floor reduced from 20098.6 ng/g to 14385.1 ng/g (p=0.009). There was no significant difference in the decrease of the amount of HDM allergens between the two groups (p=0.497) (Table 7).

Table 7. Amount of House Dust Mite Allergens Between Experimental and Control Groups before and after Bedding Control (ng/g of dust)

Group	Before		After		Z^w	Difference (After-Before)		Z^b
	Mean ± SD	(Range)	Mean ± SD	(Range)		Mean ± SD	(Range)	
Der fl								
Exp.	19877.7 ± 14726.4	(55820)	14054.6 ± 9949.6	(33410)	2.61†	-5823.1 ± 11902.7	(63550)	0.81
Cont.	18314.1 ± 17358.8	(70590)	16394.5 ± 19432.4	(90610)	1.61	-1919.6 ± 25222.5	(134760)	
Der pl								
Exp.	220.8 ± 318.5	(1273)	330.5 ± 627.8	(2741)	1.93	109.7 ± 375.5	(1900)	2.34*
Cont.	1687.4 ± 4741.1	(18760)	1484.9 ± 4599.6	(19380)	0.64	-202.5 ± 723.7	(3676)	
Total								
Exp.	20098.6 ± 14937.0	(56062)	14385.1 ± 10317.3	(33487)	2.61†	-5713.5 ± 11893.3	(63753)	0.68
Cont.	20001.5 ± 18234.5	(70745)	17879.4 ± 19669.6	(90760)	1.61	-2122.1 ± 25423.1	(135336)	

*p<.05, †p<.01.
Exp., Experimental group (n=22); Cont., Control group (n=20); Z^w, Wilcoxon Signed Rank test within group; Z^b, Wilcoxon Rank Sum test between groups.

The amount of Der fl

After bedding control, the amount of *Der fl* reduced significantly from 19877.7 ng/g to 14054.6 ng/g (p=0.009). There was no significant difference in the reduction of the amount of *Der fl* between the two groups (p=0.420) (Table 7).

The amount of Der pI

After bedding control, the amount of *Der pI* increased from 220.8 ng/g to 330.5 ng/g (p=0.053). There was significant difference in the change of the amount of *Der pI* between the two groups (p=0.019) (Table 7).

Asthma symptoms

After bedding control, the frequency of cough reduced from 41.14 to 22.27 (p=0.386) in the experimental group, that of dyspnea from 2.55 to 1.18 (p=0.122), that of wheezing from 2.23 to 0.27 (p=0.041), that of sputum from 20.81 to 10.63 (p=0.009), and that of asthma attack from 0.32 to 0.14 (p=0.655). Meanwhile, the frequency of sleep disturbance increased from 1.86 to 3.09 (p=0.394). There was significant difference in the decrease of the frequency of dyspnea (p=0.002) and in the increase of sleep disturbance (p=0.022) between the two groups after bedding control (Table 8).

PEFR (peak expiratory flow rate)

After bedding control, PEFR in the morning increased from 86.45% to 88.60% (p=0.200), but there was no significant difference in this increase of PEFR in the morning between the two groups (p=0.101). PEFR in the evening increased from 88.09% to 90.27% (p=0.211), but again there was no significant difference in this increase between the two groups (p=0.075) (Table 9).

DISCUSSION

The methods of controlling environment to reduce the number of HDMs include chemical and physical methods. Chemical methods include the use of benzile benzoate, liquid nitrogen, and tannic acid. These methods are effective in decreasing the number of HDMs and asthma symptoms[13] but their effectiveness is not permanent,[14] poisonous substances can remain in the bedding for a long time, and the chemicals are expensive, so they can not be used extensively.[15,16]

The physical methods of controlling environment are achieved through lowering humidity, which is the most important factor in the growth and proliferation of HDMs. Studies have reported

Table 8. Frequency of Asthmatic Symptoms between Experimental and Control Groups before and after Bedding Control (freq/week)

Group	Before		After		Z^w	Difference (After-Before)		Z^b
	Mean ± SD	(Range)	Mean ± SD	(Range)		Mean ± SD	(Range)	
Cough								
Exp.	41.14 ± 81.68	(290.00)	22.27 ± 50.05	(220.00)	1.50	-18.86 ± 59.85	(293.00)	0.48
Cont.	38.95 ± 48.29	(140.00)	36.85 ± 63.44	(260.00)	0.87	-2.10 ± 35.85	(190.00)	
Dyspnea								
Exp.	2.55 ± 5.19	(22.00)	1.18 ± 2.79	(10.00)	1.55	-1.36 ± 5.17	(30.00)	3.09**
Cont.	0.85 ± 3.57	(16.00)	2.20 ± 4.69	(18.00)	2.07*	1.35 ± 2.78	(10.00)	
Wheeze								
Exp.	2.23 ± 4.87	(17.00)	0.27 ± 1.08	(5.00)	2.05*	-1.95 ± 4.81	(18.00)	1.18
Cont.	3.40 ± 11.48	(50.00)	2.00 ± 6.70	(30.00)	0.95	-1.40 ± 5.02	(22.00)	
Sputum								
Exp.	20.81 ± 39.09	(180.00)	10.63 ± 24.94	(110.00)	2.62**	-10.18 ± 19.33	(75.00)	1.22
Cont.	16.35 ± 28.27	(99.00)	14.65 ± 26.95	(95.00)	0.56	-1.70 ± 20.54	(114.00)	
Sleep disturbance								
Exp.	1.86 ± 7.43	(35.00)	3.09 ± 14.28	(67.00)	0.85	1.23 ± 16.46	(102.00)	2.29*
Cont.	1.15 ± 4.69	(21.00)	2.05 ± 6.49	(29.00)	2.04*	0.90 ± 1.97	(8.00)	
Asthma attack								
Exp.	0.32 ± 1.49	(7.00)	0.14 ± 0.47	(2.00)	0.45	-0.18 ± 1.37	(8.00)	0.00
Cont.	0.95 ± 4.25	(19.00)	0.75 ± 3.13	(14.00)	0.45	-0.20 ± 1.15	(6.00)	

Exp., Experimental group; Cont., Control group.

Table 9. PEFR between Experimental and Control Groups before and after Bedding Control (%)

Group	Before		After		Z^w	Difference (After-Before)		Z^b
	Mean ± SD	(Range)	Mean ± SD	(Range)		Mean ± SD	(Range)	
Morning PEFR								
Exp.	86.45 ± 14.89	(65.00)	88.60 ± 13.66	(58.40)	1.28	2.15 ± 7.12	(32.30)	1.64
Cont.	92.45 ± 13.92	(58.00)	89.43 ± 17.33	(63.00)	1.03	-3.02 ± 8.57	(31.70)	
Evening PEFR								
Exp.	88.09 ± 13.88	(55.00)	90.27 ± 13.46	(65.00)	1.25	2.18 ± 7.51	(37.00)	1.78
Cont.	93.50 ± 12.42	(57.00)	91.10 ± 17.28	(60.00)	1.21	-2.40 ± 10.69	(50.00)	

Exp., Experimental group; Cont., Control group.

that the amount of HDM antigen is decreased th rough the use of air-conditioning and dehumidifiers in summer.[15] The proliferation of HDMs is prevented by avoiding the temperature and humidity appropriate for HDM growth.

The second method is the use of a vacuum cleaner which is effective at removing dead HDM bodies and feces but not at decreasing the number of live HDMs.[5,17] In addition, some studies have reported that the amounts of dust and HDM

antigen are higher in bedrooms cleaned often.[18]

The third method is the wrapping of bedding with special encasings coated with polyurethane to prevent the transfer of HDMs. Although many studies[19-21] abroad have reported the effectiveness of this method, Kang[22] reported that while this method decreased the amount of dust it was not effective in reducing the amount of HDM antigen and asthmatic symptoms.

The fourth method includes washing bedding in water hotter than 55℃ every 2 weeks, based on the 3 - 4 weeks life cycle of HDMs and their weak resistance against heat. This method is effective in removing HDMs and group I antigen but is not effective against heat-resistant group II antigen. The protein denaturation of group II HDM antigen, occurs only at temperatures higher than 100℃, so this HDM is difficult to remove with hot water in an ordinary household.[23]

According, to the results of present study, the total amount of HDM antigen within the bedroom decreased from 20098.6 ng/g before treatment to 14385.1 ng/g after treatment, showing a decrease of 28.4%. This reduction was lower than that of 83.6% by controlling 4 different environmental factors in the bedroom in the study by Choi (1996).[24] This lower decrease in our study compared with that of previous study was probably due to differences in the treatment methods.

According to the present study, more HDM antigen was present in bedding than in the bedroom floor, supporting the results of previous study.[25] Our result in which the experimental group showed a decrease in the amount of antigen in both bedding and the bedroom floor agreed with the result of previous study[7] reporting a correlation between the amounts of antigen in bedding and bedroom floor. This result confirms that managing bedding, the main habitat of HDM, will also control the amount of HDM antigen in the bedroom floor.

The amount of total HDM antigen in the present study decreased in both groups, probably due to the Hawthorne effect in which home visits by the researcher caused the control subjects to have more interest in controlling their environment, so they would wash or boil their bedding cover more frequently. On the other hand, the amount of *Der pI* increased significantly in the experimental group but decreased in the control group. This result was probably due to the increase in *Der pI* more sensitive to humidity in the experimental group where bedding was washed every 2 weeks. In contrast, the amount of *Der fI* decreased significantly in the study group. Thus, longer in-depth studies should be repeated in the future with more subjects in order to understand this result.

The results of the present study showed that the pre-study proportion, 71.4%, of the subject households showing a total of amount HDM antigen more than the threshold level for inducing asthma symptoms, i.e., 10,000 ng/g, was reduced to 61.9% after the study.

Despite the fact that the present study was done during wintertime when the environment was adverse for the growth and proliferation of HDMs with the average in-door humidity of 44.8%, this result indicated that various environmental factors were contributing to the survival of HDM; namely the 52.4% of subjects residing in apartments, 73.8% using beds, and 35.7% having pet dogs. In other words, the optimum HDM growth environment could be provided in apartments where the temperature and humidity are maintained at constant levels due to central heating and exclusion of outside air, so that the growth environment for HDM could be maintained. Using beds would increase the possibility for continuous accumulation of HDM antigen since washing and drying of mattresses are impossible. Animal dander from pets could provide HDMs with a good feeding source, leading to their proliferation. Thus, various environmental factors within a bedroom should be considered in the management of HDM. Furthermore, active management for HDMs in bedrooms is urgent for asthmatic patients.

Asthmatic symptoms including cough, dyspnea, wheezing, sputum, and asthmatic attacks decreased in the experimental group after the study compared with before the study. Especially, the incidences of wheezing and sputum decreased significantly. The incidences of cough, wheezing, sputum and asthmatic attacks also decreased in the control group, but the incidences of dyspnea and sleep disturbance increased significantly after the study compared with before the study, pro-

bably due to the slight decrease in the level of HDM antigen after the study compared with the level before the study that was higher than the asthma symptom induced threshold level, i.e., 10,000 ng/g. The incidence of dyspnea decreased in the experimental group after the study compared with before the study, probably due to the larger decrease in the antigen amount in the experimental group. The control group also showed an improvement in some asthmatic symptoms. As mentioned earlier, this improvement was probably due to the Hawthorne effect; thereby indicating that asthmatic symptoms could be improved through supportive nursing such as home visits by promoting the subjects to perform activities to suppress HDMs.

It is difficult to account for the increased incidence of sleep disturbance in the experimental group. According to previous studies, changes in asthmatic symptoms according to treatment were expressed based on summative subjective scores, unlike the present study where each symptom was measured according to its incidence. This difference poses a limitation on comparing the results of the present study to those of previous studies. Thus, in order to objectively examine changes in asthmatic symptoms according to different treatments, it would be desirable for the subjects to record the incidence of each symptom every day.

PEFR measured in the morning and at night in the present study showed an increase in the experimental group but a decrease in the control group after the study, partially supporting the result of the study by Van der Heide, et al.[26] who reported that even a slight change in the degree of exposure to HDM antigen had some affect on the airway responses. However, the finding of no significant increase in PEFR was similar to previous studies. For example, no difference was present in PEFR according to a study that followed 20 adult asthmatic patients for 6 months after using cotton bedding prepared with special encasings coated with polyurethane,[22] and no difference was present in PEFR 3 and 6 months after different treatments in a study that used other preventive measures by dividing 45 asthmatic patients into 4 groups.[27] In a study done on 70 asthmatic children a 100% decrease was seen in the amount of HDM antigen 6 weeks after spraying mattresses with HDM acaricides and covering the mattresses and bedding with special encasings, and the lung functions improved over 24 weeks.[28] According to the results of these studies, the improvement in PEFR according to the decrease in the amount of HDM antigen might not become apparent within a short period of time, and long-term use of various methods would be more effective than the short-term use of only a single method of preventing HDM.

Overall, the present study suffers from the following three limitations: the method of managing bedding was applied only for a short period of time, and was limited to wintertime, and only a few subjects were included. However, the economical, simple and non-toxic method used in the present study decreased the amount of HDM antigen, thereby partially improving asthmatic symptoms and PEFR. Thus, additional future studies are needed, with a larger number of subjects and longer follow-up periods.

Training patients and their families regarding methods to control the environment and the importance of managing the environment is important. Furthermore, they should be provided with effective methods that are simple to be applied for long periods of time.

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