Environmental Tobacco Smoke and Adult-Onset Asthma: A Population-Based Incident Case–Control Study

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Objectives. The authors assessed the effects of environmental tobacco smoke (ETS) on the development of asthma in adults.

Methods. In the Pirkanmaa district of South Finland, all 21- to 63-year-old adults with new cases of asthma diagnosed during a 2.5-year period (n=521 case patients, out of 441 000 inhabitants) and a random sample of control subjects from the source population (932 control subjects) participated in a population-based incident case–control study.

Results. Risk of asthma was related to workplace ETS exposure (adjusted odds ratio [OR]=2.16; 95% confidence interval [CI]=1.26, 3.72) and home exposure (OR=4.77; 95% CI=1.29, 17.7) in the past year. Cumulative ETS exposure over a lifetime at work and at home increased the risk.

Conclusions. This study indicates for the first time that both cumulative lifetime and recent ETS exposures increase the risk of adult-onset asthma. (*Am J Public Health.* 2003;93:2055–2060)

Environmental tobacco smoke (ETS) contains over 4000 compounds, including several carcinogens, irritants, and toxic agents.[1,2] There is convincing evidence of parental smoking causing asthma in children,[3,4] but the evidence on ETS exposure and development of adult asthma is limited.[4,5] This question is of major public health relevance, as the occurrence of both ETS exposure and asthma is relatively common in working-age populations.

Only 6 studies have addressed the question of ETS and asthma in adulthood,[6–11] indicating an increased risk of asthma in relation to ETS exposure at home,[7,10,11] at work,[6,9] or both.[8] Most of these studies were vulnerable to selection and information bias because of cross-sectional designs or asthma diagnosis being based on self-report. In addition, Robbins and colleagues[12] reported a study on ETS and airway obstructive disease, including both asthma cases and cases of chronic obstructive pulmonary disease, that was based on the same study population of Californian Seventh Day Adventists as the study by Greer et al. that focused on asthma.[6] Stronger evidence of the causal role of ETS exposure in asthma among the working population would support more strict preventive measures and health policies in workplaces.

We conducted a large population-based study in South Finland to assess the effect of ETS exposure in the workplace and at home on the development of asthma in the working-age population. We recruited all adults with new cases of asthma in a population of approximately 441 000 people over 2.5 years.

METHODS

Study Design

This study was a population-based case–control study of incident asthma. The source population consisted of adults 21 to 63 years of age living in the Pirkanmaa Hospital District. This district is a geographically defined administrative area in South Finland with a population of 440 913 inhabitants (in 1997). Our goal was to recruit all individuals in the source population with new asthma diagnoses during the study. Control subjects were selected randomly from the source population based on the 1997 census.

Definition and Selection of Case Patients

We systematically recruited all individuals with new cases of asthma, first in the city of Tampere, beginning on September 15, 1997, and then from March 10, 1998, to March 31, 2000, in the whole Pirkanmaa Hospital District. Patients were recruited at all health care facilities diagnosing asthma, including the Department of Pulmonary Medicine at the Tampere University Hospital and offices of the privately practicing pulmonary physicians in the region, as well as public health care centers. To ensure the participation of those asthma patients not caught by our screening system, the National Social Insurance Institution of Finland carried out a computerized search of all patients who had received reimbursement for asthma medications for the first time during the period from September 1, 1997, through May 1, 1999, and who had not yet been recruited.

The diagnostic criteria for asthma included a history of at least one asthma-like symptom (prolonged cough, wheezing, attacks of or exercise-induced dyspnea, or nocturnal cough or wheezing) and demonstration of reversible airway obstruction in lung function investigations.[13,14] The health care facilities were responsible for reporting the cases to us. Our research nurse was based at the Tampere University Hospital, and the cases were reported to her immediately when they were identified. At the Tampere University Hospital, patients were recruited at their first visit resulting from suspected asthma, and the diagnosis was verified in clinical examinations. At the other health care facilities, patients were recruited immediately when the asthma diagnosis was verified.

The National Social Insurance Institution contacted patients 6 months to 2 years after their diagnosis was established. For these patients, the date and criteria of the asthma diagnosis were confirmed from their medical records so that the diagnoses of asthma patients included in our study fulfilled our criteria. Eligible subjects were invited to participate in the study, and their informed consent was requested by their physician or through a letter sent by the National Social Insurance Institution. The medical records of all patients were checked, and only those with no previous history of diagnosed asthma or long-term use of asthma medication were included in the study.

A total of 362 patients (response rate 90%) participated through the health care system, and 159 patients participated through the National Social Insurance Institution (response rate 78%), totaling 521 patients. After current and previous smokers had been excluded, the study population comprised 239 asthma patients. We compared the patients identified through different sources, health care facilities, and the National Social Insurance Institution according to the characteristics listed in Table 1 and according to the exposure distributions in Table 2. There were no major differences between the patients identified through the various sources.

Selection of Control Subjects

The control subjects were randomly drawn from the source population via the national population registry, which has a full coverage of the population. The general eligibility criteria (21–63 years, resident of the Pirkanmaa Hospital District) were applied for control subjects, who were recruited by means of invitation letters sent at regular intervals throughout the study period. After up to 3 invitation letters and telephone calls, 1016 control subjects participated in the study (the response rate was 80% among those who had a telephone number in the Pirkanmaa area). After exclusion of 76 persons who reported previous or current asthma, 6 persons older than 63 years, and 2 persons returning incomplete questionnaires, the number of control subjects was 932. Current and previous smokers were excluded, and the final study population included 487 control subjects.

Exposure Assessment

Exposure assessment was based on questionnaire information on exposure to ETS during the past 12 months and a time-exposure matrix for lifetime exposure both at home and at work. For each age period (0–1, 1–6, 7–10, 11–15, 16–20, 21–30, 31–40, and ≥ 41 years), individuals were asked about number of cigarettes per day and duration of exposure during the period. In the case of individuals exposed to cigar or pipe smoking, values were converted, with 1 cigar or pipeful of tobacco corresponding to 1 cigarette.

TABLE 1—Characteristics of the Study Population: South Finland, 1997–2000

	Patients, No. (%)	Control subjects, No. (%)
Gender		
Men	56 (23.4)	171 (35.1)
Women	183 (76.6)	316 (64.9)
Age, y		
21–29	54 (22.6)	75 (15.4)
30–39	44 (18.4)	125 (25.7)
40–49	55 (23.0)	116 (23.8)
50–59	66 (27.6)	133 (27.3)
60–63	20 (8.4)	38 (7.8)
Parental allergic diseases		
Maternal atopy	43 (18.0)	55 (11.3)
Paternal atopy	24 (10.4)	27 (5.5)
Maternal asthma	30 (12.6)	29 (6.0)
Paternal asthma	35 (14.6)	20 (4.1)
Any	56 (23.4)	71 (14.6)
Education[a]		
No vocational schooling	41 (17.2)	74 (15.3)
Vocational course	40 (16.8)	43 (8.9)
Vocational institution	64 (26.9)	124 (25.6)
College-level education[b]	57 (24.0)	142 (29.3)
University or equivalent education[c]	36 (15.1)	102 (21.0)
Pets at home		
Sometimes	149 (62.3)	318 (65.3)
In the past 12 months	77 (32.2)	201 (41.3)
Over 12 months ago	146 (61.1)	306 (62.8)
Visible mold or mold odor in the home	32 (13.4)	57 (11.7)
Visible mold or mold odor in the workplace	34 (14.2)	56 (11.5)
Occupational exposures	139 (58.2)	316 (64.9)
Total	239 (100)	487 (100)

[a]Information on education was missing for 3 subjects (1 patient and 2 control subjects).
[b]Education resulting in a lower professional degree.
[c]Education resulting in a higher professional degree.

Measurement Methods

Questionnaire. The self-administered questionnaire, modified from the Helsinki Office Environment Study questionnaire for use in general populations,[15,16] included 6 sections: personal characteristics, health information, active smoking and ETS exposure, occupation and work environment, home environment, and dietary questions.

Lung function measurements. The same diagnostic protocol was applied for all patients with suspected asthma.[13,14] The only exception was patients recruited through the National Social Insurance Institute, for whom data were obtained through abstraction from medical records.

Baseline spirometry and bronchodilation tests were recorded with a pneumotachograph spirometer connected to a computer and a disposable flow transducer (Medikro 905, Medikro, Kuopio, Finland), according to the standards of the American Thoracic Society.[17] Presence of obstruction was judged via reference values derived from a Finnish population.[18]

The criteria for significant improvement in regard to the bronchodilation test were as follows: a 15% or more improvement in forced expiratory volume in 1 second, a 15% or more improvement in forced vital capacity, and a 23% or more improvement in peak expiratory flow (PEF). All patients underwent PEF follow-up for at least 2 weeks, with a mini-Wright meter performing measurements twice a day. During the second week, measurements were performed before and 15 minutes after patients had taken short-acting bronchodilating medication. Criteria for significant PEF variability included 20% or more daily variations or 15% or more improvement in response to bronchodilating medication during at least 2 days. Those for whom there was a strong suspicion of asthma underwent a 2-week oral steroid treatment if the other diagnostic tests were negative. Patients underwent 2 weeks of PEF follow-up during this treatment, along with a spirometric measurement at the end of the treatment period to judge responses.

Statistical Methods

We used exposure odds ratios (ORs) to quantify the relationship between exposure to ETS and adult-onset asthma. We estimated odds ratios in logistic regression analyses adjusting for gender, age, parental atopy or asthma, education (as an indicator of socioeconomic status), visible mold or mold odor at home or in the workplace, any history of

TABLE 2—Exposure to Environmental Tobacco Smoke (ETS)

	Patients, No. (%)	Control subjects, No. (%)
Exposure to ETS during past 12 months (cigarettes per day)		
Home and workplace combined[a]		
No	196 (83.8)	436 (89.9)
Yes	38 (16.2)	49 (10.1)
1–9 cigarettes per day	17 (7.3)	22 (5.6)
≥10 cigarettes per day	14 (6.0)	17 (3.5)
Workplace[b]		
No	184 (84.4)	415 (91.0)
Yes	34 (15.6)	41 (9.0)
1–9 cigarettes per day	15 (6.9)	19 (4.2)
≥10 cigarettes per day	12 (5.5)	12 (2.6)
Home[c]		
No	224 (97.0)	475 (98.3)
Yes	7 (3.0)	8 (1.7)
1–9 cigarettes per day	4 (1.7)	3 (0.7)
≥10 cigarettes per day	2 (0.9)	5 (1.0)
Cumulative lifetime ETS exposure (cigarette-years)		
Home and workplace combined		
0	104 (43.5)	231 (47.4)
1–49	26 (10.9)	91 (18.7)
50–99	22 (9.2)	44 (9.0)
100–149	19 (8.0)	25 (5.1)
≥150	68 (28.5)	96 (19.7)
Workplace		
0	163 (68.2)	355 (72.9)
1–49	32 (13.4)	70 (14.4)
50–99	15 (6.3)	17 (3.5)
100–149	7 (2.9)	18 (3.7)
≥150	22 (9.2)	27 (5.5)
Home		
0	140 (58.6)	293 (60.2)
1–49	24 (10.0)	66 (13.6)
50–99	13 (5.4)	38 (7.8)
100–149	12 (5.0)	21 (4.3)
≥150	50 (20.9)	69 (14.2)
Total	239 (100)	487 (100)

[a]Both work and home ETS exposure missing for 5 patients and 2 control subjects; daily smoking rate missing for 7 patients and 10 control subjects.
[b]Work exposure missing for 21 patients and 31 control subjects; smoking rate missing for 7 patients and 10 control subjects.
[c]Home exposure missing for 8 patients and 4 control subjects; smoking rate missing for 1 patient.

pets in the home, and self-reported occupational exposure to sensitizers, dusts, or fumes (apart from ETS).

We studied the effects of exposure during the past 12 months and cumulative lifetime exposure, focusing on home and work exposures separately and combined. We calculated cumulative exposure by multiplying, for each time period, daily rate of cigarette exposure by duration in years and then summing these values, yielding exposure parameters in cigarette-years. Because ETS exposure during the past 12 months and cumulative ETS exposure may be related, we adjusted for each exposure parameter when assessing the other. We elaborated exposure–response relations by fitting both ordinal scale categorical and continuous exposure variables.

We quantified the effect of exposure as an attributable fraction[19] or etiologic fraction[20] providing the proportion of exposed case patients for whom the disease was attributable to the exposure.[19] We calculated the attributable fraction (AF) as follows:

$$1) \qquad AF=(OR-1)/OR,$$

where OR is the adjusted odds ratio for the exposure of interest, an unbiased estimate of an incidence ratio in a population-based case–control study.[20] We calculated 95% confidence intervals (CIs) using the corresponding odds ratio interval. Finally, we calculated the attributable fraction for the whole population (AF_T)—that is, the fraction of all asthma cases among the working-age population caused by ETS exposure—using the proportion of exposed cases (P_E), as follows[20]:

$$2) \qquad AF_T=P_E\,(OR-1)/OR=P_E\,(AF).$$

RESULTS

Characteristics and Exposures of Patients and Control Subjects

Larger proportions of patients than control subjects were women, were young, reported parental atopy, were at lower education levels, and reported visible mold or mold odor at work or at home (Table 1). Table 2 presents the distributions of ETS exposure in the workplace and at home during the previous 12 months as well as cumulative lifetime ETS exposure. Higher percentages of patients than control subjects reported ETS exposure in the workplace (15.6% vs 9.0%) and at home (3.0% vs 1.7%) during the past year.

TABLE 3—Crude and Adjusted Odds Ratios (ORs) of Asthma in Relation to Environmental Tobacco Smoke Exposure During the Previous 12 Months

Exposure	Crude OR	Adjusted OR[a] (95% Confidence Interval)	Adjusted OR[b] (95% Confidence Interval)
Home and workplace combined			
No	1.00	1.00	1.00
Yes	1.73	1.97 (1.19, 3.25)	1.66 (0.99, 2.78)
1–9 cigarettes per day	1.72	2.13 (1.05, 4.30)	1.88 (0.92, 3.86)
≥10 cigarettes per day	1.83	2.14 (0.95, 4.82)	1.56 (0.67, 3.61)
Quantity missing	1.56	1.45 (0.52, 4.01)	1.38 (0.49, 3.88)
Workplace			
No	1.00	1.00	1.00
Yes	1.87	2.16 (1.26, 3.72)	1.83) (1.05, 3.21)
1–9 cigarettes per day	1.78	2.06 (0.97, 4.36)	1.85 (0.89, 3.98)
≥10 cigarettes per day	2.26	2.90 (1.14, 7.34)	2.10 (0.81, 5.47)
Quantity missing	1.56	1.62 (0.57, 4.58)	1.62 (0.56, 4.67)
Home			
No	1.00	1.00	1.00
Yes	1.86	4.77 (1.29, 17.7)	3.83 (0.99, 14.8)
1–9 cigarettes per day	2.83	3.93 (0.80, 19.4)	3.62 (0.71, 18.6)
≥10 cigarettes per day	0.85	0.75 (0.13, 4.29)	0.56 (0.10, 3.30)
Quantity missing	2.10	2.74 (0.15, 48.8)	1.85 (0.10, 33.1)

[a]Adjusted for gender, age, parental atopy/asthma, education, pets at home, visible mold or mold odor in the home or in the workplace, self-reported occupational exposures, and environmental tobacco smoke in the other environment (workplace or home).
[b]Adjusted for gender, age, parental atopy/asthma, education, pets at home, visible mold or mold odor in the home or in the workplace, self-reported occupational exposures, and cumulative environmental tobacco smoke exposure.

ETS Exposure and Risk of Asthma

Risk of asthma was significantly related to total ETS exposure (OR=1.97; 95% CI=1.19, 3.25) and to workplace ETS exposure (OR=2.16; 95% CI=1.26, 3.72) during the previous 12 months (Table 3). Use of continuous exposure variables revealed statistically significant exposure–response relations for total ETS exposure, with an odds ratio of 1.33 per 10 cigarettes per day (95% CI=1.02, 1.75), and for workplace ETS exposure, with an odds ratio of 1.44 per 10 cigarettes per day at work (95% CI=1.03, 2.01). Adjustment for cumulative lifetime exposure slightly lowered the risk estimates. The risk of asthma also increased in relation to home exposure (OR=4.77), but the confidence interval was wide (95% CI=1.29, 17.7). The point estimate for the highest exposure category was lower than the estimate for the lower category. Small numbers of subjects exposed at home limited inferences regarding home exposures.

Table 4 presents data on asthma risk according to cumulative lifetime ETS exposure. Although some indication of an exposure–response relation was observed, especially for total cumulative exposure and home exposure, the continuous exposure variables revealed no statistically significant exposure–response relations. Cumulative work exposure appeared to have a stronger effect than home exposure. The risk estimates were slightly lower when we adjusted for exposure during the past 12 months.

Risk of Asthma Attributable to ETS Exposure

The fraction of asthma attributable to total ETS exposure during the past year was 49.2% (95% CI=16.0, 69.2) among individuals exposed to ETS. The attributable fraction for the whole working-age population was 8.0%.

DISCUSSION

The present study included the largest number of asthma cases among the studies (of which we are aware) that have addressed relations between ETS exposure and adult asthma. Our incident case–control study design was very efficient relative to a cohort study, which would yield a similar amount of information. With an asthma incidence rate of 1 case per 1000 person-years, our study corresponds to a follow-up of approximately 100 000 adults for 5 years.

We found that both workplace and total ETS exposures during the past 12 months were significantly related to an increased risk of new asthma in the working-age population. A significant exposure–response pattern was observed in average number of cigarettes individuals were exposed to daily. We also observed an increase in the risk related to home ETS exposure, but the estimate was less precise because of the small number of subjects exposed at home. An indication of a dose-dependent effect was observed with cumulative ETS exposure at home and with total cumulative exposure, although the trends were not statistically significant. The effect of cumulative workplace ETS exposure appeared to be greater than that of home exposure.

The fraction of asthma attributable to ETS exposure during the past year was quite high: 49% among those who were exposed to ETS, or 8% among the whole population. Biologically plausible mechanisms underlying the relation between ETS and asthma have been proposed,[4,5] including inflammatory responses of the airways to the irritant compounds of tobacco smoke and an increase in epithelial permeability to environmental allergens.

Validity Issues

We were able to include a high proportion of individuals in the study area with new cases of asthma because of our thorough recruitment strategy, which involved the health care system and the National Social Insurance Institution. The latter provides health care insurance to all citizens of Finland, and its medication files involve practically full coverage of asthma patients in Finland fulfilling our rather strict diagnostic criteria. The response rate among the control subjects was also relatively

TABLE 4—Crude and Adjusted Odds Ratios (ORs) of Asthma in Relation to Cumulative Lifetime Exposure to Environmental Tobacco Smoke

Exposure	Crude OR	Adjusted OR[a] (95% Confidence Interval)	Adjusted OR[b] (95% Confidence Interval)
Home and workplace combined (cigarette-years)			
0 (reference)	1.00	1.00	1.00
1–49	0.64	0.80 (0.48, 1.36)	0.79 (0.46, 1.34)
50–99	1.11	1.30 (0.71, 2.35)	1.28 (0.70, 2.34)
100–149	1.69	2.01 (1.02, 3.99)	1.76 (0.87, 3.55)
≥150	1.57	1.84 (1.21, 2.80)	1.71 (1.11, 2.64)
Workplace (cigarette-years)			
0 (reference)	1.00	1.00	1.00
1–49	1.00	1.17 (0.71, 1.93)	1.08 (0.65, 1.80)
50–99	1.92	2.35 (1.07, 5.14)	2.25 (1.03, 4.93)
100–149	0.85	1.28 (0.49, 3.31)	0.93 (0.34, 2.57)
≥150	1.78	2.21 (1.15, 4.27)	1.84 (0.93, 3.64)
Home (cigarette-years)			
0 (reference)	1.00	1.00	1.00
1–49	0.76	0.95 (0.55, 1.64)	0.99 (0.57, 1.71)
50–99	0.72	0.78 (0.39, 1.57)	0.81 (0.40, 1.62)
100–149	1.20	1.05 (0.48, 2.30)	1.09 (0.50, 2.40)
≥150	1.52	1.37 (0.87, 2.16)	1.40 (0.89, 2.20)

[a]Adjusted for gender, age, parental atopy/asthma, education, pets at home, visible mold or mold odor in the home or in the workplace, self-reported occupational exposures, and cumulative environmental tobacco smoke in the other environment (workplace or home).
[b]Adjusted for gender, age, parental atopy/asthma, education, pets at home, visible mold or mold odor in the home or in the workplace, self-reported occupational exposures, and environmental tobacco smoke exposure during the past 12 months.

high. Thus, our study was not likely to have involved any major selection bias.

One of the strengths of our study was the assessment of recent ETS exposure as well as cumulative lifetime exposure in both the home and work environments. The study was introduced to the participants as an investigation of environmental factors in general (the Finnish Environment and Asthma Study), with no special focus on ETS, to reduce information bias in participants' reports. Information on exposures was collected from patients and control subjects in a similar way.

Development of asthma is likely to take several months to several years after the relevant exposure, and consequently exposure assessment over a long time period is needed[21,22]; as a result, we concluded that a well-designed questionnaire was the best exposure assessment method for our aims. We included only incident cases of asthma in the study, which diminished concerns related to the potential effects of diagnosed asthma on ETS exposure. Asthma was defined on the basis of objective clinical findings to eliminate information bias concerning the outcome.

We were able to adjust for a number of potential confounders in logistic regression analyses to eliminate these factors as potential explanations for our results. As a means of controlling for genetic predisposition, we adjusted for parental atopy, but not for participants' own atopy, as it may be part of the causal pathway of effects of ETS.

Synthesis with Previous Knowledge

Our risk estimates for ETS and adult asthma were slightly higher than those of earlier studies,[6–11] although the confidence intervals include these previous estimates. There are at least 2 likely explanations for these results. First, we based diagnoses on clinical examinations, including lung function measurements; thus, our criteria were stricter than those of earlier studies that based diagnoses on self-reports.[6–8,10,11] Second, earlier studies with cross-sectional designs (including prevalent asthma cases) most likely underestimated risks because of the tendency of subjects with diagnosed asthma to avoid ETS exposure.[7,8,10] Some evidence of an exposure–response relationship between ETS exposure and risk of asthma was indicated in 3 earlier studies,[7,8,10] but our study was the first to investigate the effects of cumulative ETS exposure.

CONCLUSIONS

This large, population-based study provides evidence of the effect of ETS exposure on development of asthma in adulthood. Both workplace and home ETS exposures seem to be of importance. The adverse effect of cumulative workplace exposure seems to be strong, but from a preventive point of view it is also important that past-year ETS exposure has a great effect on people's risk of developing asthma. Elimination of or reductions in ETS exposure in adulthood could prevent occurrence of asthma considerably. Our results indicate that ETS is an important preventable cause of asthma in adulthood. ■

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Contributors

M.S. Jaakkola was the principal investigator for the study; obtained funding; supervised the study; participated in the conception and design of the study, acquisition of data, and analysis and interpretation of data; and wrote the article with the help of the other authors. R. Piipari participated in acquisition of data, interpretation of data, and critical revision of the article. N. Jaakkola participated in acquisition of data, analysis and interpretation of data, and critical revision of the article. J.J.K. Jaakkola participated in the conception and design of the study, acquisition of data, and analysis and interpretation of data, provided statistical expertise, and participated in writing the article.

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Human Participant Protection

The ethics committees of the Finnish Institute of Occupational Health and the Tampere University Hospital approved the study. Participants provided informed consent.

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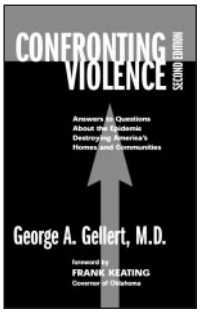

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Confronting Violence

George A. Gellert, MD

With a foreword by Frank Keating, Governor of Oklahoma

This book discusses interpersonal violence, including child and elder abuse, sexual assault, murder, suicide, stranger violence, and youth violence. It is written in a series of easy-to-reference questions and answers, and provides tips for avoiding high-risk situations. *Confronting Violence* includes lists of organizations and public agencies that provide help.

The 2nd Edition includes a new preface by APHA Executive Director Mohammad N. Akhter, MD, MPH, as well as new statistics and new references to recent events, such as the Columbine High School massacre and the child sex abuse scandal in the Catholic Church.

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