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Original Article

Exposure to Domestic Abuse and the Subsequent Development of Atopic Disease in Women

Katrina Nash, MBChB[a,b], Sonica Minhas, MBBS[c], Nicholas Metheny, PhD[d], Krishna M. Gokhale, MSc[c], Julie Taylor, PhD[e,f], Caroline Bradbury-Jones, PhD[e], Siddhartha Bandyopadhyay, PhD[g], Krishnarajah Nirantharakumar, MD[c], Nicola J. Adderley, PhD[c,*], and Joht Singh Chandan, PhD[c,*] *Oxford, Reading, and Birmingham United Kingdom; and Miami, Fla*

What is already known about this topic? Previous studies demonstrated that exposure to domestic violence and abuse is associated with a heightened risk for atopy, which could be explained by an increased allostatic load. These studies are limited by a cross-sectional design, small sample size, and recall bias.

What does this article add to our knowledge? We conducted a population-based retrospective cohort study using an anonymized UK primary care database. Our results indicated that exposure to domestic abuse is positively associated with atopic diseases: asthma, allergic rhinoconjunctivitis, and atopic eczema.

How does this study impact current management guidelines? Domestic violence and abuse are a major public health problem. Our results demonstrate a significant associated risk for developing atopic disease. Implementation of public health strategies for prevention and adapting a consideration of domestic violence and abuse in atopic assessments are necessary.

BACKGROUND: Exposure to domestic violence and abuse (DVA) is a global public health issue associated with substantial morbidity and mortality. There are few high-quality studies that assess the impact of DVA exposure on the development of atopic disease.

OBJECTIVE: To examine the association between exposure to DVA and the subsequent development of atopy.

METHODS: In this population-based, retrospective, open cohort study, we identified women with no history of atopic disease between January 1, 1995 and September 30, 2019 from IQVIA Medical Research Data, an anonymized UK primary care dataset. We used clinical codes to identify exposed patients (those with a code identifying exposure to DVA; n = 13,852) and unexposed patients (n = 49,036), who were matched by age and deprivation quintile. Cox proportional hazards regression was used to calculate hazard ratios (HRs) (with 95% CIs) of developing atopic disease: asthma, atopic eczema, or allergic rhinoconjunctivitis.

RESULTS: During the study period, 967 exposed women (incidence rate, 20.10/1,000 person-years) developed atopic disease, compared with 2,607 unexposed women (incidence rate, 13.24/1,000 person-years). This translated to an adjusted HR of 1.52 (95% CI, 1.41-1.64) accounting for key confounders; asthma (adjusted HR = 1.69; 95% CI, 1.44-1.99), atopic eczema (adjusted HR = 1.40; 95% CI, 1.26-1.56), and allergic rhinoconjunctivitis (adjusted HR = 1.63; 95% CI, 1.45-1.84).

CONCLUSIONS: Domestic violence and abuse is a significant global public health issue. These results demonstrate a significant associated risk for developing atopic disease. Public health approaches to the prevention and detection of DVA are necessary to reduce the associated ill health burden.

[a]Oxford University Clinical Academic Graduate School, University of Oxford, Oxford, United Kingdom
[b]Royal Berkshire Hospital, Reading, United Kingdom
[c]Institute of Applied Health Research, College of Medical and Dental Sciences, University of Birmingham, Birmingham, United Kingdom
[d]School of Nursing and Health Studies, University of Miami, Miami, Fla
[e]School of Nursing, College of Medical and Dental Sciences, University of Birmingham, Birmingham, United Kingdom
[f]Birmingham Women's and Children's Hospitals NHS Foundation Trust, Birmingham, United Kingdom
[g]Department of Economics, University of Birmingham, Birmingham, United Kingdom
*Joint senior authors.

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Corresponding author: Joht Singh Chandan PhD, Institute of Applied Health Research, College of Medical and Dental Sciences, University of Birmingham, Birmingham B152TT, United Kingdom. E-mail: j.s.chandan.1@bham.ac.uk.
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Abbreviation used
DVA- Domestic violence and abuse

Key words: *Domestic abuse; Hypersensitivity; Asthma; Atopic eczema; Allergic conjunctivitis; Retrospective studies; Cohort studies*

INTRODUCTION

Domestic violence and abuse (DVA) are a global public health problem thought to affect up to one in three women globally and one in four women in their lifetime in the United Kigdom.[1,2] Although DVA can affect anyone, it is a form of gender-based violence rooted in gender inequality, which disproportionately affects women. Statistics from the Crime Survey for England and Wales suggest that women aged greater than 16 years are twice as likely to have experienced DVA compared with men.[3] Domestic violence and abuse are associated with substantial morbidity and mortality as a result of associated physical injury, psychological consequences, and noncommunicable disease, which has been described in several studies.[4-10]

Previously published literature indicated that individuals exposed to DVA may experience changes in neuroimmunoregulation owing to the heightened allostatic load and physiologic stress response to trauma, resulting in increased levels of proinflammatory cytokines and neuropeptides.[11,12] In particular, some evidence suggests that victims exposed to stress exhibit a T_H1 to T_H2 cell shift that increases levels of IgE.[11,12] Consequently, individuals exposed to DVA may be more likely produce IgE antibodies in response to common environmental allergens, which can result in the development of one or more diseases of the atopic triad: atopic eczema, asthma, and allergic rhinoconjunctivitis.[13]

Several cross-sectional studies identified a positive association between DVA and asthma in adults.[14-17] However, these studies were limited because of potential recall bias, a small sample size, a lack of temporal data, or generalizability to the United Kingdom. To our knowledge, no cohort studies have been conducted that have assessed this relationship.[18] Furthermore, although research indicates a mechanism for the predisposition of atopy in DVA survivors,[11,12] no studies have explored the association between DVA and specifically atopic eczema and/or allergic rhinoconjunctivitis.

Further understanding of this association and awareness of the health impacts of DVA in women will enable evidence-based implementation of public health policies to prevent the subsequent development of atopic diseases in those who have been exposed to DVA. Therefore, we aimed to undertake a retrospective open cohort study to explore the risk for developing atopy in adult women (those aged 18 years and older) with a general practitioner (GP)-recorded exposure to DVA compared with those without a recorded exposure, using the IQVIA Medical Research Database (IMRD). The IMRD is an anonymized UK primary care database that is generalizable to the UK population.[19] Data extraction was facilitated using DExtER (University of Birmingham, UK).[20]

METHODS

This was a population-based, retrospective, open cohort study using IMRD, a large UK primary care database that is nationally representative of the demographic structure and common comorbidities in the United Kingdom.[19] The study period was January 1, 1995 to September 30, 2019. During that period, IMRD had 808 participating general practices that were using the Vision electronic medical records software system, providing a total of approximately 16 million anonymized longitudinal electronic patient records. Read codes are a hierarchical clinical coding system used to record symptoms, examinations, and diagnoses within IMRD.[21] Clinical information is grouped at the first coding level (eg, 14XD refers to a history of domestic abuse) and then additional detail is added at subsequent coding levels (eg, 14XD000 refers to a history of domestic emotional abuse).

To ensure that included practices were accurately recording information in their electronic system and that outcomes were not underreported, the eligibility date for general practices was defined as the later of 1 year after the following two dates: the date when the practice electronic medical records system was installed, and the date the practice attained acceptable mortality recording.[22] Comparisons of the practice's standardized mortality ratio with the national death rates are used to determine acceptable mortality recording dates, which have been agreed upon by multiple blinded reviewers to avoid biases associated with immortal periods when using IMRD-UK.[22] To ensure that patient records were complete at study entry, individual patients were eligible for inclusion from the later of the following dates: the date that the practice became eligible for inclusion, and 1 year after registration with the practice. Patients were excluded from the eligible cohort if they had a preexisting diagnosis of atopic disease (asthma, atopic eczema, and allergic rhinoconjunctivitis).

In this study, we used read codes (see Table E1 in this article's Online Repository at www.jaci-inpractice.org) to identify the exposed and unexposed cohort, as well as provide data for study covariates and outcomes of interest. Because this was an open cohort study, women were eligible to enter (index date) and leave (exit date) the study at different time points and contribute person-years of follow-up.

For exposed women, the index date was the date of the first read code (see Table E1, in this article's Online Repository at www.jaci-inpractice.org) relating to DVA (incident cases), or the date when they became eligible to enter the study if they had a history of DVA (prevalent cases). Up to four unexposed control women with no previously documented read code relating to an exposure were selected for each exposed patient. Unexposed patients were individually matched to registered exposed patients at the same general practice based on age at the index date (to within 1 year) and Townsend deprivation quintile.[23] To account for immortal time bias,[24] matched unexposed patients were assigned the same index date as corresponding exposed patients. The exit date for all patients was defined as the earliest of the following events: the patient died, the patient left the practice, the last data collection from practice, the study end date, or the patient was given a diagnosis with an outcome of interest (atopic disease).

Confounders of age at the index date (continuous data) and Townsend deprivation index (quintiles) were used as both model

TABLE I. Baseline characteristics in women who were exposed and unexposed to domestic abuse

Variable	Exposed group	Unexposed group
Women, n	13,852	49,036
Median (interquartile range) follow-up, person-years	2.45 (1.05-5.05)	3.11 (1.43-5.86)
Mean (SD) age at cohort entry, y	37.20 (12.56)	36.78 (12.32)
Body mass index, n (%)		
Underweight (<18.5 kg/m^2)	632 (4.56)	1,731 (3.53)
Normal (18.5-24.9 kg/m^2)	5,311 (38.34)	19,283 (39.32)
Overweight (25.0-30.0 kg/m^2)	2,905 (20.97)	10,569 (21.57)
Obese (>30.0 kg/m^2)	2,463 (17.78)	9,290 (18.95)
Not available	2,541 (18.34)	8,153 (16.63)
Smoking status, n (%)		
Current smoker	5,942 (42.90)	11,591 (23.64)
Not current smoker	7,470 (53.93)	35,764 (72.93)
Not available	440 (3.18)	1,681 (3.43)
Townsend index, n (%)		
(Least deprived) 1	1,250 (9.02)	4,524 (9.23)
2	1,514 (10.93)	5,376 (10.96)
3	2,334 (16.85)	8,356 (17.04)
4	3,000 (21.66)	10,660 (21.74)
5	3,139 (22.66)	10,825 (22.08)
Not available	2,615 (18.88)	9,295 (18.96)
Ethnicity, n (%)		
Black	593 (4.28)	1,664 (3.39)
Mixed	187 (1.35)	530 (1.08)
Missing	4,135 (29.85)	19,227 (39.21)
Other	393 (2.84)	1,608 (3.28)
South Asian	800 (5.78)	2,282 (4.65)
White	7,744 (55.91)	23,725 (48.38)

covariates and matching parameters. Smoking status (two categories: current smoker, nonsmoker, in which missing data for smoking statistics was classified as nonsmokers, because the risk for misclassification from doing so was thought to be low[25]) was also adjusted for in analyses. These variables were selected based on biological plausibility and have been informed by published reviews of risk factors for the development of atopy.[26,27] A higher Townsend deprivation quintile indicates greater socioeconomic deprivation and is informed by information on employment status, household overcrowding, and car or home ownership.[23] Missing data for Townsend were treated as a separate missing category in the final analysis.

Atopy was defined by the diagnosis of an atopic disease: asthma, atopic eczema, or allergic rhinoconjunctivitis.[28] Crude incidence rates per 1,000 person-years were calculated within each cohort by dividing the number of outcomes by person-years. Because data satisfied the proportional hazard assumption, we used Cox regression analysis to calculate the hazard ratios (HRs) of atopy between the exposed and unexposed groups. Hazard ratios were also adjusted for age at the index date, smoking status, and Townsend deprivation quintile. The HRs are presented with 95% CIs, and statistical significance was determined at P less than .05. We also performed a sensitivity analysis using only incident cases (exposure to DVA occurred during the study period) and corresponding matched controls.

Exposed women were matched at a 1:4 ratio by age (with variations of up to 1 year allowed) and Townsend deprivation quintile to unexposed women (those with no GP-recorded exposure to DVA). Cox proportional hazards regression was used to calculate crude and adjusted HRs for outcomes in exposed compared with unexposed women. The methods for this study are similar to those in previously published work using IMRD to examine health outcomes for survivors of DVA.[9] Two sensitivity analyses were undertaken to assess the robustness of the results: (1) restricting the study to women whose exposure to DVA occurred during the study period (incident-only women); and (2) adding ethnicity as a covariate of interest, with missing ethnicity as a missing category.

Ethical approval

Anonymized data were used from the data provider to the University of Birmingham. Use of IMRD is approved by the UK Research Ethics Committee (Reference No. 18/LO/0441). In accordance with this approval, the study protocol must be reviewed and approved by an independent scientific review committee. The IMRD incorporates data from The Health Improvement Network (THIN), a Cegedim Database. Reference made to The Health Improvement Network is intended to be descriptive of the data asset licensed by IQVIA. This work will use deidentified data provided by patients as a part of routine primary care. For this project, an amendment to approved Scientific Review Committee Protocol 18THIN034 has been approved.

Data access statement

The data and analysis code can be obtained from the corresponding author following appropriate ethical approval.

RESULTS

During the study period, 13,852 women were identified as having an exposure to DVA. They were matched with 49,036 women with no recorded exposure to DVA. Women in the exposed group were observed for a shorter median of 2.45 years (interquartile range [IQR], 1.05-5.05 years) compared with the unexposed cohort, which was observed for 3.11 years (IQR, 1.43-5.86 years). Mean age at cohort entry and Townsend deprivation index were similar between groups because of matching. Those in the exposed group were more likely to be current smokers than those in the unexposed group (42.90% vs 23.64%). There was a high proportion of missing ethnicity data. Table I lists further details about the demographic characteristics of both groups.

During the study period, 967 women (incidence rate, 20.10/1,000 person-years) were given the diagnosis of atopic disease in the exposed cohort compared with 2,607 (incidence rate, 13.24/1,000 person-years) in the unexposed cohort. After adjustment for potential confounders, this translated to an adjusted HR (aHR) of 1.52 (95% CI, 1.41-1.64). The association was strongest in the development of asthma (aHR = 1.69; 95% CI, 1.44-1.99) and allergic rhinoconjunctivitis (aHR = 1.63; 95% CI, 1.45-1.84) compared with atopic eczema (aHR = 1.40; 95% CI, 1.26-1.56). Table II lists further details.

When we restricted results to women whose exposure to DVA occurred during the study period (incident-only women), we identified 3,047 eligible exposed women who were matched with 10,855 unexposed women. The demographic details were similar

TABLE II. Risk for developing atopic disease during study period in women exposed and unexposed to domestic abuse

	All atopic disease		Asthma		Atopic eczema		Allergic rhinoconjunctivitis	
Outcomes and effect sizes	**Exposed**	**Unexposed**	**Exposed**	**Unexposed**	**Exposed**	**Unexposed**	**Exposed**	**Unexposed**
Outcomes, n	967	2,607	220	491	456	1,311	369	971
Person-years	48099	196866	51163	205623	50169	202176	50479	203551
Incidence rate (per 1,000 person-years)	20.10	13.24	4.30	2.39	9.09	6.48	7.31	4.77
Hazard ratio (95% CIs)*	1.50 (1.40-1.62)		1.80 (1.53-2.11)		1.39 (1.25-1.55)		1.52 (1.35-1.71)	
P	<.001		<.001		<.001		<.001	
Adjusted hazard ratio (95% CIs)†	1.52 (1.41-1.64)		1.69 (1.44-1.99)		1.40 (1.26-1.56)		1.63 (1.45-1.84)	
P	<.001		<.001		<.001		<.001	

*Unadjusted hazard ratio.
†Adjusted hazard ratio: adjusted for age, Townsend deprivation quintile, and smoking status at study entry.

to the main cohort (see Table E2, in this article's Online Repository at www.jaci-inpractice.org). Those exposed to DVA were at a greater risk for developing atopic disease (aHR = 1.18; 95% CI, 1.00-1.40), with the strongest association seen for the development of asthma (aHR = 1.57; 95% CI, 1.12-2.19). Further details can be seen in Table E3 (in this article's Online Repository at www.jaci-inpractice.org). In addition, when we included ethnicity as a covariate in the main model, the results remained robust with the primary analysis (see Table E4 in this article's Online Repository at www.jaci-inpractice.org).

DISCUSSION

To our knowledge, this is the first attempt to explore the relationship between DVA and the development of multiple atopic diseases in adults using a longitudinal study design in a large generalizable UK database. Our study found that women with recorded exposure to DVA had a 52% increased risk for developing atopic disease. These findings remained robust in a sensitivity analysis of incident-only cases.

Our results support existing evidence that exposure to DVA may be associated with subsequent diagnoses and development of asthma, and provide new evidence that DVA is positively associated with a diagnosis of atopic eczema and allergic rhinoconjunctivitis. Because existing cohort evidence has not been previously explored, it is not possible to compare our observed incidence rates with the existing literature. This is similarly the case for the observed associations with eczema and allergic rhinoconjunctivitis. Although not directly comparable, some cross-sectional evidence exists reporting increased odds of asthma in those exposed to DVA compared with those who were unexposed.[14-17]

Our findings are of relevance in a UK setting and to the global population. The prevalence of atopic diseases is increasing, in which the United Kingdom has one of the highest prevalences globally and an estimated 20% of the global population is affected.[29,30] Because DVA is estimated to affect one in three women,[1] a considerable proportion of atopic diagnoses may be affected by DVA. This points to the need for the swift implementation of public health measures to reduce DVA and its secondary consequences. Because gender inequality is a fundamental cause of DVA, behavioral and policy interventions are critical to addressing this form of gender-based violence. Second, clinicians should be alert to the potential for DVA in patients presenting with atopic disease. Third, in those who have experienced abuse, interventions to prevent the development of ill health are imperative, because evidence indicates that suitable intervention programs (such as social support programs and psychotherapy) are effective in ameliorating the harmful consequences of DVA.[31-33]

In terms of the limitations of our study, we could not accounted for unrecorded or unsuspected cases of DVA, which may have resulted in misclassification bias. Also, it is possible that some exposed patients might have been misclassified as victims whereas they could have been perpetrators. However, DVA is a gendered crime, and most perpetrators are male; even in cases of male domestic homicide, the suspect is more commonly male.[34] Hence, we anticipate this misclassification bias to be low. Equally, it is possible that GPs may not have accurately utilized read codes or completely recorded the outcomes of interest. However, because some atopic diseases feature in the Quality and Outcomes Framework,[35] it is anticipated that records of these conditions are likely to be complete. Nevertheless, it is possible that the outcome was affected by misclassification bias, in which diagnosticians were unable to differentiate atopic from nonatopic causes of eczema and asthma. However, we would expect this to be nondifferential between groups. The use of the IMRD database provided a large and diverse sample that is generalizable to the UK population in terms of age structure and the prevalence of chronic disease.[19] There are also substantial levels of missing ethnicity data in the IMRD dataset. As noted in the literature, ethnicity recording in UK primary care is limited until the mid-2000s.[36] Hence, we anticipate improved recording in the latter years of the study. Finally, the median follow-up for the exposed and unexposed groups was relatively short, at 2.45 person-years (IQR, 1.05-5.05) and 3.11 person-years (IQR, 1.43-5.86), respectively. Given the natural history of atopic diseases and their relapsing-remitting nature, this may have resulted in a right-censoring bias.

Our study found that women who are exposed to DVA are at increased risk for developing atopic disease, in alignment with previous observational data. Therefore, implementation of systematic public health measures, adopting the consideration of DVA in clinical interactions with patients who present with ill health, and encouragement of measures to prevent DVA in wider society by public health professionals and its devastating downstream consequences are urgently needed.

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ONLINE REPOSITORY

TABLE E1. Read codes for exposure and outcomes

Clinical code	Description
Domestic violence and abuse	
14X3.00	History of domestic violence
14X8.00	Victim of domestic violence
14XD.00	History of domestic abuse
14XD000	H/O domestic emotional abuse
14XD100	H/O domestic physical abuse
14XD200	H/O domestic sexual abuse
14XE.00	History of being victim of domestic violence
14XG.00	Victim of domestic abuse
Allergic rhino-conjunctivitis	
F4A3100	Vernal conjunctivitis of limbus and cornea
F4C0600	Acute atopic conjunctivitis
F4C0611	Acute allergic conjunctivitis
F4C1300	Vernal conjunctivitis
F4C1400	Other chronic allergic conjunctivitis
F4C1411	Allergic conjunctivitis
H17.00	Allergic rhinitis
H17.11	Perennial rhinitis
H170.00	Allergic rhinitis due to pollens
H170.11	Hay fever- pollens
H170.12	Pollinosis
H171.00	Allergic rhinitis due to other allergens
H171.11	Cat allergy
H171.12	Dander (animal) allergy
H171.13	Feather allergy
H171.14	Hay fever-other allergen
H171.15	House dust allergy
H171.16	House dust mite allergy
H171000	Allergy to animal
H171100	Dog allergy
H172.00	Allergic rhinitis due to unspecified allergen
H172.11	Hay fever-unspecified allergen
H17z.00	Allergic rhinitis NOS
H330.13	Hay fever with asthma
H330011	Hay fever with asthma
Hyu2000	[X]Other seasonal allergic rhinitis
Hyu2100	[X]Other allergic rhinitis
Asthma MEDICAL_CODE_ID	
H312000	Chronic asthmatic bronchitis
H33..00	Asthma
H33..11	Bronchial asthma
H330.00	Extrinsic (atopic) asthma
H330.11	Allergic asthma
H330.12	Childhood asthma
H330.13	Hay fever with asthma
H330.14	Pollen asthma
H330000	Extrinsic asthma without status asthmaticus
H330011	Hay fever with asthma
H330100	Extrinsic asthma with status asthmaticus
H330111	Extrinsic asthma with asthma attack
H330z00	Extrinsic asthma NOS
H331.00	Intrinsic asthma
H331.11	Late-onset asthma
H331000	Intrinsic asthma without status asthmaticus
H331100	Intrinsic asthma with status asthmaticus
H331111	Intrinsic asthma with asthma attack
H331z00	Intrinsic asthma NOS
H332.00	Mixed asthma
H333.00	Acute exacerbation of asthma
H334.00	Brittle asthma
H33z.00	Asthma unspecified
H33z.11	Hyperreactive airways disease
H33z000	Status asthmaticus NOS
H33z011	Severe asthma attack
H33z100	Asthma attack
H33z111	Asthma attack NOS
H33z200	Late-onset asthma
H33zz00	Asthma NOS
H33zz11	Exercise-induced asthma
H33zz12	Allergic asthma NEC
H33zz13	Allergic bronchitis NEC
Atopic eczema	
M07y.11	Pustular eczema
M102.11	Pustular eczema
M11..00	Atopic dermatitis and related conditions
M111.00	Atopic dermatitis/eczema
M113.00	Flexural eczema
M114.00	Allergic (intrinsic) eczema
M115.00	Besnier's prurigo
M117.00	Neurodermatitis -atopic
M11z.00	Atopic dermatitis NOS
M12z100	Eczema NOS
M12z200	Infected eczema
M12z400	Erythrodermic eczema
Myu2.00	[X]Dermatitis and eczema
Myu2200	[X]Exacerbation of eczema

H/O, history of; NOS, not otherwise specified; NEC, not elsewhere classified.

TABLE E2. Incident-only cases: baseline characteristics in women who were exposed and unexposed to domestic violence and abuse

Variables	Exposed group	Unexposed group
Number of patients	3,047	10,855
Median (interquartile range) follow-up, person-years)	2.63 (1.12-5.15)	3.00 (1.38-5.48)
Mean (SD) age at cohort entry, y	37.76 (12.62)	37.33 (12.43)
Body mass index, n (%)		
Underweight (<18.5 kg/m^2)	116 (3.81)	372 (3.43)
Normal (18.5-24.9 kg/m^2)	1,274 (41.81)	4,348 (40.06)
Overweight (25.0-30.0 kg/m^2)	687 (22.55)	2,449 (22.56)
Obese (>30.0 kg/m^2)	563 (18.48)	2,147 (19.78)
Not available	407 (13.36)	1,539 (14.18)
Smoking status, n (%)		
Current smoker	1,263 (41.45)	2,508 (23.10)
Not current smoker	1,751 (57.47)	8,025 (73.93)
Not available	33 (1.08)	322 (2.97)
Townsend index, n (%)		
(Least deprived) 1	332 (10.90)	1,232 (11.35)
2	365 (11.98)	1,285 (11.84)
3	529 (17.36)	1,879 (17.31)
4	638 (20.94)	2,271 (20.92)
5	672 (22.05)	2,356 (21.70)
Not available	511 (16.77)	1,832 (16.88)
Ethnicity, n (%)		
Black	111 (3.64)	400 (3.68)
Missing	1,075 (35.28)	3,929 (36.20)
Mixed	34 (1.12)	95 (0.88)
Other	75 (2.46)	380 (3.50)
South Asian	166 (5.45)	574 (5.29)
White	1,586 (52.05)	5,477 (50.46)

TABLE E3. Incident-only cases: Risk for developing atopic disease during study period in women who were exposed and unexposed to domestic violence and abuse

	All atopic disease		Asthma		Atopic eczema		Allergic rhinoconjunctivitis	
Outcomes and effect sizes	**Exposed**	**Unexposed**	**Exposed**	**Unexposed**	**Exposed**	**Unexposed**	**Exposed**	**Unexposed**
Outcomes, n	185	596	50	117	81	290	69	221
Person-years	10,660	40,957	11,147	42,819	11,049	42,133	11,073	42,372
Incidence rate per 1,000 person-years	17.36	14.55	4.49	2.73	7.33	6.88	6.23	5.22
Hazard ratio (95% CIs)*	1.19 (1.01-1.40)		1.64 (1.18-2.29)		1.06 (0.83-1.36)		1.19 (0.91-1.56)	
P	.042		.003		.647		.205	
Adjusted hazard ratio (95% CIs)†	1.18 (1.00-1.40)		1.57 (1.12-2.19)		1.05 (0.82-1.35)		1.23 (0.93-1.62)	
P	.052		.009		.693		.142	

*Unadjusted hazard ratio.
†Adjusted hazard ratio, adjusted for age, Townsend deprivation quintile, and smoking status at study entry.

TABLE E4. Ethnicity analysis: Risk for developing atopic disease during study period in women who were exposed and unexposed to domestic abuse

	All atopic disease		Asthma		Atopic eczema		Allergic rhinoconjunctivitis	
Outcomes and effect sizes	**Exposed**	**Unexposed**	**Exposed**	**Unexposed**	**Exposed**	**Unexposed**	**Exposed**	**Unexposed**
Outcomes, n	967	2,607	220	491	456	1,311	369	971
Person-years	48,099	196,866	51,163	205,623	50,169	202,176	50,479	203,551
Incidence rate (per 1,000 person-years)	20.10	13.24	4.30	2.39	9.09	6.48	7.31	4.77
Hazard ratio (95% CIs)*	1.50 (1.40-1.62)		1.80 (1.53-2.11)		1.39 (1.25-1.55)		1.52 (1.35-1.71)	
P	<.001		<.001		<.001		<.001	
Adjusted hazard ratio (95% confidence intervals)†	1.52 (1.41-1.64)		1.69 (1.44-1.99)		1.40 (1.26-1.56)		1.63 (1.45-1.84)	
p-value	<.001		<.001		<.001		<.001	
Adjusted hazard ratio (95% CIs)‡	1.47 (1.37-1.59)		1.68 (1.43-1.98)		1.36 (1.22-1.52)		1.55 (1.37-1.75)	
P	<.001		<.001		<.001		<.001	

*Unadjusted hazard ratio.
†Adjusted hazard ratio, adjusted for age, Townsend deprivation quintile, and smoking status at study entry.
‡Adjusted hazard ratio, adjusted for age, Townsend deprivation quintile, ethnicity (missing category), and smoking status at study entry.