

The relationship between intimate partner violence and children's asthma in 10 US states/territories

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Intimate partner violence (IPV) has been shown to negatively impact the health of both the adults who experience IPV and the children who are exposed to IPV. Although IPV experienced by women has been linked to children's asthma, this study is the first to examine this question among both women and men, and the first study in the United States to examine this question as part of a population-based data set. In 2005, ten US states/territories administered an IPV module and a children's asthma module within the Behavioral Risk Factor Surveillance System (BRFSS). Lifetime IPV was assessed by four questions that asked about threatened, attempted, or completed physical violence, as well as unwanted sex, by a current or former intimate partner. The children's asthma module asked respondents to report whether a randomly selected child in their household had ever been diagnosed with asthma and whether the same child currently had asthma. Women who experienced lifetime IPV, in contrast to women who never experienced IPV, were significantly more likely to report that their children had ever had asthma and currently have asthma. Among men, significant differences were not found when comparing men who reported lifetime IPV to those that did not report lifetime IPV. The results highlight the importance of primary prevention of IPV, as reducing the occurrence of IPV could improve not only the long-term health of those who experience IPV but also the health of their children.

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Intimate partner violence (IPV) is a significant public health problem associated with a range of short- and long-term negative mental and physical health outcomes among those who experience IPV (1). IPV is defined as threatened, attempted or completed physical or sexual violence, or emotional abuse by a current or former intimate partner. IPV can be committed by a spouse, ex-spouse, current or former boyfriend or girlfriend, dating partner or date (2). Prior studies have demonstrated the deleterious effects of exposure to IPV on the health and well-being of children, including anxiety, depression, deficient school performance, and poor physical health (3).

While much of the work examining asthma has implicated the role of environmental exposures, it has also been demonstrated that asthma is socially patterned given its association with poverty, race, and urban residence (4). Stressful life events have been implicated as a potential explanation for variations in asthma morbidity by way of the impact of psychological stress on immune system functioning (5). The biologic response to psychological stress (e.g., elevated heart rate, release of stress hormones) can be functional in the short term, but also harmful should these responses continue over long periods because of the effects of stress (6). Chronic stress has been shown to permanently alter the

level at which physiological systems operate. For example, there is evidence to suggest that a chronically elevated stress response can lead to attenuation of stress hormones (i.e., cortisol) that counteract the inflammatory response that precipitates asthma (6).

Recently, researchers have examined a particularly stressful experience, exposure to violence, in relation to increased levels of asthma (7, 8). Investigators have also examined the role of a particular form of violence, IPV, and children's asthma. One study found that children at 36 months of age were more likely to have been diagnosed with asthma if their mother had previously reported chronic IPV (9). In the only population-based study we are aware of, a cross-sectional study in India found that mothers who had experienced IPV were more likely to report that one of their children had asthma (10).

Previous studies have examined whether women's experience of IPV was associated with asthma among their children. However, no previous studies have examined whether men's experience of IPV similarly affects children. The current study examines IPV experienced by both men and women and is the first study in the United States to examine this question as part of a population-based data set. It is hypothesized that the children of both women and men who report IPV will be more likely to have been diagnosed with asthma, and more likely to be currently experiencing asthma. While a number of previous studies have examined the relationship between parental IPV and children's health, most, if not all have based these findings on IPV victimization reported by mothers or children's self-report of witnessing violence between parents. Consequently, there have been few, if any, studies specifically examining the associated health outcomes of fathers' IPV victimization on children.

Methods

The BRFSS is an annual random-digit-dial telephone survey developed by CDC to provide surveillance of health behaviors and health risks among the non-institutionalized adult population of the United States and several US territories. In 2005, an IPV module, a random child selection module, and a children's asthma module were available for use at the discretion of each state/territory. Nine states and one territory administered all three modules: AZ (Council of American Survey Research Organization response rate: 48.6%), IA (60.2%), MI (51.1%), NM (58.9%), OH (49.5%), OR (51.5%), RI

(41.3%), VT (52.8%), VA (51.7%), and Puerto Rico (72.7%) (11). In total, 11,193 respondents (7212 women, 3981 men) completed each of the modules. The design and characteristics of BRFSS are described in greater detail elsewhere (11).

Measures

Respondents were classified as having experienced IPV victimization if they reported having EVER experienced any of the following: threatened, attempted, or completed physical violence and/or non-consensual sex perpetrated by a current or former intimate partner. IPV module questions were designed to be behaviorally specific and were based upon the CDC's uniform definitions of IPV (2). Precise question wording has been previously published (11).

All BRFSS respondents were asked how many children <18 yr of age lived in the household. If a child resided in the home, then one of the children was randomly selected by the interviewer. Respondents who reported that their relationship with the randomly selected child was that of parent or guardian remained in the analysis, whereas those related in other ways to the child (i.e., grandparent, sibling) were excluded. The childhood asthma module asked, 'has a doctor, nurse or other health professional ever said that the child has asthma?' If a 'yes' response was given, the respondent was asked, 'does the child still have asthma?'

Analysis

Data were weighted to provide estimates representative of each state's population. The dependent variables were whether the child had ever been diagnosed with asthma and whether the child currently had asthma. Analyses were stratified by sex of respondent. Bivariate logistic regression was used to model parent's lifetime IPV predicting child's asthma status. Next, multivariate logistic regression models were calculated by adding relevant controls: age of child, parental race/ethnicity, income, education, health care coverage, previous asthma diagnosis, current smoking, and rural/urban status.

Results

Among respondents, a number of parent demographic characteristics varied in relation to the lifetime experience of IPV (Table 1). Specifically, race/ethnicity, prior asthma diagnosis, and current smoking varied by IPV status for both men

Table 1. Demographic characteristics by sex and IPV status

	Women			Men		
	IPV% (s.e.)	No IPV% (s.e.)	p-Value	IPV% (s.e.)	No IPV% (s.e.)	p-Value
Race/Ethnicity						
White NH	74.2 (1.4)	67.2 (1.1)	<0.001	66.3 (2.9)	74.0 (1.2)	<0.01
Black NH	8.6 (0.9)	7.5 (0.6)		10.4 (2.2)	4.3 (0.6)	
Other NH	5.1 (0.7)	5.3 (0.6)		8.3 (1.8)	5.2 (0.6)	
Hispanic	11.9 (1.0)	19.7 (1.0)		14.9 (2.0)	16.1 (1.0)	
Parent age						
18–24	10.0 (1.4)	10.6 (1.0)	0.40	5.0 (1.7)	5.0 (0.9)	0.11
25–34	37.2 (1.6)	33.0 (1.1)		35.6 (2.9)	27.8 (1.3)	
35–44	37.2 (1.6)	39.6 (1.1)		35.6 (2.8)	38.7 (1.3)	
45–54	14.6 (1.1)	15.9 (0.8)		21.4 (2.3)	24.4 (1.1)	
55–64	0.9 (0.2)	0.9 (0.2)		2.2 (0.7)	3.5 (0.5)	
65+	–	–		0.3 (0.2)	0.6 (0.3)	
Income						
<\$15k	12.3 (1.1)	7.8 (0.8)	<0.001	4.5 (1.1)	4.1 (0.7)	0.07
\$15k–\$25k	16.1 (1.2)	12.6 (0.7)		15.7 (2.5)	9.5 (0.8)	
\$25k–\$35k	12.2 (1.0)	9.4 (0.7)		9.4 (1.5)	8.7 (0.7)	
\$35k–\$50k	14.2 (1.2)	15.0 (0.8)		12.7 (1.8)	14.7 (0.9)	
\$50k+	40.1 (1.7)	46.4 (1.1)		51.8 (3.0)	59.5 (1.3)	
Unknown	5.1 (0.7)	8.9 (0.6)		6.0 (1.5)	3.6 (0.5)	
Education						
Did not Grad. HS	8.7 (1.0)	7.2 (0.8)	<0.001	8.4 (1.8)	7.0 (0.8)	0.13
HS Grad.	30.2 (1.6)	27.3 (1.0)		28.1 (2.8)	26.0 (1.2)	
Some college	33.9 (1.6)	27.7 (1.0)		28.0 (2.5)	24.1 (1.1)	
College Grad.	27.3 (1.4)	37.9 (1.1)		35.3 (2.8)	43.0 (1.3)	
Residence						
Urban	62.1 (1.6)	62.6 (1.0)	0.62	60.9 (2.9)	62.3 (1.2)	0.53
Suburban	16.0 (1.3)	16.9 (0.8)		18.2 (2.3)	15.4 (0.9)	
Rural	22.0 (1.3)	20.6 (0.8)		21.0 (2.3)	22.3 (1.0)	
Health care coverage						
Yes	84.6 (1.2)	87.8 (0.8)	0.11	83.1 (2.3)	86.9 (1.1)	0.23
No	15.1 (1.2)	12.2 (0.8)		16.8 (2.3)	13.0 (1.1)	
Asthma diagnosis						
Yes	21.3 (1.4)	12.1 (0.7)	<0.001	17.9 (2.4)	10.3 (0.8)	<0.01
No	78.7 (1.4)	87.7 (0.7)		81.8 (2.4)	89.7 (0.8)	
Current smoking						
Yes	34.9 (1.6)	16.8 (1.0)	<0.001	29.7 (2.7)	18.8 (1.1)	<0.001
No	65.1 (1.6)	83.2 (1.0)		70.3 (2.7)	81.2 (1.1)	

NH, non-hispanic; s.e., standard error; IPV, intimate partner violence.

and women, and varied by income and education for women. Also, a number of parent demographic characteristics varied in relation to children's lifetime asthma diagnosis and current asthma (Table 2). Women's and men's race/ethnicity, age, income, education, and prior asthma diagnosis were significantly associated with children's lifetime asthma diagnosis, as was women's lifetime experience of IPV. Women's and men's race/ethnicity, income, and prior asthma diagnosis were associated with children's current asthma, as was men's age and women's lifetime experience of IPV.

Relationship between mothers' IPV and children's asthma

The percentage of women who reported that their child ever had asthma was 14.4% (95%

confidence interval [CI] = 12.6–16.2) among those who never experienced IPV, compared to 18.7% (95% CI = 16.2–21.2) among those who experienced IPV (Table 3). Logistic regression modeling indicated that the children of women who experienced IPV were significantly more likely to have ever had asthma (crude odds ratio [COR] = 1.50, 95% CI = 1.22–1.83). This relationship remained significant after adding control variables into the model (adjusted odds ratio [AOR] = 1.29, 95% CI = 1.01–1.65).

The percentage of women who reported that their child currently had asthma was 8.9% (95% CI = 7.7–10.0) among those who never experienced IPV, compared to 13.6% (95% CI = 11.5–15.7) among those who experienced IPV. Logistic regression modeling indicated that the children of women who experienced IPV were

Table 2. Demographic characteristics by sex of parent and child asthma status

	Women			Men			Women			Men		
	Child asthma ever% (s.e.)	No asthma% (s.e.)	p-Value	Child asthma ever% (s.e.)	No asthma% (s.e.)	p-Value	Child current asthma% (s.e.)	No current asthma% (s.e.)	p-Value	Child current asthma% (s.e.)	No current asthma% (s.e.)	p-Value
Race/Ethnicity												
White NH	59.1 (2.2)	71.1 (0.9)	<0.001	64.0 (3.2)	74.0 (1.2)	<0.05	60.6 (2.6)	70.4 (0.9)	<0.01	70.6 (3.9)	73.1 (1.2)	<0.05
Black NH	10.6 (1.3)	7.3 (0.5)		5.8 (1.8)	5.1 (0.7)		11.9 (1.7)	7.3 (0.5)		4.9 (1.9)	5.3 (0.7)	
Other NH	4.7 (1.1)	5.3 (0.5)		6.6 (1.7)	5.6 (0.6)		5.6 (1.4)	5.2 (0.5)		4.8 (1.7)	5.7 (0.6)	
Hispanic	24.3 (1.8)	16.1 (0.8)		23.1 (2.8)	14.9 (1.0)		20.9 (2.1)	16.9 (0.8)		19.7 (3.5)	15.5 (0.9)	
Parent age												
18–24	5.5 (1.2)	11.1 (0.9)	<0.001	1.0 (0.6)	5.4 (0.8)	<0.001	6.6 (1.6)	10.8 (0.9)	0.10	1.0 (0.8)	5.3 (0.8)	<0.001
25–34	32.7 (2.2)	34.4 (1.0)		16.3 (2.6)	30.5 (1.2)		34.7 (2.6)	34.1 (1.0)		15.9 (3.3)	30.1 (1.2)	
35–44	39.5 (2.1)	38.8 (1.0)		50.5 (3.4)	36.9 (1.2)		39.2 (2.5)	38.9 (1.0)		47.6 (4.2)	37.5 (1.2)	
45–54	21.1 (1.9)	14.7 (0.6)		26.5 (2.8)	23.6 (1.1)		18.3 (2.0)	15.3 (0.7)		28.7 (3.7)	23.5 (1.0)	
55–64	1.2 (0.4)	0.9 (0.1)		5.5 (1.4)	3.0 (0.4)		1.2 (0.5)	0.9 (0.1)		6.4 (2.0)	3.1 (0.4)	
65+	—	—		0.3 (0.2)	0.5 (0.2)		—	—		0.4 (0.3)	0.5 (0.2)	
Income												
<\$15k	11.2 (1.2)	8.7 (0.7)	<0.001	7.1 (1.6)	3.8 (0.6)	<0.001	11.3 (1.5)	8.9 (0.7)	<0.001	8.3 (2.2)	3.8 (0.6)	<0.001
\$15k–\$25k	17.3 (1.7)	12.9 (0.7)		10.4 (1.9)	10.4 (0.8)		16.5 (2.0)	13.3 (0.7)		7.9 (1.8)	10.7 (0.8)	
\$25k–\$35k	12.0 (1.5)	9.9 (0.6)		10.9 (2.1)	8.6 (0.7)		13.7 (1.9)	9.8 (0.6)		9.9 (2.6)	8.8 (0.7)	
\$35k–\$50k	15.4 (1.6)	14.6 (0.7)		13.2 (2.0)	14.5 (0.9)		14.9 (1.9)	14.7 (0.7)		12.6 (2.5)	14.5 (0.9)	
\$50k+	34.9 (2.2)	46.2 (1.0)		57.6 (3.2)	58.5 (1.3)		33.6 (2.5)	45.7 (1.0)		60.7 (4.0)	58.1 (1.2)	
Unknown	9.3 (1.4)	7.6 (0.5)		1.0 (0.4)	4.3 (0.5)		9.9 (1.6)	7.6 (0.5)		0.6 (0.4)	4.2 (0.5)	
Education												
Did not Grad. HS	7.8 (1.1)	7.6 (0.7)	<0.05	4.3 (1.0)	7.5 (0.8)	<0.01	7.9 (1.3)	7.6 (0.7)	0.12	4.7 (1.4)	7.4 (0.8)	0.08
HS Grad.	24.0 (1.9)	28.9 (1.0)		22.9 (2.7)	26.7 (1.2)		24.6 (2.3)	28.6 (0.9)		22.9 (3.3)	26.6 (1.1)	
Some college	34.0 (2.1)	28.7 (0.9)		32.7 (3.0)	23.8 (1.1)		34.8 (2.6)	28.9 (0.9)		32.6 (3.8)	24.3 (1.0)	
College Grad.	34.2 (2.2)	34.9 (0.9)		40.2 (3.4)	41.9 (1.3)		32.8 (2.5)	35.0 (0.9)		39.8 (4.3)	41.6 (1.2)	
Residence												
Urban	62.7 (2.3)	62.6 (0.9)	0.10	58.1 (3.6)	62.4 (1.2)	0.19	64.3 (2.7)	62.4 (0.9)	0.53	57.8 (4.4)	62.1 (1.2)	0.43
Suburban	17.3 (1.9)	16.3 (0.7)		16.4 (2.7)	16.0 (0.9)		16.9 (2.2)	16.4 (0.7)		14.8 (3.0)	16.2 (0.9)	
Rural	20.1 (1.8)	21.1 (0.7)		25.6 (3.2)	21.6 (1.0)		18.8 (2.0)	21.2 (0.7)		27.3 (4.1)	21.7 (1.0)	
Health care coverage												
Yes	86.6 (1.6)	86.8 (0.8)	0.41	89.8 (2.2)	85.9 (1.1)	0.48	86.8 (1.9)	86.8 (0.7)	0.32	91.3 (2.4)	85.9 (1.1)	0.10
No	12.9 (1.5)	13.2 (0.8)		10.3 (2.2)	14.1 (1.1)		12.8 (1.9)	13.2 (0.7)		8.7 (2.4)	14.1 (1.1)	
Asthma diagnosis												
Yes	28.6 (2.0)	12.5 (0.7)	<0.001	19.6 (2.6)	10.4 (0.8)	<0.01	31.2 (2.4)	13.0 (0.7)	<0.001	22.5 (3.3)	10.5 (0.8)	<0.01
No	71.3 (2.0)	87.4 (0.7)		80.2 (2.6)	89.6 (0.8)		68.8 (2.4)	87.0 (0.7)		77.2 (3.3)	89.5 (0.8)	
Current smoking												
Yes	24.6 (1.9)	21.8 (0.9)	0.12	22.0 (2.7)	20.3 (1.1)	0.64	24.2 (2.2)	21.9 (0.9)	0.33	18.4 (2.8)	20.7 (1.1)	0.46
No	75.4 (1.9)	78.2 (0.9)		78.0 (2.7)	79.7 (1.1)		75.8 (2.2)	78.1 (0.9)		81.6 (2.8)	79.3 (1.1)	
Lifetime IPV												
Yes	38.2 (2.2)	28.9 (0.9)	<0.001	16.7 (2.4)	15.5 (0.9)	0.42	39.8 (2.6)	29.1 (0.9)	<0.001	17.0 (2.9)	15.6 (0.9)	0.65
No	61.8 (2.2)	71.1 (0.9)		83.3 (2.4)	84.5 (0.9)		60.2 (2.6)	70.9 (0.9)		83.0 (2.9)	84.4 (0.9)	

NH, non-hispanic; s.e., standard error; IPV, intimate partner violence.

Table 3. Association between intimate partner violence and children's asthma

Parent Sex	Parent IPV status	Child asthma, ever (95% CI)	COR (95% CI)	AOR* (95% CI)	Child asthma, current (95% CI)	COR (95% CI)	AOR* (95% CI)
Male	No IPV	13.1% (10.3–15.9)	1.07 (0.74–1.53)	1.09 (0.73–1.62)	6.8% (5.6–8.0)	1.10 (0.72–1.69)	1.11 (0.68–1.80)
	Lifetime IPV	12.3% (8.8–15.9)			7.4% (4.8–10.1)		
Female	No IPV	14.4% (12.6–16.2)	1.50 (1.22–1.83)	1.29 (1.01–1.65)	8.9% (7.7–10.0)	1.61 (1.28–2.03)	1.40 (1.06–1.85)
	Lifetime IPV	18.7% (16.2–21.2)			13.6% (11.5–15.7)		

AOR, adjusted odds ratio; COR, crude odds ratio; CI, confidence interval; IPV, intimate partner violence.

*Control variables include: age of child, parental race/ethnicity, income, education, health care coverage, previous asthma diagnosis, current smoking, and rural/urban status.

significantly more likely to currently have asthma (COR = 1.61, 95% CI = 1.28–2.03). This relationship remained significant after adding control variables into the model (AOR = 1.40, 95% CI = 1.06–1.85).

Relationship between fathers' IPV and children's asthma

The percentage of men who reported that their child ever had asthma was 13.1% (95% CI = 10.3–15.9) among those who never experienced IPV, compared to 12.3% (95% CI = 8.8–15.9) among those who experienced IPV. The relationship between whether a child ever had asthma and men's lifetime IPV was non-significant both with and without controls entered into the logistic regression models (COR = 1.07, 95% CI = 0.74–1.53; AOR = 1.09, 95% CI = 0.73–1.62).

The percentage of men who reported that their child currently had asthma was 6.8% (95% CI = 5.6–8.0) among those who never experienced IPV, compared to 7.4% (95% CI = 4.8–10.1) among those who experienced IPV. The relationship between children's current asthma and men's lifetime IPV was non-significant both with and without controls entered into the logistic regression models (COR = 1.10, 95% CI = 0.72–1.69; AOR = 1.11, 95% CI = 0.68–1.80).

Conclusions

Consistent with previous studies, and consistent with our hypothesis, women who experienced IPV were more likely to report that their child had ever been diagnosed with asthma and were more likely to report that their child currently had asthma. Contrary to our hypothesis, IPV experienced by men was not associated with asthma among their children.

Further work is needed to directly test the specific mechanisms by which IPV, and violence in general, is associated with a greater likelihood of developing asthma among children. Previous

studies have shown that children who had previously been exposed to IPV were more likely to display elevated heart rate and higher cortisol levels, indicative of heightened autonomic arousal and hypothalamic–pituitary–adrenal (HPA) axis functioning (12). Stressful life events in general have been implicated as predictors of asthma diagnosis and severity among adolescents (5). IPV, as well as other types of violence witnessed or experienced by children, have been conceptualized as one of a number of 'social toxins' that may be important in explaining health disparities between groups of people (13).

There are a number of possible explanations for the results found in this study. Previous studies have shown that maternal stress may adversely affect a fetus *in utero* such that a child can be born with a permanently heightened response to stress (14). Further, one study examined the relationship between pregnant women's prior experience of physical or sexual violence and the presence of elevated IgE levels in cord blood at the birth of a child (15). Elevated IgE levels, a predictor of subsequent atopic risk among children, was associated with women's reports of having previously experienced physical or sexual violence. These studies indicate that children's risk for developing asthma and other atopic disorders may be due in part to violence that occurred prior to their conception. This work also provides a potential explanation for the association between children's asthma and IPV among mothers, but not among fathers.

Another important early influence on a child's stress regulation system is the quality of maternal caregiving (16, 17). Maternal stress has been shown to negatively impact the quality of interactions mothers have with their infant children (18). Further, the relative quality of mother–infant interactions has been shown to affect cortisol levels and the reactivity of young children's HPA system (17). The significant relationship between mother's IPV and children's asthma, but not for father's IPV and children's asthma, might be explained by these previous

findings. In addition, the effect of stress on mother–infant interactions may be amplified by the larger role women continue to play, relative to men, in parenting children (19).

Another possible explanation for the discrepant findings among the children of males and females who experienced IPV is the difference in the severity of IPV experienced by men and women. Previous studies have indicated that, on average, women experience more severe IPV than men and are more likely to be injured (20). The relatively greater severity of the IPV experienced by women on average may affect children by way of relatively greater levels of stress among mothers, or by direct witnessing of more severe IPV by children. However, it is important to note that the current data do not capture whether children personally witnessed the IPV experienced by parents, or when the IPV occurred. As a result, it is not possible to estimate the degree to which direct witnessing of IPV by children played a role in the findings.

This study has a number of limitations. First, because of the cross-sectional nature of the study, we were unable to assess temporal precedence or causality. Also, the number and range of questions that could be included in the IPV module were limited by space within BRFSS. Consequently, our models were not able to factor in variation in the severity, chronicity, and context of IPV experienced by individuals, nor were we able to factor in IPV victimization by a parent/guardian who was living in the home but not selected to participate in the survey. Additionally, findings may not generalize to all children, only children that were living with a parent at the time of the study. Finally, children's asthma was assessed by parental reports of a prior diagnosis of asthma. The use of self-reported questions regarding a child's asthma diagnosis, in contrast to physical measurements or a series of standardized questions assessing asthma and wheezing, is also a limitation and may have led to misclassification. Further, other factors such as recall bias or a respondent's access to health insurance may have negatively impacted the accuracy of this method of assessment.

Future studies could provide a more complete explanation of the findings by assessing a broader range of environmental exposures as well as assessing how IPV is associated with stress biomarkers (e.g., cortisol). The measurement of stress biomarkers could test the mediating step that is supposed by these findings. In addition, future studies might separately assess both the direct witnessing of IPV by children as well as IPV not witnessed by children, particularly IPV

victimization that may have occurred prior to a child's birth. It is likely that children are negatively affected by the direct witnessing of IPV, regardless of the sex of the parent who is victimized. However, the recent research indicating that maternal stress may uniquely influence the development of children's asthma supports the assessment and examination of maternal violence, both before and during pregnancy, as a potential separate contributing factor.

In sum, this study provides additional support for the far-reaching influence of IPV. The results highlight the importance of primary prevention of IPV, as reducing the occurrence of IPV could not only benefit the health of those who experience IPV but also the health of their children.

Disclaimer

The findings and conclusions in this report are those of the author(s) and do not necessarily represent the official position of the Centers for Disease Control and Prevention/the Agency for Toxic Substances and Disease Registry.

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