Correlates of Isolated Nocturnal Hypertension and Target Organ Damage in a Population-Based Cohort of African Americans: The Jackson Heart Study

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BACKGROUND
African Americans have higher rates of nocturnal hypertension and less nocturnal blood pressure (BP) dipping compared with whites. Although nocturnal hypertension is associated with increased cardiovascular morbidity and mortality, its clinical significance among those with normal daytime BP is unclear. This paper reports the prevalence and correlates of isolated nocturnal hypertension (INH) in a population-based cohort of African Americans enrolled in the Jackson Heart Study (JHS).

METHODS
The study sample included 425 untreated, normotensive and hypertensive JHS participants who underwent 24-hour ambulatory BP monitoring (ABPM), echocardiography, and 24-hour urine collection. Multiple logistic regression and 1-way analysis of variance models were used to test the hypothesis that those with INH have worse target organ damage reflected by greater left ventricular (LV) mass and proteinuria compared with normotensive participants.

RESULTS
Based on 24-hour ABP profiles, 19.1% of participants had INH. In age and sex-adjusted models, participants with INH had greater LV mass compared with those who were normotensive ($P = 0.02$), as well as about 3 times the odds of LV hypertrophy and proteinuria ($Ps < 0.10$). However, multivariable adjustment reduced the magnitude and statistical significance of each of these differences.

CONCLUSIONS
INH was associated with increased LV mass compared with normotension in a population-based cohort of African Americans enrolled in the JHS. There were trends toward a greater likelihood of LV hypertrophy and proteinuria among participants with INH vs. those who were normotensive. The clinical significance of the noted target organ damage should be explored in this population.

Keywords: ambulatory blood pressure monitoring; blood pressure; hypertension; Jackson Heart Study; nocturnal blood pressure; target organ damage.

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The use of ambulatory blood pressure monitoring (ABPM) has increased our ability to characterize the circadian pattern of blood pressure (BP) and different subtypes of hypertension, most notably of which is nighttime, or nocturnal, hypertension. Ample evidence links elevated nighttime BP to increased cardiovascular morbidity and mortality compared with daytime BP.[1–7] Among elderly hypertensive patients in the Sys Eur trial, nighttime BP was a better predictor of cardiovascular morbidity and mortality than daytime BP.[4] In the Ohasama population-based study, each 5% reduction in nocturnal BP decline was associated with up to 20% higher risk of cardiovascular mortality.[2] Similarly, a 9 mm Hg higher nighttime diastolic BP was associated with a 25% increased risk of congestive heart failure among elderly Swedish men.[3] Thus, there is no doubt about the clinical significance of nocturnal BP.[8]

Only recently has the clinical significance of nocturnal hypertension in patients with normal daytime BP been demonstrated.[9] The characteristics of patients with this previously unrecognized subtype of hypertension (elevated nighttime

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BP > 120/70 and normal daytime ambulatory BP < 135/85), termed isolated nocturnal hypertension (INH), were first described by Li *et al.* in a Chinese cohort.[10] Compared with normotensive patients, those with INH were older and had higher cholesterol and glucose levels. Although the authors demonstrated a positive relationship between INH and increased arterial stiffness, an intermediate marker of target organ damage, the prognostic significance of INH with regard to other well-established measures of target organ damage such as proteinuria and left ventricular (LV) mass is unknown. This is particularly important to ascertain in African Americans, who reportedly have higher nocturnal hypertension and less nocturnal BP dipping than whites[11,12] but who were not represented in the International Database on Ambulatory blood pressure in relation to Cardiovascular Outcomes.[9]

The Jackson Heart Study (JHS) is a population-based cardiovascular epidemiologic study in African Americans that includes 24-hour ambulatory BP assessments and extensive target organ evaluation. In this paper, we report the prevalence and clinical correlates of INH in this high-risk population. We also test the hypothesis that participants with INH have worse target organ damage reflected by greater LV mass and higher rates of proteinuria compared with normotensive participants.

METHODS

Study population

Participants were part of the JHS. The methods and overview of the JHS design have been described elsewhere.[13,14] Briefly, 5,301 noninstitutionalized African Americans adults with a mean age of 54.9 years (range: 21–94 years) participated in the study. Participants were invited to undergo 24-hour ABPM; those who agreed (N = 1,150) were fitted with a monitor during the scheduled baseline clinic examination. The group that completed ABPM was somewhat younger (53.2 vs. 57.1 years; $P < 0.01$) and included fewer females (61.6% vs. 67.9%; $P < 0.01$) than the group that did not (N = 4,151). With regard to the study outcomes, participants who completed ABPM had lower LV mass (147.3 vs. 157.1 g; $P < 0.01$) but did not differ significantly from the rest of the JHS cohort with regard to the prevalence of LV hypertrophy (7.6% vs. 8.3%; $P = 0.27$) or proteinuria (10.5% vs. 10.4%; $P = 0.52$).

Assessment of office and 24-hour ambulatory blood pressure

Operational definition of office BP was based on the average of 2 sitting BP measurements taken with a Hawksley random zero sphygmomanometer equipped with 1 of 4 cuff sizes selected by measured arm circumference. Participants were categorized as hypertensive if their systolic blood pressure was >140 mm Hg or diastolic blood pressure was >90 mm Hg. Measurements of 24-hour ABPM were obtained with a portable, noninvasive oscillometric device (Spacelabs 90207; Medifacts International Ltd, Rockville, MD). Trained technicians instructed participants in the proper use, application, and removal of the ABPM device. The monitor was programmed to take readings at 20-minute intervals throughout the 24-hour monitoring period, and participants were instructed to proceed through their normal daily activities and keep their arm still and extended at their side during each BP reading. Participants returned to the clinic 24 hours after attachment of the ABPM device for removal. The monitor was connected to a computer, and the 24-hour BP readings were downloaded with a commercially available software (Medicom, version 3.41; Medifacts Ltd). These analyses were based on participants with a minimum of 54 valid readings or 75% of the programmed readings during 24 hours of monitoring.

Definition of ambulatory BP subtypes

Daytime and nighttime BP readings were defined by participants' sleep diaries. Consistent with recommendations,[15] nocturnal hypertension was defined as nighttime systolic blood pressure >120 mm Hg or diastolic blood pressure >70 mm Hg, while normal awake ambulatory BP was defined as systolic blood pressure <135 mm Hg and diastolic blood pressure <85 mm Hg. Participants with nocturnal hypertension and normal awake BP were categorized as having INH, while those with normal nighttime BP and elevated awake BP were categorized as having isolated daytime hypertension. Participants with elevated awake and nighttime BP and those with normal awake and nighttime BP were categorized as daytime–nighttime hypertension and normotension (NT), respectively. Participants on antihypertensive medications were excluded from the analysis.

Echocardiography measurements

Echocardiograms were performed by certified ultrasonography technicians (Sonos 4500 echocardiograph; Hewlett Packard, Andover, MA) and following American Society of Echocardiography recommendations.[16] The 2D and M-mode examination was similar to typical clinical echocardiography with parasternal, apical and subcostal windows, and assessment of all 4 cardiac chambers. A single observer who was blinded to participants' clinical data read all measurements. The calculation of LV mass was based on the standard formula: LV mass (g) = 0.8×1.04 [(LV end diastolic diameter + IVST + PWT)^3 − (LV end diastolic diameter)^3] + 0.6, where IVST is interventricular septal thickness and PWT is posterior wall thickness. LV mass was divided by height$^{2.7}$ to calculate LV mass index; a standard cutoff of LV mass index ≥51 g/m^2 was used to define LV hypertrophy.[17]

Laboratory and self-report measurements

After an overnight fast, venous blood samples were collected from all participants to assess their blood glucose and lipid profile. Urine albumin, urinary sodium, and creatinine excretion were assessed with a 24-hour urine collection. Levels of urinary sodium were expressed in mmol/dl, while urinary sodium creatinine ratio and spot

urinary albumin creatinine ratio were expressed in mg/g. Proteinuria was defined as levels of urinary albumin creatinine ratio >30 mg/g. Participants were categorized as having diabetes if their fasting glucose was >126 mg/dl. Information on demographic data, smoking status, alcohol intake, physical activity, and medication use was assessed with validated self-report instruments.[14] All participants had their weight and height measured with standard scales, and their body mass index was derived from these measurements using the formula: weight, in kilograms, divided by height, in meters squared (kg/m^2).

Data analysis

The χ^2 tests and 1-way analysis of variance models were used to compare the 4 ABP subtype groups on a set of demographic and clinical variables (shown in Table 1). Variables that differed significantly overall ($P < 0.05$) were followed up with specific pairwise comparisons to test differences between the groups and identify correlates of INH. Multiple logistic regression models were used to test the hypothesis that participants with INH have a greater likelihood of LV hypertrophy and proteinuria than those with NT. A 1-way analysis of variance was used to test the

Table 1. Participant characteristics by ambulatory blood pressure subtype

Characteristic	Normotension (N = 176)	Isolated daytime hypertension (N = 16)	Isolated nocturnal hypertension (N = 81)	Day–night hypertension (N = 152)	*P* value
Women, %	66.5	37.5	61.7	58.6	0.10
Age, years	52.2 (10.8)[a]	51.6 (10.1)[a,b]	55.5 (11.6)[b]	55.9 (10.8)[b]	0.01
Body mass index, kg/m^2	30.2 (7.5)	28.4 (4.5)	30.2 (6.6)	29.4 (5.7)	0.49
Blood glucose, mg/dl	96.76 (26.96)	93.40 (9.85)	99.40 (24.23)	101.56 (29.21)	0.37
Diabetes, %)	9.8[a]	6.3[a,b]	19.0[a,b]	20.3[b]	0.03
Total cholesterol, mg/d	198.9 (36.4)[a]	217.7 (37.5) [a,b]	216.0 [b] (39.0)	201.4 [a] (36.6)	< 0.01
HDL cholesterol, mg/d	54.5 (14.3)	57.7 (12.9)	54.2 (15.0)	53.8 (13.0)	0.78
LDL cholesterol, mg/d	126.1 (34.5)[a]	143.9 (40.2)[b,c]	139.6 (33.0)[c]	126.9 (31.9)[a,b]	< 0.01
Metabolic syndrome, %	18.9[a]	33.3[a,b]	41.78[b]	41.4[b]	< 0.01
Lipid lowering therapy, %	3.2	0	0	5.7	0.16
Urinary sodium excretion, mmol/dl	148.5 (62.9)	152.1 (54.7)	162.3 (61.6)	160.9 (74.1)	0.39
Urinary sodium creatinine ratio, mg/g	103.2 (40.2)[a]	101.3 (37.2)[a,b]	116.6 (43.3)[a,b]	122.5 (68.1)[b]	0.02
Home and Yard Physical Activity Index	2.4 (0.7)	2.1 (0.6)	2.3 (0.7)	2.3 (0.6)	0.31
Current smoker, %	12.0	6.3	16.0	9.9	0.49
Current drinker, %	50.0[a,c]	81.3[b]	56.3%[a,b]	46.3[a]	0.04
Clinic SBP, mm Hg	117.1 (16.0)[a]	126.0 (23.7)[b,c]	124.1 (15.3)[b]	133.3 (16.3)[c]	< 0.01
Clinic DBP, mm Hg	74.3 (9.0)[a]	84.3 (13.7)[b]	75.7 (9.4)[a]	79.0 (12.1)[b]	< 0.01
Awake SBP, mm Hg	119.7 (7.2)[a]	132.7 (8.6)[b]	127.2 (5.9)[c]	143.4 (11.6)[d]	< 0.01
Awake DBP, mm Hg	73.9 (6.0)[a]	84.5 (5.8)[b]	76.5 (6.4)[c]	86.7 (8.4)[b]	< 0.01
Sleep SBP, mm Hg	107.9 (6.9)[a]	108.4 (5.7)[a]	123.6 (8.4)[b]	134.6 (14.5)[c]	< 0.01
Sleep DBP, mm Hg	61.7 (5.3)[a]	61.9 (5.7)[a]	71.3 (6.7)[b]	76.9 (9.2)[c]	< 0.01
Awake pulse pressure, mm Hg	45.8 (7.0)[a]	48.2 (11.7)[a,b]	50.7 (7.9)[b]	56.7 (12.5)[c]	< 0.01
Sleep pulse pressure, mm Hg	46.2 (6.2)[a]	46.5 (6.1)[a]	52.2 (9.7)[b]	57.7 (13.1)[c]	< 0.01
24-hour SBP, mm Hg	115.6 (6.7)[a]	124.3 (6.5)[b]	125.9 (5.7)[b]	140.3 (11.8)[c]	< 0.01
24-hour DBP, mm Hg	69.6 (5.5)[a]	77.0 (6.3)[b]	74.6 (6.0)[b]	83.2 (8.3)[c]	< 0.01
LV mass, g	136.2 (35.0)[a]	147.8 (37.7)[a,b,c]	152.5 (46.0)[b]	169.8 (59.3)[c]	< 0.01
LV hypertrophy, %	3.5[a]	6.7[a,b]	9.9[b]	18.4[b]	< 0.01
Proteinuria, %	2.9[a]	0.0[a,b]	9.0[a,b]	11.7[b]	0.03

Standard deviations are in parenthesis for continuous measures. For characteristics that differ significantly across groups, the groups that differ from each other are indicated by different superscripts.

Abbreviations: DBP, diastolic blood pressure; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LV, left ventrical; SBP, systolic blood pressure.

hypothesis that participants with INH have higher LV mass than those with NT. Following unadjusted analyses (model 1; Table 2), all models were adjusted for age and gender (model 2; Table 2) and then adjusted for age, gender, and the set of statistically significant clinical characteristics (model 3; Table 2). This included diabetes, total cholesterol, low-density lipoprotein cholesterol, metabolic syndrome, daytime pulse pressure, and alcohol use. Although nighttime pulse pressure was also significantly different, it was not included due to concerns about multicollinearity.

RESULTS

Participant characteristics by subtypes of ambulatory blood pressure

Of the 1,150 participants who underwent 24-hour ABPM, 327 were excluded due to lack of valid 24-hour ABPM readings (i.e., missing sleep BP or < 54 valid ABP readings overall) or missing sleep diary data, which are needed to improve the accuracy of ambulatory BP subtype classification. Of the remaining participants, 398 were excluded due to antihypertensive medication use. In the final study sample of 425 participants, the prevalence of each ambulatory BP subtype was as follows: 41.4% were normotensive, 19.1% had INH, 3.8% had isolated daytime hypertension, and 35.8% had sustained daytime–nighttime hypertension. The prevalence of INH was similar in the present sample of 425 and in the overall sample of 1,150 participants who underwent ABPM (19.1% and 20.0%, respectively).

Table 1 presents the characteristics of the final study sample categorized according to the 4 ambulatory BP subtypes. Overall, the clinical and demographic characteristics of the subtypes were generally similar. However, compared with those with NT, participants with INH were somewhat older, had a higher prevalence of metabolic syndrome, had higher total cholesterol and low-density lipoprotein cholesterol, had significantly higher daytime and nighttime pulse pressures.

Table 2. Left ventricular mass indices and proteinuria by ambulatory blood pressure subtype

Model	Normotension (N = 176)	Isolated daytime hypertension (N = 16)	Isolated nocturnal hypertension (N = 81)	Day–night hypertension (N = 152)
LV Mass (g); N = 416				
Model 1: Unadjusted	136.16 (3.58)	147.84 (12.16)	152.46 (5.23)	169.84 (3.88)
		P = 0.36	*P* = 0.01	*P* < 0.01
Model 2: Age and gender adjusted	137.42 (3.53)	142.26 (11.99)	152.32 (5.13)	169.00 (3.82)
		P = 0.70	*P* = 0.02	*P* < 0.01
Model 3: Multivariable adjusted[a]	136.32 (3.98)	139.03 (13.86)	147.54 (5.81)	162.33 (4.45)
		P = 0.85	*P* = 0.12	*P* < 0.01
LV Hypertrophy (LVMI ≥ 51 g/m2); N = 415				
Model 1: Unadjusted Prevalence	1.0 3.5%	1.98 (0.22, 17.59) 6.7%	3.03 (1.02, 9.05) 9.9%	6.23 (2.49, 15.55) 18.4%
		P = 0.54	*P* = 0.05	*P* < 0.01
Model 2: Age and gender adjusted	1.0	2.18 (0.24, 19.73)	2.89 (0.96, 8.69)	5.99 (2.38, 15.08)
		P = 0.49	*P* = 0.06	*P* < 0.01
Model 3: Multivariable adjusted[a]	1.0	[b]	2.58 (0.75, 8.94)	4.64 (1.60, 13.48)
			P = 0.13	*P* < 0.01
Proteinuria (UACR > 30 mmol/dl); N = 340				
Model 1: Unadjusted Prevalence	1.0 2.9%	[c]	3.34 (0.91, 12.28) 9.0%	4.49 (1.44, 14.04) 11.7%
			P = 0.07	*P* = 0.01
Model 2: Age and gender adjusted	1.0	[c]	3.29 (0.89, 12.19)	4.41 (1.40, 13.94)
			P = 0.08	*P* = 0.01
Model 3: Multivariable adjusted[a]	1.0	[c]	1.95 (0.46, 8.22)	2.66 (0.77, 9.19)
			P = 0.37	*P* = 0.12

Means and standard errors are shown for LV mass; odds ratios and 95% confidence intervals are shown for LV hypertrophy and proteinuria. Unadjusted prevalence rates for LV hypertrophy and proteinuria for each group are shown in the second line of the unadjusted models for these dependent variables. *P* values indicate significance level for contrast with normotension, 2-tailed test.

Abbreviations: LV, left ventricular; LVMI, left ventricular mass index; UCAR, urinary albumin creatinine ratio.

[a] Covariates: diabetes, total cholesterol, LDL cholesterol, metabolic syndrome, urinary sodium creatinine ratio, daytime pulse pressure, alcohol use.

[b] Isolated daytime hypertension dropped from analysis because there were no cases of LV hypertrophy in this group after removing cases with missing data for the covariate variables.

[c] Isolated daytime hypertension dropped from analysis because there were no cases of proteinuria in this group.

Other differences among the 4 groups are indicated in Table 1 using superscripts.

Left ventricular mass indices and proteinuria by subtypes of ABP

The unadjusted and adjusted multivariable models comparing the values of LV mass, LV hypertrophy, and proteinuria according to each subtype of ambulatory BP are shown in Table 2. Not surprisingly, there was an overall main effect of the ABP subtypes on each of the 3 outcomes in unadjusted and multivariable adjusted models (all *P*s < 0.05). Pairwise contrasts indicated that in unadjusted models, participants with INH had significantly greater LV mass than those with NT (152.5 g vs. 136.2 g) as well as significantly higher odds of LV hypertrophy (odds ratio = 3.03; 95% confidence interval, 1.02–9.05); they also had higher odds of proteinuria (odds ratio = 3.34; 95% confidence interval, 0.91–12.28), but this difference was only marginally statistically significant. Adjustment for age and gender diminished the differences in each outcome somewhat such that results for LV hypertrophy and proteinuria became marginally significant (*P*s < 0.10). Although the trends remained, differences between INH and NT in multivariable adjusted models were not statistically significant for any of the outcomes.

DISCUSSION

Among an untreated population-based sample of African Americans enrolled in the JHS, the prevalence of INH determined by ABPM was 19.1%. In age- and gender-adjusted models, participants with INH had significantly greater LV mass compared with those who were normotensive, as well as about 3 times the odds of LV hypertrophy and proteinuria, though these differences were only marginally statistically significant. Differences in each measure of target organ damage were diminished in multivariable models and were no longer statistically significant.

To our knowledge, this is the first study to document the prevalence and correlates of INH in a population-based sample of African Americans. The term "isolated nocturnal hypertension" was first introduced by Li *et al.*[10] in 2007; they reported a prevalence of 10.9% in a Chinese cohort of >600 participants. In this study, the authors found that INH was associated with increased arterial stiffness, and thus postulated it as a new subtype of ambulatory BP with clinical relevance. Later, Wijkman *et al.* examined correlates of INH in a clinic-based sample of 414 patients with diabetes.[18] Similar to results from Li *et al.,* the authors found a relationship between INH and arterial stiffness. They also examined LV mass index in a subset of patients but did not find an association with INH.

The following important differences between our study and previous studies should be noted. First, the prevalence of INH in our study sample is much higher (19%) than those reported in the International Ambulatory Blood Pressure Database for Chinese, South Africans, Japanese, and Eastern and Western Europeans (10.9%, 10.5%, 10.2%, 7.9%, and 6.0%, respectively).[10] This is not surprising given the well-documented evidence that African Americans have higher nocturnal BP and less nocturnal BP dipping compared with whites.[10,11] It is also possible that the high prevalence of INH is a reflection of the relatively higher fasting glucose levels in the JHS cohort compared with the Chinese[10] cohort (5.23 mml/L vs. 4.38 mml/L, respectively). There is evidence that patients with diabetes have high levels of nocturnal hypertension and less dipping compared with nondiabetic patients.[19,20]

Second, in contrast to the Wijkman *et al.*[18] study, participants with INH had greater LV mass compared with those with NT, although this difference was diminished and no longer statistically significant after multivariable adjustment. This might be explained by several important differences between the JHS population and the other population. First, patients taking antihypertensive medications were excluded from our analysis. Our rationale for restricting the analytic sample to untreated adults was 2-fold: this strategy enabled us to assess the true prevalence of INH without a confounding effect of treatment, given the known effect of antihypertensive medications on nighttime blood pressure,[21] and, there is a strong correlation between hypertension and nocturnal BP, with hypertensive patients experiencing less frequent dipping than nonhypertensive patients. This is in contrast to the Wijkman *et al.* study[18] in which treated and untreated participants were grouped together. Also, the definition of INH in the Wijkman *et al.* study was based on clinic BP as well as daytime ABP, which may have led to misclassification of patients with INH.

As suggested by Li *et al.*, the mechanism for INH may be attributed to higher rates of urinary sodium excretion noted in this population.[10] Such high levels may serve as an adaptive mechanism for pressure natriuresis and could explain the elevated nighttime BP in the group with INH compared with the other subtypes.[22] Often the high urinary sodium/creatinine ratio is a reflection of increased intake of nutritional sodium, which may be attributed to lifestyle or genetic factors. However, in our study sample, there was no significant difference in urinary sodium/creatinine ratio in participants with INH compared with the group with NT. Furthermore, while we did not collect this data, there is some evidence to suggest that nocturnal hypertension is strongly related to poor sleep quality, and this may actually mediate the differences in dipping status between African Americans and whites.[23] Thus INH may be secondary to poor sleep quality regardless of race or ethnic background, and this is especially true for African Americans given the higher prevalence and severity of obstructive sleep apnea noted in this population.[24] Similarly, the higher body mass index noted in our study sample compared with that of the Chinese cohort in Li *et al.* (29.8 kg/m^2 vs. 22.2 kg/m^2, respectively) may also contribute to poor sleep quality with subsequent elevated nighttime BP.

We should note the following limitations of this study. First, while the JHS is a large, population-based study, only 21.7% of participants underwent ABPM and many of those were on antihypertensive medications. Thus, the resulting sample of 425 participants may have provided insufficient statistical power to detect differences between INH and NT. In fact, we observed suggestive trends supporting our hypotheses, with odds ratios for LV hypertrophy and proteinuria ranging from 1.95 to 3.29 in the adjusted models.

Second, as is true for other studies, the short-term reproducibility of INH is unknown. Because participants completed only 1 ABPM, this question could not be addressed in the present study. Third, we did not assess central blood pressure and other markers of arterial stiffness in our study sample, thus making it impossible to speculate about the relationship between INH and this important intermediate measure of target organ damage in the JHS cohort. Thus, we could not make any meaningful comparison between our study sample and those of previous studies based on these parameters. Finally, as previously noted, it is not clear to us that clinical variables alone account for INH in this study sample, given the well-established relationship between poor sleep quality and nocturnal hypertension.[22] Future studies should investigate the correlates of isolated nocturnal hypertension while adjusting for sleep quality.

In summary, we found a substantially higher prevalence of INH in this population-based sample of African Americans than has been reported in previous studies of other racial/ethnic groups. However, our hypothesis that participants with INH would have greater target organ damage than those with NT was only partly supported. Although the differences in LV hypertrophy and proteinuria fell short of statistical significance in adjusted models, there were trends in the expected direction. Future longitudinal analyses of the JHS participants will reveal whether the differences in target organ damage we observed are maintained or change over time and will help to determine whether or not INH is an important risk factor in African Americans.

DISCLOSURE

The authors have no conflicts of interest to declare.

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