Linking discrimination and sleep with biomarker profiles: An investigation in the MIDUS study

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ABSTRACT

Self-reported experiences of discrimination and sleep dysfunction have both been shown to adversely impact biological functioning; however, few studies have examined how they are jointly associated with health. The current study draws from two samples of the Midlife in the United States (MIDUS) data (n = 617 participants; 59.8% female; 72.3% White and 27.7% African American; Age: Mean = 52.6, SD = 12.22) to identify profiles of sleep (duration, variability, onset latency, wake after sleep onset, naps) and discrimination (everyday, lifetime, impact). Associations with latent profiles of biomarkers of inflammation (CRP, fibrinogen, IL-6) and endocrine stress (cortisol, epinephrine, norepinephrine) were examined. Three profiles were identified for sleep/discrimination (*good, fair, poor*) and for biomarkers (*average, high inflammation, high neuroendocrine*). Chi-square analyses indicated that adults in the *good sleep/low discrimination* profile were more likely to be in the *average biomarker* profile but less likely to be in the *high inflammation* profile. Adults in the *fair sleep/moderate discrimination risk* profile were more likely to be in the *high inflammation* profile. Adults in the *poor sleep/high discrimination risk* profile were less likely to be in the *average biomarker* profile but more likely to be in the *high inflammation* profile. The current study identified configurations of sleep and discrimination among midlife adults which were associated with profiles of biological risk. The findings provide implications for identifying individuals who may be at increased risk of developing stress-related tertiary outcomes of morbidity and disease.

1. Introduction

Sleep is a universal biological necessity that is associated with health and functioning. Sleep serves a restorative purpose, allowing humans to process the day's experiences and adequately prepare for the next. Interfering with the restorative benefits of sleep has detrimental health consequences. Sleep disturbances are linked to a host of short- and longer-term health outcomes including compromised cognitive functioning, academic and occupational outcomes and daytime sleepiness [1]. Sleep duration and quality are influenced by social and environmental contexts [2]; structural and socioeconomic contextual factors such as neighborhood and housing conditions, and individual-level proximal influences such as stress [3]. Systematic differences in

area-level contexts have been linked to ethnic/racial disparities in sleep, and help to explain why non-White individuals suffer from more sleep disturbances compared to White individuals, including shorter sleep duration and poorer sleep quality [4,5]. Interpersonal experiences of discrimination are also a source of stress that have been linked to sleep [6–9].

In light of the discrimination-sleep link, there is also increasing research on how discrimination and sleep jointly impact health. Drawing upon the theoretical framework of the race-based disparities in stress and sleep in context model [10], the current study investigates how psychological and biological responses to discrimination intersect, and are implicated in health outcomes and disparities. Existing research has focused on how the impact of discrimination stress may be exacerbated

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(i.e., moderated) by high levels of sleep disturbance (e.g., short sleep duration, poor sleep quality) or buffered when coupled with low levels of sleep disturbance (e.g., good sleep quality). High levels of discrimination coupled with poor sleep quality has been found to be especially harmful for health and academic outcomes [11,12]. At the same time, good sleep quality has been observed to buffer the impact of discrimination stress on health [11,12]. However, to date, studies have taken a variable-centered approach to investigating the joint impact of sleep and discrimination using traditional regression methods. In fact, while many scholars have implicated discrimination stress a source of sleep disturbances, recent work has found the association may be more reciprocal and birdirectional [5,8]. The current study advances this science by offering a novel application of how common profiles, combinations or typologies of sleep and discrimination are jointly implicated in health. Regression methods introduce methodological and analytical assumptions related to an arbitrarily predetermined association between the variables; for example, whether discrimination stress or sleep is selected as the independent variable or the moderator (i.e., does sleep moderate the effects of discrimination on health, or does discrimination moderate the effects of sleep on health?). The current study contributes to research on discrimination and sleep disturbance by deriving data-driven, person-centered, and bottom-up typologies, thus enabling the identification of *existing* configurations of sleep and discrimination in a group of people (e.g., those with similar levels of sleep quality and discrimination) [13], rather than presuming a priori associations. This approach simultaneously considers multiple combinations of discrimination and sleep, as well as multiple sleep dimensions to derive unique profiles of their combination (rather than unidimensional indicators of low, moderate, and high levels). Discrimination and multiple dimensions of sleep (e.g., duration, quality, variability) may interact with one another to influence health in complex ways. Given the complexity and multiple possible combinations of sleep, discrimination and health, a profile approach to analysis offers a more parsimonious analytical approach to consider multiple variables simultaneously [14]. The resultant profiles are then examined in relation to biomarker profiles to investigate how sleep and discrimination relate to distinct risk and protective biomarker profiles.

1.1. Sleep and discrimination

Discrimination, defined as differential treatment, particularly that which is considered to be unfair, is commonly considered to be a source of psychosocial stress [15,16]; and stress is associated with sleep disturbances [17]. There is a growing evidence base linking discrimination stress to sleep disturbances [8,18–20]. A recent systematic review of 17 studies investigating discrimination stress and sleep found a significant association among all of the studies in the synthesis [19]. Among diverse youth in Australia, direct and vicarious reports of discrimination were associated with shorter self-reported sleep duration, longer sleep onset latency, and more sleep disruption [21]. Yet, the research linking actigraphy-recorded and detailed assessments of sleep and discrimination is new and the nature of their association is only starting to emerge [22]. A cross-sectional study of African American and White adults found that discrimination was associated with poorer sleep quality assessed with polysomnography and wrist actigraphy [23]. A daily diary study of discrimination and sleep found that while discrimination was associated with shorter sleep duration, counterintuitively, it was associated with better sleep quality among African American adolescents (wake after sleep onset) [9]. Researchers interpreted this effect as a possible indication of how discrimination may be linked to psychological and physical exhaustion, reducing wake minutes after falling asleep, and resulting in "better" sleep quality metrics. Research with Latinx adolescents has focused on sleep variability and found that discrimination was associated with greater night-to-night variability in self-reported sleep duration [24]. Finally, research using wrist actigraphy suggests that better sleep quality assists adolescents' ability to cope with subsequent discrimination on a daily basis [25]. Building off of this research, the current study

extends research to an adult sample and further, investigates how discrimination-related stress and the restorative function of sleep are collectively associated with biological markers.

The current research underscores the value of investigating how various combinations of sleep and discrimination may be differentially associated with biological health indices. The research also makes clear the importance of focusing on both sleep quality, duration, and variability. Although duration has been the primary focus of sleep research, sleep quality and variability are important correlates of health [26]. Existing research is limited in its investigation of associations between sleep and discrimination and health indices; and the current study investigates the joint impact of sleep and discrimination on health in mid-to later adulthood on biological health indicators. This study focuses on underlying inflammation and endocrine markers of stress (epinephrine, norepinephrine, and cortisol) [27], which have been linked to clinical risk indicators, including those related to cardiometabolic disease [28]. This study examines how sleep/discrimination profiles are associated with profiles of biological dysregulation, as indicated by CRP, fibrinogen, IL-6, cortisol, epinephrine, and norepinephrine. We use "sleep and discrimination" to refer to the constructs or indicators, and "sleep/discrimination" to refer to the latent profiles.

2. Material and methods

2.1. Participants and procedures

Data were drawn from two samples from the Midlife in the United States (MIDUS) study, a longitudinal investigation of health and well-being among middle-aged adults [29]. Informed consent was obtained for all participants. The first sample was drawn from the second wave (MIDUS 2; M2), including a 10-year follow-up of 4963 (70%) participants from the original MIDUS baseline cohort (MIDUS 1) in 2004–2009 and a new sample of 592 African Americans from Milwaukee (total $n = 5555$). Higher retention rates were found among White, female, and married participants and those with better health and more education [30]. The second sample was drawn from the MIDUS Refresher (MR) study, designed to replenish the MIDUS 1 sample and contained the same comprehensive assessments as M2. The MR study recruited a national probability sample of 3577 adults and a sample of 508 African Americans from Milwaukee (total $n = 4085$) in 2011–2014. Both M2 and MR surveyed all participants on demographic, psychosocial, and health information, and collected biomarker and sleep data from subsamples. Following previous research, the current study combined the discrimination, sleep, and biomarkers variables from the M2 and MR samples [31]. Compared to the M2 sample, the MR sample was younger (t (8857) = -10.31, $p < .001$), had fewer female (χ^2 (1) = 7.48, $p < .01$) and White participants (χ^2 (1) = 99.01, $p < .001$), higher education (t (8842) = 10.93, $p < .001$), smaller household size (t (8856) = -12.36, $p < .001$), higher household income (t (6650) = 8.04, $p < .001$), fewer wake minutes after sleep onset (t (717) = -2.24, $p < .05$), higher lifetime discrimination (t (6694) = 4.73, $p < .001$) and everyday discrimination (t (6854) = 2.56, $p < .001$), lower serum interleukin-6 (t (2093) = -2.09, $p < .05$), and cortisol (t (2100) = 15.11, $p < .001$), epinephrine (t (2069) = 10.01, $p < .001$), and norepinephrine (t (2103) = 3.29, $p < .01$). The two samples did not differ on any other demographic or primary analytic variables. To address potential cohort differences, all key variables were standardized within each sample and sample membership was covaried using a dichotomous sample designator (M2 vs. MR).

The M2 and MR survey data were collected through phone interviews and self-administered questionnaires. Subsamples from the primary (M2: $n = 1054$; MR: $n = 746$) and Milwaukee samples (M2: $n = 201$; MR: $n = 117$) participated in the biomarker data collection (M2: $n = 1255$; MR: $n = 863$). Biomarker participants visited one of the three General Clinical Research Centers (i.e., UCLA, University of Wisconsin-Madison, Georgetown University) for an overnight visit, completed a medical history questionnaire, self-reported sleep assessments, and a blood draw.

Participants who visited University of Wisconsin-Madison (M2: $n = 533$; MR: $n = 334$) were provided with wrist actigraphs (Actiwatch® 64; Mini Mitter, Bend, OR, USA, now Philips Respironics) to collect objective sleep assessments for 7 consecutive days, while concurrently completing a paper and pencil Daily Sleep Diary [32].

The final analytic sample includes 617 (of 867) participants who visited the University of Wisconsin-Madison (Fig. 1). The majority of participants were White (58.9%) or African American (35.2%); for the sake of parsimony, participants from less well-represented ethnic/racial groups were excluded from the current analyses ($n = 55$; 10 Latinx, 15 Native American, 7 Asian, 19 other, 4 missing). To screen for obstructive sleep apnea, participants who reported that they had trouble sleeping because they "could not breathe comfortably" and "coughed and snored" more than once per week during the past month were excluded (n = 23 [33]). Participants were also excluded if they did not have objective sleep assessments ($n = 125$), had more than 3 days of invalid sleep data ($n = 13$) or had more than 2 missing values for discrimination indicators ($n = 6$).

Comparing the analytic sample with the participants excluded from the sleep sample ($n = 250$), the analytic sample had less in variability in sleep duration ($t (716) = -2.35, p < .05$), fewer wake minutes after sleep onset (WASO) ($t (717) = -5.00, p < .001$), shorter nap duration ($t (736) = -2.40, p < .05$), lower levels of lifetime discrimination ($t (853) = -3.78, p < .001$), impact of discrimination ($t (781) = -5.80, p < .001$), CRP ($t (849) = -2.49, p < .05$), fibrinogen ($t (850) = -3.66, p < .001$), IL-6 ($t (857) = -3.28, p < .01$), and higher levels of cortisol ($t (855) = 2.06, p < .05$). The analytic sample was older ($t (865) = 4.34, p < .001$), more likely to be White ($\chi^2 (1) = 161.01, p < .001$) employed ($\chi^2 (1) = 7.04, p < .01$), and had higher levels of education ($t (864) = 6.77, p < .001$) and household income ($t (849) = 2.93, p < .01$).

2.2. Measures

For all primary study variables pertaining to sleep, discrimination, and health, scores were standardized within each sample. Correlations, descriptive statistics, and reliabilities for measures are reported in Table 1. Statistics were based on the combined sample unless otherwise specified.

2.2.1. Sleep indicators

Both actigraphy and diary data were cleaned by MIDUS researchers to identify instances where these two sources of data yielded discrepant results. Because sleep researchers use daily diaries to corroborate actigraphy data, to further exclude outlying data, discrepancies between actigraphy and self-reported daily sleep duration that were greater than 3 standard deviations were coded as missing ($n = 64$ days). Of the possible maximum of 7 days, participants had data for an average of 6.79 days ($SD = 0.61$), and all participants had at least 4 days of valid data. Daily sleep indicators were averaged across 7 days to obtain a weekly estimate, and values that were greater than 3 SD were winsorized ($n = 5$ for sleep duration, $n = 7$ for sleep duration variability, $n = 7$ for sleep onset latency, $n = 7$ for wake after sleep onset, $n = 11$ for nap duration). We conducted analyses to investigate whether there were racial differences in the subset of extreme values (n = 64) and there were no significant differences. Therefore, we do not have reason to believe that removing extreme values contributed in the data.

Sleep duration was the actigraphy-recorded sleep time in minutes from sleep onset to offset.

Sleep duration variability was the mean squared successive difference (MSSD) of actigraphy-recorded sleep duration, that incorporates both day-to-day variability and temporal dependency in time series designs [34]. The squared difference between sleep duration on consecutive days (e.g., [sleep duration of day 1 – sleep duration of day 2]^2) were averaged.

Sleep onset latency was actigraphy-recorded duration from bedtime to sleep onset in minutes.

Wake after sleep onset (WASO) was actigraphy-recorded wake minutes between sleep onset and sleep offset.

Nap duration was self-reported in minutes in the sleep diaries. On days in which participants did not report taking a nap, duration was coded as 0.
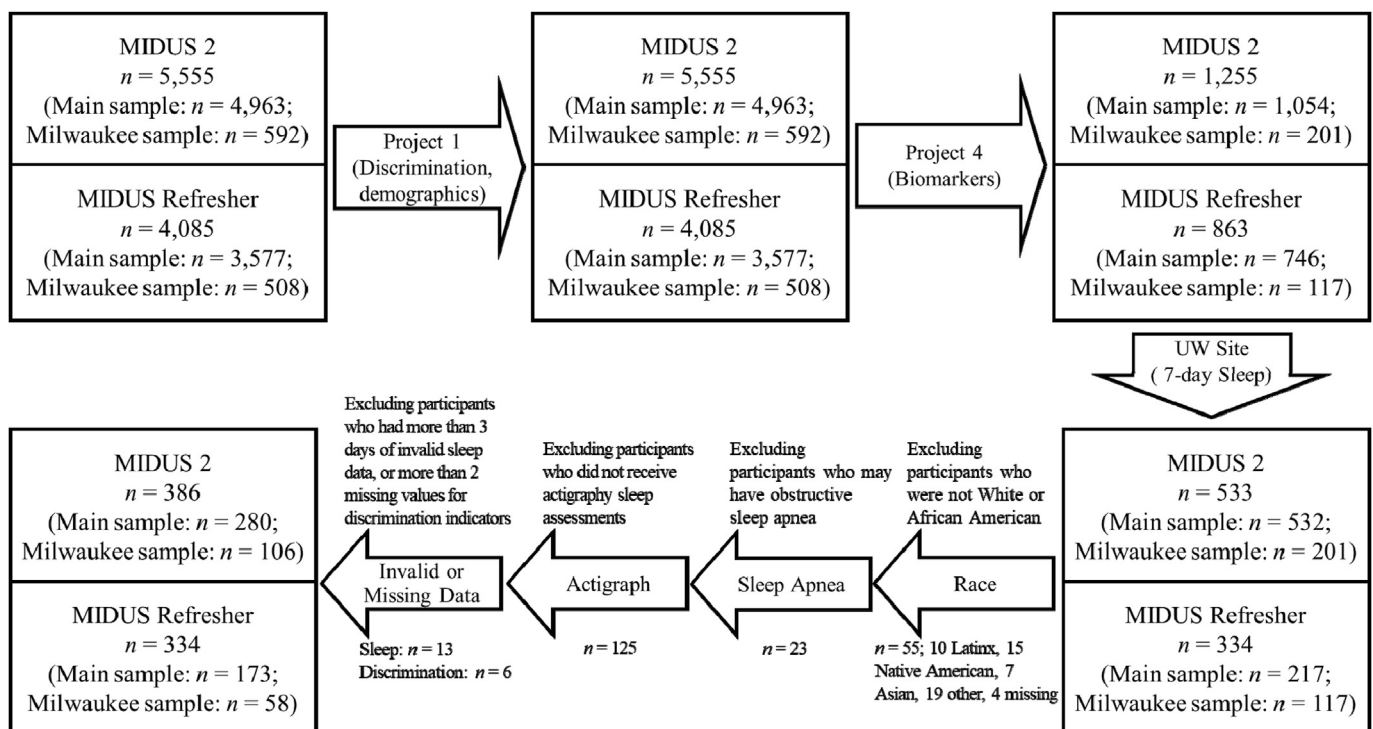

Fig. 1. Sample selection flow chart.

Table 1
Correlations and descriptive statistics for primary study variables.

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Sleep duration															
2. Sleep duration variability	-.09*														
3. Sleep duration variability (square root)[1]	-.10*	.96***													
4. Sleep onset latency	-.16**	.31***	.34***												
5. Wake after sleep onset	.28***	.32***	.31***	.32***											
6. Nap duration	-.20***	.26***	.25***	.11**	.14**										
7. Lifetime discrimination	-.13**	.08	.09*	.15**	.13**	.14***									
8. Everyday discrimination	-.01	.08*	.09*	.07	.08	.16***	.42***								
9. Impact of discrimination	-.16**	.22***	.20***	.25***	.23***	.17***	.59***	.41***							
10. C-reactive protein (CRP)	-.02	.14***	.14***	.20***	.10*	.10*	.14**	.06	.18***						
11. Fibrinogen	-.04	.09*	.10*	.17***	.04	.10*	.08	.00	.13**	.49***					
12. Serum interleukin-6 (IL-6)	-.04	.06	.06	.13**	.11**	.17**	.08*	.02	.11*	.50***	.39***				
13. Cortisol	-.03	.00	.00	.00	-.04	-.06	-.04	.06	-.01	-.04	-.10*	-.11**			
14. Epinephrine	-.07	.04	.05	.14***	-.02	.01	-.01	-.01	-.01	-.03	.00	.05	.22***		
15. Norepinephrine	-.02	-.01	-.01	.14***	.07	.05	.01	.05	.03	.01	.08	.15***	.22***	.30***	
Sample size	617	617	617	617	617	612	613	617	559	610	610	615	611	604	617
Number of items	7	6	6	7	7	7	11	9	2	1	1	1	1	1	1
Reliability (Cronbach's α)[2]	.79	.49	.60	.76	.82	.72	.74	.91	.90	n/a	n/a	n/a	n/a	n/a	n/a
Mean	419.31	12375.20	97.99	28.24	45.63	14.27	1.19	2.74	1.37	3.02	345.95	2.97	1.45	.24	2.16
SD	67.89	13674.59	52.70	25.08	23.06	20.66	1.91	3.03	.74	3.63	79.70	2.35	1.04	.41	1.88
Min	209.02	73.45	8.57	.21	6.50	.00	.00	.00	1.00	.05	15.00	.16	.02	.00	.10
Max	633.04	75950.94	275.59	129.16	135.97	92.30	11.00	9.00	4.00	18.50	612.47	12.18	7.19	4.19	19.54

Note. [1] Sleep duration variability (square root) was not used for data analysis, but is reported here to provide an interpretable metric. [2] Sleep indicators' Cronbach's α's were calculated across multiple days of data. *p < .05. **p < .01. ***p < .001.

2.2.2. Discrimination indicators

Three measures of discrimination were assessed in the self-administered questionnaires.

Lifetime discrimination was assessed with 11 items asking how many times participants experienced discriminatory events (e.g., "You were discouraged by a teacher or advisor from seeking higher education.") along with an attribution "because of such things as your race, ethnicity, gender, age, religion, physical appearance, sexual orientation, or other characteristics" [35]. Items were dummy coded as 0 (*none*) and 1 (*once or more*) and summed, with higher scores indicating more lifetime discrimination.

Everyday discrimination was assessed with 9 items rating the frequency of unfair interpersonal interactions (e.g., "You are treated with less courtesy than other people") using a scale from 1 (*often*) to 4 (*never*) without an attribution [36]. Due to low frequencies, items were dichotomized (0 = *never*, 1 = *rarely, sometimes, or often*) and summed, with higher scores indicating more everyday discrimination. Sensitivity analyses combining *never* and *rarely, sometimes* = 0 compared to *often* = 1 were conducted and the raw scores yielded identical results.

Discrimination impact was assessed with 2 items (e.g., "how much harder has your life been because of discrimination?") using a scale from 1 (*a lot*) to 4 (*not at all*). Since the two items were correlated at $r = 0.83$, if the participants reported no discrimination experiences (lifetime or everyday), scores were coded as 4 (*not at all*) for both items. Items were reverse coded, ranging from 1 (*not at all*) to 4 (*a lot*), and averaged such that higher scores indicate more impact.

2.2.3. Biomarkers

The Biomarker Project included a 12-hr urine specimen and fasting blood specimen collected by clinic nursing staff. All biomarker levels were quantified in duplicate, and values were determined by standard procedures (see Love et al., 2010). C-reactive protein (CRP), fibrinogen, and serum interleukin-6 (IL-6) were assessed by blood specimen and are considered to be physiological indicators of inflammation. Cortisol, epinephrine, and norepinephrine were assessed by urine specimen and are considered to be endocrine stress indicators. These indicators were selected as markers of underlying inflammation and endocrine functioning and the most proximate downstream measures to sleep. Values greater than 3 *SD*s were winsorized ($n = 12$ for CRP, $n = 4$ for fibrinogen, $n = 13$ for IL-6, $n = 12$ for cortisol, $n = 10$ for epinephrine, $n = 11$ for norepinephrine) and were standardized for each sample.

C-reactive protein (CRP) was assessed by a particle enhanced immunonephelometric assay using the BNII nephelometer (N Antiserum to Human Fibrinogen, Dade Behring Inc., Deerfield, IL, USA). The inter-assay coefficient of variance (CV) was between 2.1 and 5.7% for M2 and 1.1–4.3% for MR, and the intra-assay CV was between 2.3 and 4.4% for M2 and MR.

Fibrinogen was also measured by the BNII nephelometer. The inter-assay CV was 2.6% for M2 and 4.13–6.64% for M2, and the intra-assay CV was 2.7% for M2 and MR.

Serum interleukin-6 (IL-6) was assessed by high-sensitivity enzyme-linked immunosorbent assay (ELISA; Quantikine, R&D Systems, Minneapolis, MN, USA). Values larger than 10 pg/mL were rerun in diluted sera to fall on the standard curve. The inter-assay CV was 12.31% for M2 and 15.66% for MR, and the intra-assay CV was 3.25% for M2 and 3.73% for MR.

Cortisol levels were assessed by Enzymatic Colorimetric Assay and Liquid Chromatography-Tandem Mass Spectrometry (LC-MS/MS), and the inter-assay CV was 6.1% for M2 and 8.08–15.49% for MR.

Epinephrine levels were assessed by High-Pressure Liquid Chromatography (HPLC). The inter-assay CV was between 7.8 and 7.9% for M2 and 12.59–18.61% for MR, and the intra-assay CV was 8% for M2.

Norepinephrine levels were also assessed by HPLC, and the inter-assay CV was between 6.7 and 6.9% for M2 and 10.43–16.77% for MR, and the intra-assay CV was 8% for M2.

2.2.4. Covariates

Gender (1 = *female*, 0 = *male*), age, race (1 = *White*, 0 = *African American*), education (12-point scale ranging from "no school/some grade school" to "PhD, MD, JD, or other professional degree"), employment status (1 = working or self-employed, 0 = looking for work or unemployed, temporarily laid off, retired, homemaker, full-time student, part-time student, maternity or sick leave, permanently disabled, or other), household size (sum of number children and other household members), household income (total annual income from wage, pension, social security, and other), and sample (1 = M2, 0 = MR) were included as covariates.

2.3. Analysis plan

We first explored distinct patterns of sleep and discrimination and biomarker patterns using latent profile analysis (LPA [13]) in M*plus* 8[37]. LPA is a person-centered approach that uses continuous indicators to derive unique subgroups of individuals. For each set of LPAs, the indicators were standardized, and a series of models were fitted sequentially estimating one to six profiles. The optimal solution was chosen based on multiple fit indices [38], including Bayesian information criterion (BIC), the sample size adjusted BIC (ABIC), entropy, and a log-likelihood-based test (i.e., Lo-Mendel-Rubin test), as well as the conceptual meanings and interpretability of the profiles. Missing data were handled by full information maximum likelihood (FIML), an approach that uses all the available information to provide a maximum likelihood estimation [39].

We examined the sleep/discrimination profiles using LPAs for respondents with actigraphy data ($n = 617$). Because biomarker data were also in the full biomarker sample ($n = 2118$), we conducted separate LPAs comparing the analytic to the full sample to investigate the stability and replicability of the profiles. LPA profiles from the two samples were compared using chi-square tests. Significant chi-square tests (i.e., agreement between the full and analytic samples) were interpreted to indicate stable and replicable profiles. If the full and analytic sample profiles were consistent, we used the analytic sample profiles in subsequent analyses. To further validate the profiles and to identify potential influences of covariates (e.g., gender, age, race, education, etc…), we conducted LPAs with covariates and compared the profiles to the original LPA results using chi-square tests. If the profile membership was stable including and excluding covariates, we used the profiles without covariates in subsequent analyses. To investigate the demographic distribution across profiles, chi-square analyses were conducted by gender, age, race, education, and employment status in SPSS 25.0 (SPSS, Chicago, IL, USA). The latter two sets of analyses involving covariates were conducted for both sleep/discrimination and biomarker profiles.

To investigate the primary research aim exploring associations between sleep/discrimination profiles with biomarker profiles, chi-square analyses were conducted in SPSS 25.0. An omnibus chi-square test examined the extent to which the membership in sleep/discrimination profiles was associated with the membership in biomarker profiles. Cramér's *V* provides an estimate of effect size, ranging from 0 to 1. If the omnibus chi-square test was significant (i.e., indicating an association between sleep/discrimination with biomarker profiles), two sets of post-hoc tests were conducted to identify the specific location of the significant differences [40]. Cells with the absolute values of standardized residuals or adjusted residuals larger than 2.0 were interpreted to indicate that individuals with a certain sleep/discrimination profile were more likely to also belong to a biomarker profile [41]. Next, *z* tests compared cells in a row with their column percentages [42], indicating the likelihood that individuals of a particular biomarker profile were also likely to be members of a specific sleep/discrimination profile relative to other sleep/discrimination profiles.

3. Results

3.1. Descriptive statistics

The final analytic sample was comprised of 617 participants (59.8% female; 72.3% White and 27.7% African American) with an average age of 52.61 ($SD = 12.22$). The modal education level was college graduation ($n = 133$). Within the final analytic sample, there are 386 M2 participants and 231 MR participants. MR participants were younger (t (615) = -4.20, $p < .001$), more educated (t (614) = 2.17, $p < .05$). They also had fewer minutes of wake after sleep onset (t (615) = -2.62, $p < .01$), higher levels of cortisol (t (609) = 9.61, $p < .001$), higher levels of epinephrine (t (602) = 6.22, $p < .001$). MR and M2 participants did not differ in any other demographic or primary variables.

3.2. Sleep/discrimination profiles

The three-profile solution was identified as optimal, based on multiple model fit indices and class interpretations [37]. BIC and ABIC values decreased from the one-profile solution to the six-profile solution, with steeper decreases (>300) from the one-profile solution to the three-profile solution compared to decreases (<200) from the three-profile solution to the six-profile solution, indicating improvements in model fit. The LMR tests for the two- and three-profile solution were significant ($p < .05$), indicating a better fit to the data compared with the previous solution. The entropy values were all above 0.80, indicating desirable classifications. The proportion of individuals belonging to each class (<5%) declined dramatically after the three-profile solution [43] and the three-profile solution yielded more conceptually meaningful classes compared to the two-profile solution. Therefore, the three-profile solution was selected. Adding covariates did not change the classifications (χ^2 (4) = 1078.15, $p < .001$, Cramér's $V = 0.96$) and 100.0%, 98.1%, and 100.0% of the three profiles were stable including and excluding covariates. Therefore, results from the LPA model without covariates are reported here.

The resultant sleep/discrimination profiles are depicted in Fig. 2. The majority of participants (80%, $n = 494$) were characterized by the *good sleep/low discrimination* profile. To facilitate the interpretation of this profile relative to the full sample, this profile had an average daily sleep duration of 424.97 min compared to the overall sample average of

419.31 min (Supplemental Table-1). The second group (12%, $n = 73$) had below-average levels of sleep duration, and above-average levels of sleep disturbance (i.e., sleep duration variability, sleep onset latency, wake after sleep onset, nap duration) and discrimination and was labeled *fair sleep/moderate discrimination*. To facilitate the interpretation of this profile relative to the full sample, this profile had an average daily sleep duration of 413.15 min compared to the overall sample average of 419.31 min. The third and smallest group (8%, $n = 50$) had similar, but more extreme patterns with below-average levels of sleep duration, high levels of sleep disturbance, and even higher levels of discrimination. This third profile was labeled *poor sleep/high discrimination*. To facilitate the interpretation of this profile relative to the full sample, this profile had an average daily sleep duration of 397.24 min compared to the overall sample average of 419.31 min. Chi-square tests linking covariates to profile membership found significant associations with gender (Table 2, χ^2 (2) = 5.99, $p = .05$, Cramér's $V = 0.10$), race (χ^2 (2) = 109.30, $p < .001$, Cramér's $V = 0.42$), and education (χ^2 (2) = 8.85, $p < .05$, Cramér's $V = 0.12$). Women were less likely to be in the *good sleep/low discrimination* profile and more likely to be in the *fair sleep/moderate discrimination* profile. White participants were more likely to be in the *good sleep/low discrimination* profile and less likely to be in the *fair sleep/moderate discrimination* or *poor sleep/high discrimination* profiles compared to African Americans. Participants who graduated college or higher were more likely to be in the *good sleep/low discrimination* profile and less likely to be in the *poor sleep/high discrimination* profile.

3.3. Biomarker profiles

Analyses of the biomarker indicators suggested an optimal three-profile solution. BIC and ABIC values decreased from the one-profile solution to the six-profile solution, with steeper decreases (>400) from the one-to the three-profile solution compared to decreases (<200) from the three-profile solution to the six-profile solution, indicating improvements in model fit. LMR tests for the two- and three-profile solutions were significant ($p < .05$), indicating a better fit to the data when comparing each model with the previous model. The entropy values were all above 0.80, indicating desirable classifications and the proportion of individuals belonging to each class (<5%) declined after the three-profile solution. Therefore, the three-profile solution offered a better fit to the
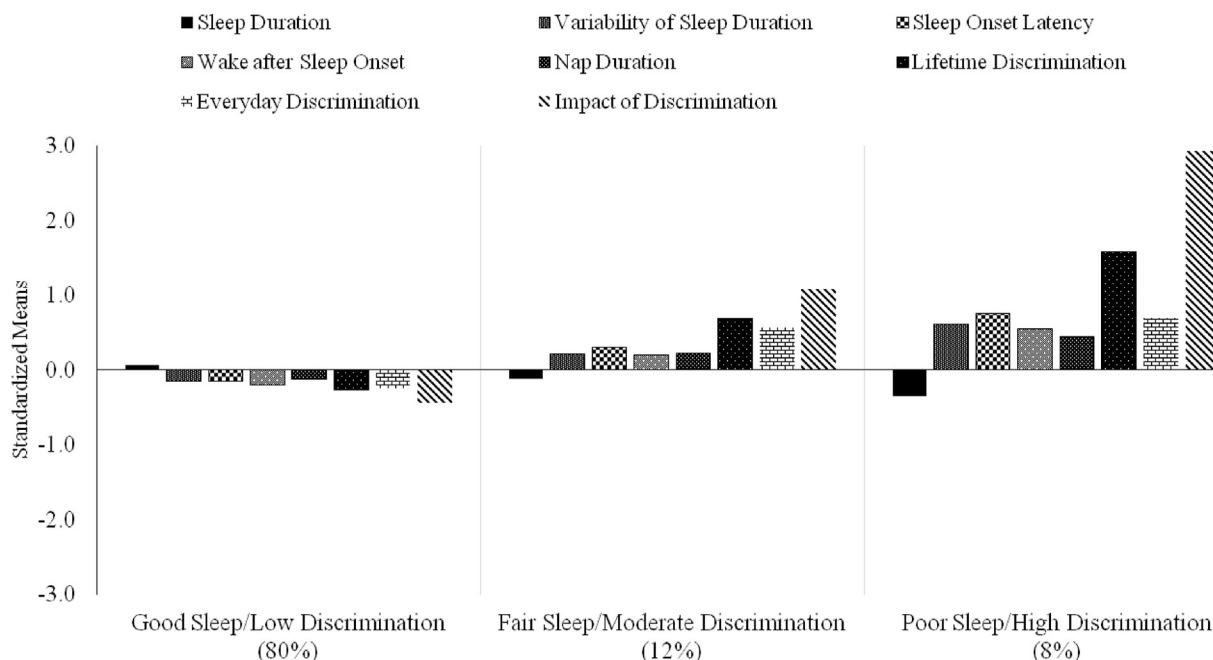

Fig. 2. Sleep/discrimination profiles.

Table 2

Chi-square analyses of latent class profiles and demographics.

	Gender		Age		Race		Education		Employment	
	Female	Male	50 or younger	Above 50	White	African American	Some college or lower	College graduation or higher	Unemployed	Employed
Sleep/discrimination profiles	$\chi^2 (2) = 5.99, p = .05$		$\chi^2 (2) = 4.09, p = .13$		$\chi^2 (2) = 109.30, p < .001$		$\chi^2 (2) = 8.85, p < .05$		$\chi^2 (2) = 2.54, p = .28$	
Good Sleep/Low Discrimination										
Number of observations	285	209	214	280	401	93	268	226	159	327
Column %	77.2[a]	84.3[b]	78.1[a]	81.6[a]	89.9[a]	54.4[b]	77.0[a]	84.3[b]	78.7[a]	82.2[a]
Fair Sleep/Moderate Discrimination										
Number of observations	53	20	31	42	35	38	42	30	23	46
Column %	14.4[a]	8.1[b]	11.3[a]	12.2[a]	7.8[a]	22.2[b]	12.1[a]	11.2[a]	11.4[a]	11.6[a]
Poor Sleep/High Discrimination										
Number of observations	31	19	29	21	10	40	38	12	20	25
Column %	8.4[a]	7.7[a]	10.6[a]	6.6[b]	2.2[a]	23.4[b]	10.9[a]	4.5[b]	9.9[a]	6.3[a]
Biomarker profiles	$\chi^2 (2) = 7.97, p < .05$		$\chi^2 (2) = 5.61, p = .06$		$\chi^2 (2) = 34.28, p < .001$		$\chi^2 (2) = 3.87, p = .15$		$\chi^2 (2) = 10.21, p < .01$	
Average Biomarker										
Number of observations	303	212	232	283	395	120	282	233	155	345
Column %	82.1[a]	85.5[a]	84.7[a]	82.5[a]	88.6[a]	70.2[b]	81.0[a]	86.9[a]	76.7[a]	86.7[b]
High Inflammation										
Number of observations	42	13	17	38	23	32	35	19	23	30
Column %	11.4[a]	5.2[b]	6.2[a]	11.1[b]	5.2[a]	18.7[b]	10.1[a]	7.1[a]	11.4[a]	7.5[a]
High Neuroendocrine										
Number of observations	24	23	25	22	28	19	31	16	24	23
Column %	6.5[a]	9.3[b]	9.1[a]	6.4[a]	6.3[a]	11.1[b]	8.9[a]	6.0[a]	11.9[a]	5.8[b]

Note. [a], [b] denotes a category of a certain covariate whose column percentage did not differ significantly from another category with the same superscript in the same row. Cells that share the same superscript (e.g., [a], [a]; [b], [b]) are not significantly different from each other. Cells with different superscripts ([a], [b]) are significantly different from each other.

data and yielded the most conceptually meaningful classes. Adding covariates did not change the classifications ($\chi^2 (4) = 952.32, p < .001$, Cramér's $V = 0.90$) and 97.6%, 88.2%, and 100.0% of the three profiles remained stable across the two models. Results from the LPA model without covariates are reported here.

As a stability check, LPA models were also conducted with the larger biomarker sample ($n = 2118$). All model indices indicated that the three-profile solution remained optimal. There was significant convergence between the three-profile solution with the biomarker sample compared to the final analytic sample ($\chi^2 (4) = 1114.64, p < .001$, Cramér's $V =$
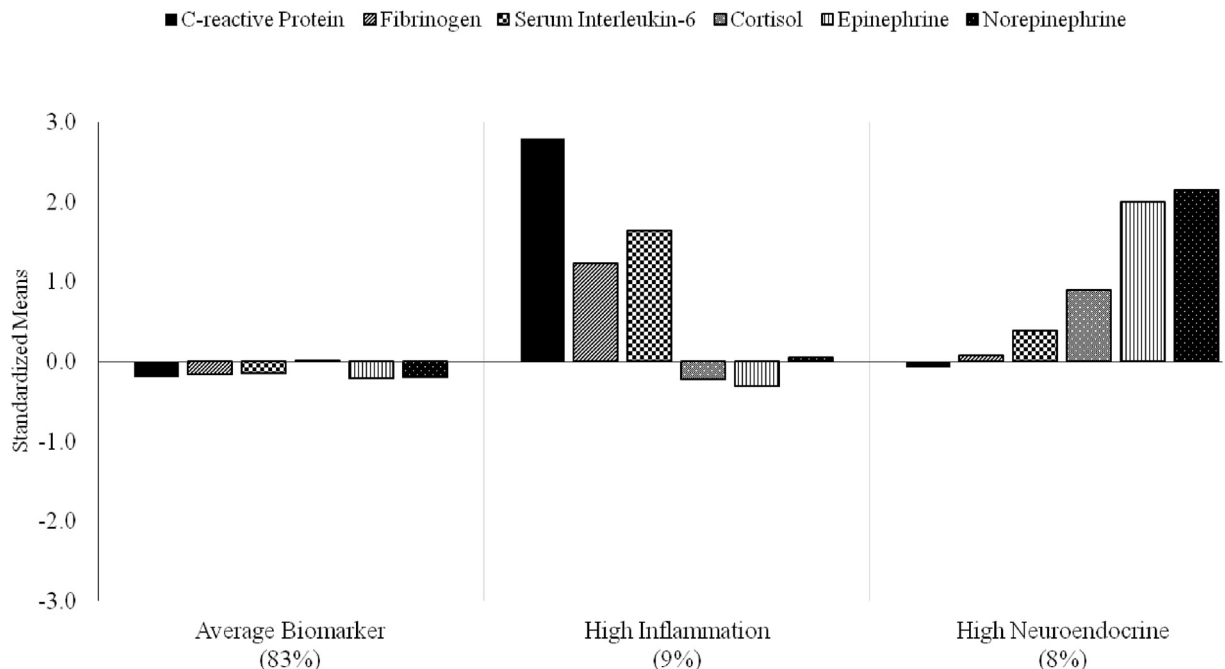

■ C-reactive Protein ▨ Fibrinogen ⊠ Serum Interleukin-6 ▤ Cortisol ▥ Epinephrine ■ Norepinephrine

Fig. 3. Biomarker profile.

0.95), with 95.7%, 98.7%, and 100.0% of the three profiles remaining stable. Adding gender, age, race, and education as covariates did not change the classifications (χ^2 (4) = 3791.78, $p < .001$, Cramér's V = 0.98) and 98.7%, 99.6%, and 98.6% of the three profiles were stable including and excluding covariates and results without covariates are reported here.

The resultant biomarker profiles are depicted in Fig. 3. The majority of the participants (83%; $n = 515$) had slightly below-average values or close-to-average values for the 6 biomarkers; this profile was labeled *average biomarker*. To facilitate the interpretation of this profile relative to the full sample, this profile had an average C-reactive protein value of 2.68 μg/mL compared to the overall sample average of 3.02 μg/mL, and an average norepinephrine value of 1.82 μg/dL compared to the overall sample average of 2.16 μg/mL (Supplemental Table-2). The second group (9%; $n = 55$) had above-average levels of CRP, fibrinogen, and IL-6, indicating high levels of inflammatory responses; this profile was labeled *high inflammation*. To facilitate the interpretation of this profile relative to the full sample, this profile had an average C-reactive protein value of 12.20 μg/mL compared to the overall sample average of 3.02 μg/mL. The third and smallest group (8%; $n = 47$) had above-average levels of cortisol, epinephrine, and norepinephrine, indicating high levels of endocrine stress markers; this profile was labeled *high neuroendocrine*. To facilitate the interpretation of this profile relative to the full sample, this profile had an average norepinephrine value of 6.02 μg/dL compared to the overall sample average of 2.16 μg/mL. Chi-square tests linking covariates to profile membership (Table 2) revealed demographic differences by gender (χ^2 (2) = 7.97, $p < .05$, Cramér's V = 0.11), race (χ^2 (2) = 34.28, $p < .001$, Cramér's V = 0.24), and employment status (χ^2 (2) = 10.21, $p < .01$, Cramér's V = 0.13). Women were more likely to be in the *high inflammation* profile but less likely to be in the *high neuroendocrine* profile. White participants were more likely to be in the *average biomarker* profile and less likely to be in the *high inflammation* or *high neuroendocrine* profiles compared to African Americans. Participants who were employed were more likely to be in the *average biomarker* profile and less likely to be in the *high neuroendocrine* profile.

3.4. Associations between sleep/discrimination profiles and biomarker profiles

To address the primary study aims, a chi-square test examined the association between the sleep/discrimination profiles and the biomarker profiles (Table 3). The omnibus test suggested that membership in the sleep/discrimination profiles was significantly associated with membership in the biomarker profiles with a small to medium effect size (χ^2 (4) = 21.94, $p < .001$, Cramér's V = 0.13). Standardized and adjusted residual values with absolute values larger than 2.0 for each cell suggest significant differences (Table 3). Looking first at residual values in each cell, participants in the *good sleep/low discrimination* profile (column 1) were more likely to be in the *average biomarker* profile (adjusted residual = 4.0, column 1, row 1) but less likely to be in the *high inflammation* profile (adjusted residual = −4.3, column 1, row 2). Participants in the *fair sleep/moderate discrimination* profile (column 2) were more likely to be in the *high inflammation* profile (adjusted residual = 2.4, column 2, row 2). Participants in the *poor sleep/high discrimination* profile (column 3) were less likely to be in the *average biomarker* profile (adjusted residual = −3.5, column 3, row 1) but more likely to be in the *high inflammation* profile (adjusted residual = 3.4, column 3, row 2).

Building upon the primary chi-square analyses that suggest an association between membership in a sleep/discrimination profile and biomarker profiles, z tests investigated the distribution of sleep/discrimination profiles across biomarker profiles. While the chi-square analyses compare the likelihood of participants from the sleep/discrimination profiles belonging to specific biomarker profiles, the z tests compare the likelihood of participants from a biomarker profile having membership in sleep/discrimination profiles. Because SPSS 25.0 does not report z values, group differences were reported using superscripts (i.e., [a]

Table 3
Chi-square analyses of sleep/discrimination and biomarker profiles.

	Sleep & discrimination profiles		
	Good Sleep/Low Discrimination	Fair Sleep/Moderate Discrimination	Poor Sleep/High Discrimination
*Biomarker profiles**			
Average Biomarker			
Number of observations	427	55	33
Expected frequencies	412.3	60.9	41.7
Likelihood[1]	103.6%	90.3%	79.1%
Column %	86.4[a]	75.3[b]	66.0[b]
Residuals	14.7	−5.9	−8.7
Standardized residuals	.7	-.8	−1.4
Adjusted residuals	4.0	−2.0	−3.5
High Inflammation			
Number of observations	32	12	11
Expected frequencies	44.0	6.5	4.5
Likelihood	72.7%	184.6%	244.4%
Column %	6.5[a]	16.4[b]	22.0[b]
Residuals	−12.0	5.5	6.5
Standardized residuals	−1.8	2.2	3.1
Adjusted residuals	−4.3	2.4	3.4
High Neuroendocrine			
Number of observations	35	6	6
Expected frequencies	37.6	5.6	3.8
Likelihood	93.1%	107.1%	157.9%
Column %	7.1[a]	8.2[a]	12.0[a]
Residuals	−2.6	.4	2.2
Standardized residuals	-.4	.2	1.1
Adjusted residuals	−1.0	.2	1.2

Note. *χ^2(4) = 21.94, $p < .001$. [1] Likelihood was calculated based on the number of observations and expected frequencies. [a, b] denotes a sleep/discrimination profile whose column percentage did not differ significantly from another sleep/discrimination profile with the same superscript in the same row. Cells that share the same superscript (e.g., [a, a]; [b, b]) are not significantly different from each other. Cells with different superscripts ([a, b]) are significantly different from each other. E.g., the column percentage for the *good sleep/low discrimination* profile corresponding to the *average biomarker* profile row (86.4%) was significantly different from the column percentage for the *fair sleep/moderate discrimination* (75.3%) and *poor sleep/high discrimination* (66.0%) profiles. Descriptive statistics of profile membership likelihood showed that for individuals having in the *average biomarker* profile, the likelihood of being in the *good sleep/low discrimination* profile was 103.6%; thus, there were 3.6% more participants in this group compared to chance level (expected frequencies); in contrast, participants were less likely to be in the *fair sleep/moderate discrimination* (100%–90.3% = 9.7%) and *poor sleep/high discrimination* (100% - 79/1% = 20.9%) profiles compared to chance.

and [b], Table 3). The z test showed that the *average biomarker* profile (row 1) had significantly different column percentages, indicating that participants in the *average biomarker* profile were 3.6% more likely to be in the *good sleep/low discrimination* profile (calculated by the number of observations and expected frequencies; see Table 3 notes for example), 9.7% less likely to be in the *fair sleep/moderate discrimination* profile, and 20.9% less likely to be in the *high sleep/discrimination* profile than expected by chance (i.e., the null hypothesis). For the *high inflammation* profile (row 2), participants were 6.9% less likely to be in the *good sleep/low discrimination* profile, 84.6% more likely to be in the *fair sleep/moderate discrimination* profile, and 144.4% more likely to be in the *poor sleep/high discrimination* profile. For the *high neuroendocrine* profile (row

3), there was no differences in participants' likelihood of being in one sleep/discrimination profile over another.

To test for cohort effects, the chi-square analyses with the combined MR and M2 samples were compared to sensitivity analyses conducted only with the M2 sample and results suggest the same pattern of results.

4. Discussion

The current study extends existing research on how sleep and discrimination are conjointly associated with biomarker profiles. Taking a person-centered approach, profiles of sleep/discrimination, and biomarkers of health were derived with support for three distinct sleep/discrimination and biomarker profiles. Consistent with research on health disparities, there were demographic differences in profiles by gender, race, education, and employment status. Importantly, these sleep/discrimination profiles were also associated with biomarker risk profiles.

The sleep/discrimination profiles contribute to a growing science focused on how these constructs are related. Complementing existing research finding a positive association between sleep and discrimination, the profiles evidence a dose-response. While the moderate and high sleep/discrimination profiles had similar patterns, the levels of the variable combinations were notably different with elevated levels of sleep disturbance and discrimination in the high profile. Moreover, these elevated levels were associated with a higher likelihood of being in the high inflammation profile (144% for the high vs. 84% moderate sleep/discrimination profile). Taken together, the current study finds that the combination and levels of sleep and discrimination are implicated in biological health indicators.

4.1. Sleep/discrimination profiles

Three combinations of sleep and discrimination emerged, presented in order from lowest to highest risk: 1) *good sleep/low discrimination* which constituted 80% of the sample, and was more likely to include male (compared to female), White (compared to African American), and more educated adults, 2) *fair sleep/moderate discrimination* which constituted 12% of the sample, and was more likely to include female (compared to male) and African American (compared to White) adults, and (3) *high-risk sleep/discrimination* which constituted 8% of the sample and was more likely to include African American (compared to White) and less educated adults. There were significant differences across all of the sleep/discrimination profiles by race, with White adults more likely to be in the lowest risk profile (i.e., *good sleep/low discrimination*) compared to the two less healthy profile combinations of sleep and discrimination (i.e., *fair sleep/moderate discrimination*). This observation is consistent with research citing health disparities by race, particularly between White and African American individuals [4,44]. Across the profiles, the moderate and high-risk profiles exhibited above-average levels on all indicators of sleep quality, below-average levels of the protective indicator of sleep duration, and higher levels of discrimination indicators. The current analyses also find that nap duration was highest in the fair and poor sleep/discrimination profiles, suggesting that napping behavior may either be an indicator or consequence of poor sleep hygiene [45]. There may be a trade-off between night-time sleep and naps suggesting a compensatory association. Non-conventional or irregular work schedules (e.g., shift work) or unemployment may explain this association [46]; however there was no evidence of employment differences in the current analyses. It is also possible that disrupted night-time sleep necessitates daytime napping to arrive at sufficient sleep duration.

The emergence of the three sleep/discrimination profiles lend support to research suggesting a link between high levels of discrimination and poor sleep quality among adults [47]. In the current study, the *good sleep/low discrimination* profile reported average levels of sleep duration, quality, and variability in sleep duration, and slightly below-average levels of discrimination impact. The two higher risk profiles displayed a positive

association between poor sleep quality and discrimination, which was particularly evident for the *poor sleep/high discrimination* profile where sleep disturbance and the three indicators of discrimination had the highest levels of all the profiles. Taken together, the profile combinations suggest a positive association between discrimination-related stress and various indicators of poor sleep quality. As further support, the data-driven profiles did not identify a combination of good sleep (i.e., high quality and consistent sleep patterns) and high levels of discrimination, or poor sleep (i.e., low quality and inconsistent sleep patterns) and low levels of discrimination. Although causal explanations are beyond the scope of the study, these findings implicate a positive association between sleep disturbance and discrimination, consistent with work identifying social bases of health disparities [48]. The results also contribute to possible cumulative associations between sleep and discrimination where repeated exposure to discrimination may result in longer-term sleep disruptions over time. For example, chronic everyday discrimination has been found to be associated with subjective sleep complaints and polysomnography-assessed sleep quality [49]. The current study contributes to the previous literature that links discrimination and sleep [11,12] by demonstrating the ways in which discrimination and sleep emerge jointly among White and African American adults.

4.2. Biomarker profiles

Three biomarker profiles were evident. The first and largest profile was the *average biomarker* profile which included 83% of the sample, was more likely to include White (compared to African American) and employed participants. The second profile, *high inflammation*, included 9% of the sample and included more female (compared to male) and African American (compared to White) participants. The third profile, *high neuroendocrine,* comprising 8% of the sample included more male, African American, and unemployed participants. As with the sleep/discrimination profiles, African Americans were disproportionately represented in the least healthy profiles (i.e., *high inflammation, high neuroendocrine*), underscoring health disparities [50]. The *high inflammation* profile was disproportionately female, whereas the *high neuroendocrine* profile was disproportionately male, pointing to possible intersections of race and sex as contributing to health vulnerabilities [51]. While high levels of cortisol play an indirect role in stress-related disease development, high levels of inflammation are particularly troubling. For example, inflammation is indicative of risk for various chronic inflammatory diseases, such as such as rheumatoid arthritis, cardiovascular diseases [52], diabetes [53] and cognitive impairment [54].

Investigating associations within the profiles, it appears that inflammation (e.g. IL-6) was inversely related to neuroendocrine stress markers (e.g., cortisol [55]), although there was marginal evidence of an association between IL-6 and neuroendocrine stress markers at the bivariate level. IL-6 can have pro- and anti-inflammatory properties [56], which may explain this data pattern. Cortisol functions as an anti-inflammatory hormone and keeps inflammation under control [57]. Since persistent stress can induce long-term changes in the HPA axis, including hypercortisolism [57], inflammatory responses may be uncontrolled and display exaggerated elevations in response to stress which could eventually lead to low-grade chronic elevation in inflammation.

4.3. Associations between sleep/discrimination and biomarker profiles

The primary aim of the study was to investigate how membership in a sleep/discrimination profile was associated with biomarker profiles. Adults in the *good sleep/low discrimination* profile were more likely to be in the *average biomarker* profile, and less likely to be in the *high inflammation* profile. There was a positive association between the sleep/discrimination risk profile and the probability of being in the high inflammation profile, with those in the *fair sleep/moderate discrimination* profile (elevated levels of sleep disturbance and discrimination) being more likely to be in the *high inflammation profile* compared to those with

average levels of sleep/discrimination; and those in the *poor sleep/high discrimination* profile having the highest probability of being in the high inflammation profile. These findings contribute to evidence that sleep and discrimination have joint effects on health and disease risk as indicated by biomarker profiles.

5. Conclusions

This study contributes to the growing science of ethnic/racial health disparities in sleep, discrimination and health [4,5,58,59]. White respondents were more likely to be in healthier "good" and "fair" sleep/-discrimination profiles compared to African Americans; these profiles in turn were associated with healthier biomarker profiles. White (compared to African American) adults were more likely to be in the *good sleep/low discrimination* profile than the *poor sleep/high discrimination* group, and in the *average biomarker* than the *high neuroendocrine* profiles. These data suggest that African Americans have increased risk exposure across multiple health indicators, including sleep, discrimination, and biomarker levels. Disadvantage and risk across multiple domains are detrimental to health and development [60]. These results call for more studies to understand the intersectionality of disadvantage and health disparities [61].

There are several noteworthy caveats. First, due to sample characteristics, analyses of other ethnic/racial groups were not sufficiently investigated; extending analyses to other ethnic/racial groups should be considered important forthcoming research. The *poor sleep/high discrimination* profile only constituted 8% of the sample, raising questions about whether this same set of sleep/discrimination profiles will replicate with larger or smaller samples. Given sleep, discrimination, and health change with age, it is unclear whether these same profiles would replicate in a younger or older sample. The cross-sectional data preclude examination of causal or temporal pathway between discrimination, sleep, and health, and research suggests that these constructs are likely to be reciprocally associated [62]. Profiles were derived at the person-level where daily sleep indicators were aggregated across several days in order to arrive at sleep/discrimination profiles, leaving open the question of how sleep and discrimination are associated on a daily basis and whether this association varies over time. It is also unclear whether certain individual indicators of sleep (i.e., duration, quality, variability) are more closely related to discrimination and health indices. Although not a specific focus of the analyses, it is noteworthy that women were more likely to be represented in the moderate sleep/discrimination and high inflammation profiles which contributes to research on this topic [63–66]. Finally, the MIDUS data include a national sample comprised primarily of White respondents, with African American respondents less well represented; and most of the African American respondents were recruited from the Milwaukee site, limiting generalizability. However, the analyses presented here have undergone several statistical tests to investigate possible cohort and/or sample effects, and the results remain robust; however, differences are still possible. At the same time, Milwaukee residents experience high levels of segregation; thus, the sample likely generalizes to African American residents in segregated urban areas.

Despite these limitations, the current study contributes to a growing area of research exploring linkages between sleep, discrimination, and health. The application of a person-centered, profile approach facilitated investigation of how unique configurations of sleep and discrimination are associated with biological profiles of health risk. The findings also provide implications for identifying individuals who may be at increased risk of developing stress-related clinical disease outcomes as well as mortality. In terms of practical significance, a careful review of related research finds clear evidence from other studies showing that: (1) discrimination is associated with elevated CRP [67], (2) sleep is associated with elevated inflammation [1], and (3) chronic low-grade inflammation is associated with poor health outcomes [68]. Based on these converging lines of evidence, and informed by a population health perspective (e.g., Rose's prevention paradox [69]), even small shifts in

the distribution of a health metric (e.g., inflammation) can be expected to have meaningful impacts on the distribution of health in a population.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.cpnec.2020.100021.

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