# UCLA
## UCLA Previously Published Works

**Title**

A sexual risk and stress reduction intervention designed for HIV-positive bisexual African American men with childhood sexual abuse histories.

**Permalink**

https://escholarship.org/uc/item/3dk3v266

**Journal**

American Journal of Public Health, 103(8)

**ISSN**

0090-0036

**Authors**

Williams, John K
Glover, Dorie A
Wyatt, Gail E
et al.

**Publication Date**

2013-08-01

**DOI**

10.2105/ajph.2012.301121

Peer reviewed

# A Sexual Risk and Stress Reduction Intervention Designed for HIV-Positive Bisexual African American Men With Childhood Sexual Abuse Histories

John K. Williams, MD, Dorie A. Glover, PhD, Gail E. Wyatt, PhD, Kimberly Kisler, MPH, Honghu Liu, PhD, and Muyu Zhang, MS

HIV/AIDS continues to disproportionately affect African Americans relative to other racial/ethnic groups.[1,2] In 2009, for example, African Americans represented only 14% of the US population but accounted for 44% of all new HIV diagnoses.[1] Male-to-male sexual contact is the greatest category of risk among Blacks, accounting for 73% of new infections among Black men.[1] HIV infection rates are higher among Black men who have sex with men (MSM) than in any other racial/ethnic MSM group.[1,2] Despite this disparity, few interventions have been designed specifically for African American MSM[3–6] or for men who have sex with men and women (MSMW).

Prevention strategies that emphasize HIV education and access to condoms may be inadequate in effecting sexual risk reduction in these populations. Preexisting risk factors and mediational mechanisms may operate differently among African American MSMW, who may also be less likely to respond to interventions that are developed for gay men[3,7–9] or to those that are not contextualized for African American experiences. Intervention strategies that focus on less commonly examined mediators may influence sexual behavior changes and HIV transmission and serve as new modalities for addressing the HIV epidemic among African American MSMW.

Individual behavior change is complicated by personal, environmental, historical, and institutional factors.[10] Trauma exposure, a social determinant operating at multiple ecological levels, may be contributing to the HIV epidemic. Childhood sexual abuse (CSA) is a significant predictor of generalized emotional distress, anxiety, and posttraumatic stress disorder (PTSD) in adulthood,[11–15] and it has also been associated with an increased risk of sexual revictimization.[16–22] Experiencing CSA not only affects people's health, but it may also have an impact on their interpersonal

*Objectives.* HIV transmission risk is high among men who have sex with men and women (MSMW), and it is further heightened by a history of childhood sexual abuse (CSA) and current traumatic stress or depression. Yet, traumatic stress is rarely addressed in HIV interventions. We tested a stress-focused sexual risk reduction intervention for African American MSMW with CSA histories.

*Methods.* This randomized controlled trial compared a stress-focused sexual risk reduction intervention with a general health promotion intervention. Sexual risk behaviors, psychological symptoms, stress biomarkers (urinary cortisol and catecholamines), and neopterin (an indicator of HIV progression) were assessed at baseline and at 3- and 6-month follow-ups.

*Results.* Both interventions decreased and sustained reductions in sexual risk and psychological symptoms. The stress-focused intervention was more efficacious than the general health promotion intervention in decreasing unprotected anal insertive sex and reducing depression symptoms. Despite randomization, baseline group differences in CSA severity, psychological symptoms, and biomarkers were found and linked to subsequent intervention outcomes.

*Conclusions.* Although interventions designed specifically for HIV-positive African American MSMW can lead to improvements in health outcomes, future research is needed to examine factors that influence intervention effects. (*Am J Public Health.* 2013;103:1476–1484. doi:10.2105/AJPH.2012.301121)

relationships in adulthood (e.g., how they select and interact with their intimate sexual partners). Societal and environmental factors such as the stigma associated with sexual abuse may also contribute to negative psychosocial health outcomes.[23]

Acute traumatic and chronic stress is also known to disrupt neurobiological mechanisms essential for survival. Preclinical research demonstrates that all vertebrates share a similar threat system.[24] Stimuli identified as potentially challenging to the basic state of equilibrium (i.e., stressors) trigger immediate release of catecholamines and subsequent release of glucocorticoids (e.g., cortisol). These regulatory systems have feedback loops that ensure a return to homeostasis when the threat subsides. However, when chronic or excessive demands are placed on these systems, long-term changes occur in the areas of growth, reproduction, and immune activity.[24] These

well-known effects are embodied in the allostatic load model,[25] which reflects the cumulative burden of stress disruptions on primary mediators such as cortisol and catecholamines. Over time, primary mediator disruptions compromise efficient and effective responses to new demands, secondary downstream mediators (e.g., blood pressure and body mass index) are dysregulated, and ultimately an increased risk of tertiary disease outcomes results.[26,27]

Major life events, including trauma and abuse experiences, and common but chronic challenges such as problems at work and home or relationship issues may all contribute to cumulative stress burdens.[28] Among members of ethnic minority groups, racially discriminatory experiences may also be perceived as threatening and precipitate a stress response.[29–31] Such chronic stress is thought to contribute to health disparities in racial/ethnic minority groups in a framework referred to

as the weathering hypothesis.[32,33] In the case of African Americans, racial, sociocultural, and political inequities, as well as trauma experiences such as CSA, may contribute to increased disease risk.

Associations between CSA and increased HIV sexual risk behaviors[34–38] and mental health symptoms have been reported among men with abuse histories.[38,39] However, these relationships are complex. For example, PTSD with co-occurring depression has been shown to act as both a moderating and a mediating variable in the relationship between CSA and number of lifetime sexual partners.[38,39] One recent study suggested that the associations of sexual risk behaviors with CSA trauma and with mental health symptoms were influenced by trauma and symptom severity.[40] Severe CSA (e.g., forcible penetration over multiple incidents) and an increased number of PTSD symptoms were linked to more sexual risk behaviors among HIV-infected African American men. Also, mixed linear regression models showed that clinically meaningful levels of PTSD symptoms predicted a composite indicator of primary neurohormones (cortisol, norepinephrine, epinephrine, and dopamine) reflecting physical health risks.

Although researchers have attempted to identify racial/ethnic differences in risk behaviors to explain disproportionate HIV rates,[41] characteristics of trauma and subsequent mental and physical health stress burden have not been adequately examined. Research exploring whether stress reduction intervention components can reduce sexual risk behaviors and improve mental and physical health is still in its infancy. Evidence from HIV interventions links social cognitive models emphasizing relaxation skills, cognitive coping strategies, and social support to the mediation of mood effects and stress-related neurohormones.[42–45] Decreased urinary cortisol levels with corresponding decreases in depressive symptoms and decreased urinary norepinephrine levels with reduced anxiety symptoms have been reported.[42,46]

Two meta-analyses showed that cognitive–behavioral interventions decreased psychological symptoms such as depression, anxiety, anger, and stress among people living with HIV/AIDS.[43,44] Unfortunately, immune functioning, as evidenced by CD4[43,44] and viral load,[43] showed little improvement. A study involving a 15-session individual stress management intervention for people with HIV reported a decreased frequency of unprotected sexual acts but no effect on depression and anxiety symptoms.[47] Another intervention, employing cognitive and stress management strategies with people who had HIV/AIDS and a history of CSA, reported decreases in both traumatic stress symptoms[48] and unprotected sex acts.[49] Comprehensive interventions designed to reduce sexual risk behaviors, psychological difficulties, and stress-related neurohormones are lacking.

We conducted a small, randomized clinical trial to develop and test the Enhanced Sexual Health Intervention for Men (ES-HIM) that targeted HIV-positive African American MSMW who did not self-identify as gay and who had histories of CSA. We compared ES-HIM with an attention-matched health promotion intervention (HP) with respect to their efficacy in reducing sexual risk behaviors (i.e., unprotected anal and vaginal sex), numbers of sexual partners, psychological symptoms of PTSD and depression, and primary neurohormonal mediators (cortisol and catecholamines). We also explored the intervention's effects on neopterin, which is produced primarily in monocytes and macrophages. Because neopterin is responsive to immune-inflammatory stimuli, it can serve as an indicator of HIV disease progression (i.e., deterioration of one's health status as a result of stress).

## METHODS

The ES-HIM project was a 4-year study conducted from 2007 to 2011 to develop and test an HIV risk and stress reduction intervention. Fliers, print advertisements, and face-to-face strategies were used to recruit a community sample of HIV-positive African American MSMW from HIV and other service agencies in Los Angeles County. Prospective participants were screened and were deemed to be eligible for the study if they were African American, male, at least 18 years of age, English speaking, and HIV positive; if they did not self-identify as gay; if they had engaged in unprotected anal or vaginal sex (or both) with a male as well as a female partner in the preceding 90 days; and

if they had a history of CSA. Individuals who had experienced any unwanted or forced sexual contact (ranging from touching and fondling to intercourse) or had had sexual experiences with someone at least 5 years older when they were less than 18 years of age were defined as having a CSA history.

Once they had provided informed consent, eligible participants were administered a 90-minute questionnaire, via a laptop computer and audio computer-assisted self-interview technology, designed to gather data on sociodemographic characteristics, trauma experiences, sexual behaviors, and PTSD and depression symptoms. Audio computer-assisted self-interviewing was used to increase confidentiality and comfort among participants during disclosure of sensitive information such as sexual and drug use behaviors.

A total of 295 men were screened, and 117 met the eligibility criteria (Figure 1); 99 had complete baseline data (the data for these men were used in an earlier baseline report on this population[40]). Of these 99 men, 88 participated in either the ES-HIM or the HP condition (i.e., attended at least the first session). Reasons for attrition among the 117 eligible men included the following: lost to follow-up after the baseline assessment (n = 20), incarceration (n = 3), entered substance abuse treatment (n = 2), relocated (n = 2), obtained employment (n = 1), and were unavailable (n = 1). There were no differences between these 29 eligible men who did not participate and the 88 enrolled intervention participants with respect to demographic characteristics.

After completing the baseline assessment, participants were randomized to either the ES-HIM or HP condition. Both conditions consisted of 6 small-group sessions 2 hours each in duration, administered over 3 weeks by a trained, ethnically matched male facilitator. After completing the intervention conditions, participants were posttested at 3 and 6 months. Biomarkers (cortisol, epinephrine, norepinephrine, and dopamine) were assayed from a 12-hour overnight urine sample collected at home by participants in a container with a preservative (0.3 g of sodium metabisulfite). Neopterin was collected in a preservative-free container requiring only a single-spot urine sample from the first morning void.[50,51] Participants completed a urine collection diary,
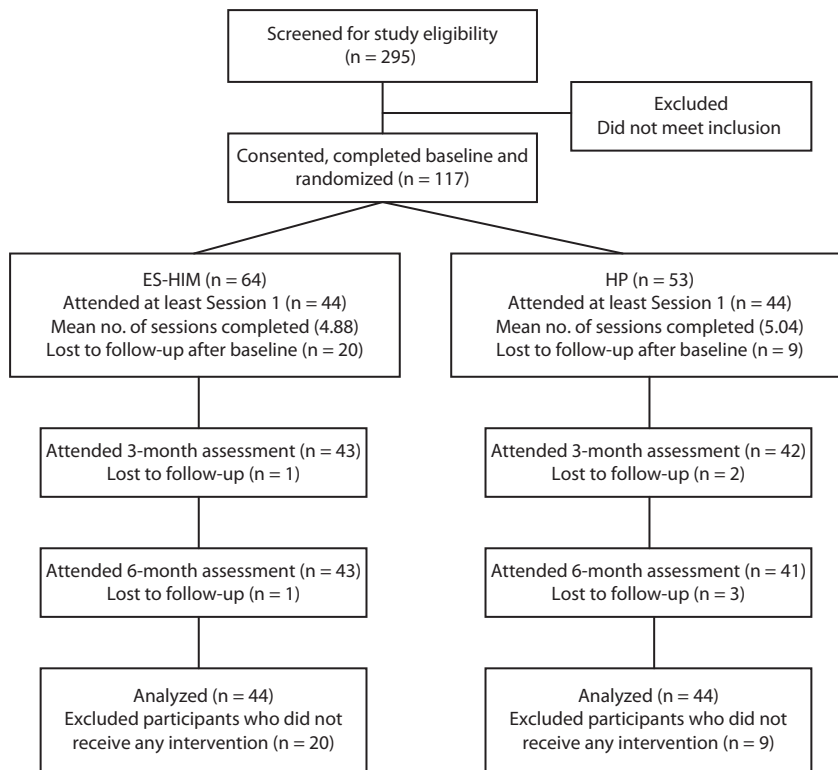
Note. ES-HIM = Enhanced Sexual Health Intervention for Men; HP = health promotion intervention. Not receiving any intervention means not having attended at least Session 1.

**FIGURE 1—Participant flow diagram: Enhanced Sexual Health Intervention for Men: Los Angeles County, CA; 2007-2011.**

including start and end collection times, and a health behavior questionnaire to assess behaviors that could influence interpretation of assay results (e.g., type and amount of exercise; use of alcohol, cigarettes, or drugs; and sleep patterns). Urine samples were collected within 10 days of participants' interviews. Participants were compensated up to $220 for full attendance and collection of biomarkers.

### Enhanced Sexual Health Intervention for Men

ES-HIM was adapted from the evidence-based Sexual Health Intervention for Men,[52] a 6-session intervention targeting HIV-positive African American and Latino men with histories of CSA, and the Women's Enhanced Sexual Health Intervention, an 11-session intervention targeting HIV-positive women with histories of CSA.[53] Guided by cognitive–behavioral approaches[54] and an ecological framework that addresses individual, interpersonal, social, and cultural factors,[55] ES-HIM focuses on changes in sexual behavior and improvements in psychological health. The conceptual model supports adaptation over time and contextualizes the interactions of overlapping ecosystems, with the individual being at the core. The concept that previous experiences, both individually and cumulatively, could affect sexual decision making was underscored.

Sexual risk reduction was framed from the perspective of each participant being a member of a triple minority group (i.e., being HIV positive and being a member of an ethnic and a sexual minority group). Issues associated with stigma and social isolation were discussed. Sexual ownership[56] (i.e., being able to make independent choices about sex and being responsible for one's own sexual and physical health) was prioritized along with caring for one's sexual partners, family, and community. Cultural and religious messages that could contradict HIV prevention efforts were

acknowledged. Sexual behaviors with male and female primary partners (e.g., spouses) and secondary (casual) partners were addressed. All discussions were framed within a culturally congruent social context. Topics included the influence of gender and ethnicity (e.g., the meaning of being an African American man); early socialization regarding gender and culture, as well as adult experiences (e.g., being a bisexual vs a heterosexual individual within the African American community); HIV stigma; and recognition of stressors (including histories of personal trauma) and use of learned strategies of coping and affect regulation to improve health outcomes.

Because trauma histories such as CSA often diminish one's self-worth and interest in self-preservation, ES-HIM addressed cognitive distortions and negative thoughts and emotions. The impact of CSA on personal decision making was framed as an important link between past experiences and current cognitive, affective, and behavioral patterns surrounding sexual behaviors and HIV-positive status. Participants were taught how to identify stress triggers and how these triggers could lead to unhealthy decisions and high-risk behaviors. Participants also learned communication skills, with an emphasis on negotiation and assertiveness training, that could be used to establish safe-sex boundaries and garner social support.

### Health Promotion Intervention

The HP condition was designed to control for the Hawthorne effect (i.e., research participants altering their behavior or responses because they know they are being studied rather than because of the experimental intervention itself) and reduce the likelihood that the effects of ES-HIM could be attributed to special attention and group interaction. Participants in both conditions received valuable information that extended beyond usual care. HP addressed health issues such as certain cancers, hypertension, diabetes, and heart disease, all of which are common among African American men, but did not focus on sexual behaviors. Participants were taught that these diseases could be prevented by changing personal behaviors (e.g., increasing physical activity, consuming a healthy diet, and ceasing cigarette smoking and alcohol and drug abuse) and managed with early detection and screening.

## Measures

*Childhood sexual abuse.* CSA was measured with 9 questions from the Revised Wyatt Sex History Questionnaire (WSHQ-R),[57] which assesses incidents of fondling, frottage, and attempted or completed intercourse prior to the age of 18 years. The WSHQ-R has been found to be reliable with African American MSM.[52,57] The age at which the experience occurred, the gender of the perpetrator, the relationship of the perpetrator to the victim, the overall number of incidents, and the use or threat of force were also assessed. A CSA severity index ranging from 0 to 7 was devised by selecting items that strongly correlated with reports of fear (a PTSD criterion). One risk point was assigned for each of the following: penetration, use of force, fear at time of incident, perpetrator identified as an immediate male family member, perpetrator identified as an immediate female family member, multiple perpetrators, and perpetrator identified as at least 5 years older than the victim. Scores were categorized as low severity (0–3) or high severity (4–7). This approach to characterizing CSA is more descriptive than simply assessing whether CSA was reported.[53,58]

*Sexual risk behaviors.* Sexual risk behaviors with male and female partners, including unprotected sexual intercourse (in which transmission of HIV is more likely to occur), were measured with 3 items derived from the WSHQ-R.[52,57] These items focused on episodes of unprotected insertive and receptive anal and vaginal sex as well as the number of times each type of episode had occurred in the preceding 3 months.

*Number of sexual partners.* Two items were used to assess total number of male and female sexual partners in the preceding 3 months.

*Posttraumatic stress disorder symptoms.* PTSD symptoms were measured with the 17-item Posttraumatic Diagnostic Scale (PDS),[59,60] which yields both a PTSD diagnosis (according to the criteria of the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision*) and a measure of symptom severity.[61] The scale includes a checklist of traumatic events (e.g., accident or fire, natural disaster, sexual abuse, imprisonment, life-threatening illness). Participants identify which events they have ever experienced or witnessed and specify the event that disturbed

them the most in the past month. Items focus on intrusive images, nightmares, reliving of trauma, memory loss, irritability, and hyper-vigilance. Responses are made on a 4-point scale ranging from not at all or only one time (0) to 5 or more times a week or almost always (3). The score range is 0 to 51. The instrument has been reported to have excellent reliability and validity[59,60]; the reliability coefficient in this study was 0.95.

*Depressive symptoms.* The Beck Depression Inventory-II (BDI-II) is a 21-item, multiple-choice instrument that measures the presence and degree of depressive symptoms on a 4-point scale.[62] Sum scores are calculated, and the score range is 0 to 63. The BDI-II has been used extensively.[63] In this study, the instrument's reliability coefficient was 0.94.

*Biomarker composite.* A biomarker composite of primary mediators of the stress response system was calculated (as in previous studies[64,65]). We calculated a sum score for each participant representing the number of indicators falling in the top quartile of risk for the sample as a whole. Scores ranged from 0 to 4, reflecting the possible number of biomarkers in a high-risk range. Because both elevated and blunted biological responses to stress[66–68] and low resting cortisol levels have been found among PTSD samples,[69–71] bidirectional risk strategies have been recommended for some biomarkers (i.e., extremely high or low values represent health risk).[72,73] As in previous studies, we used a bidirectional strategy to assess cortisol levels, with values in the highest or lowest 12.5% of the total sample considered to indicate risk.[74–76]

The Health Behaviors Questionnaire (D. A. G, unpublished instrument, 2002) was administered to assess activities in the 24 hours prior to urine collection. Questions focused on consumption of caffeinated beverages, alcoholic beverages, and cigarettes; quality and amount of aerobic and anaerobic exercise, sleep, and meals; use of prescribed and recreational drugs; and presence of illness symptoms. Information from the questionnaire was used to exclude urine samples that may have been contaminated by known confounders and to identify potential associations with the biomarker index. As mentioned, participants also completed a urine collection diary.

## Statistical Analyses

We conducted repeated measures analyses of variance to examine group and time effects for all outcome measures. Planned contrasts were used to examine changes from baseline to the 3-month follow-up and from the 3-month to 6-month follow-ups. Log transformations were necessary for all sexual risk outcomes. Age, tobacco use, and caffeine use, along with neopterin level (as a control for HIV progression), were included as covariates in all biomarker composite analyses. SAS statistical software, version 9.2 (SAS Institute, Cary, NC) was used for the analyses, and the macro procedure %GLIMMIX was used to fit the generalized linear repeated measures models.

## RESULTS

The ES-HIM and HP conditions each had 44 participants. The overall sample was predominantly middle aged (mean = 46.6 years, SD = 8.3), had completed high school (mean grade completed: 12.1; SD = 1.9), and was unemployed or unable to work (disabled; 83.9%); the majority had an annual income of $15 000 or below (90.59%). Slightly more than two thirds of the sample (68.6%) reported having spent more than 1 day in a jail or prison (mean number of times incarcerated: 5.5; SD = 4.5), and 30.5% were currently on parole or probation. Whereas all of the men were behaviorally bisexual, 25% and 20.9% self-identified as being "very to extremely" heterosexual and homosexual, respectively.

The mean number of children reported was 1.05 (SD = 1.67). Although the majority of the participants had never been married (72.4%), more HP participants (18.4%) than ES-HIM participants (9.2%) had been married ($\chi^2$ (1,87) = 3.94, $P < .05$). There were no other significant differences between groups according to demographic characteristics or mean number of sessions attended (ES-HIM: 4.88; HP: 5.04).

### Childhood Sexual Abuse

Sixty-seven percent of the participants reported having experienced touching, fondling, or frottage before the age of 18 years, whereas 47% and 52%, respectively, reported that they had been forced to perform and to receive oral sex. Approximately 48% reported attempted receptive anal penetration

(i.e., attempted rape), and 46.6% reported attempts at being forced to engage in anal sex (i.e., being forced to attempt anal sex as the inserter). Nearly 48% of the participants reported actual receptive anal penetration (i.e., being raped), and 42.5% reported being forced to engage in anal sex as the inserter.

The mean age at which the first incident occurred was 11.2 years (SD = 4.34). Among the men who reported experiences with male perpetrators, the mean number of incidents was 5.79 (SD = 8.59), whereas the mean number of incidents among those who reported experiences with female perpetrators was 4.65 (SD = 8.06). Forty-two percent of the participants identified the perpetrator as a male family member, and 33% identified the perpetrator as a female family member. Male and female first-degree relatives were identified as the perpetrators by 22% and 16% of the participants, respectively. In the case of 58% and 67% of the participants, respectively, the perpetrator was identified as not being a family member.

## Childhood Sexual Abuse Severity and Psychological Symptoms

A baseline analysis of a larger cohort from this same sample[40] showed that severe CSA and a greater number of PTSD symptoms were significantly associated with increased sexual risk behaviors. In addition, clinically meaningful PTSD symptom levels (PDS score above 17) predicted baseline biomarker composite scores. In this smaller intervention subset sample, nearly half (45.2%) of ES-HIM participants had experienced high-severity CSA, as compared with a little more than a quarter of the HP participants (26.3%). However, intervention group statistical comparisons showed no significant differences with respect to CSA severity or individual types of CSA experiences. Similarly, clinically meaningful symptom levels were not significantly different across intervention groups in the case of either PTSD (ES-HIM, 20.9%; HP, 27.3%) or depression (BDI-II score above 13) (ES-HIM, 20.5%; HP, 9%).

## Biomarker Composite

There was a significant intervention group effect for baseline biomarker composite scores. ES-HIM participants had lower biomarker composite scores (mean = 0.75, SE = 0.19) than HP participants (mean = 1.24, SE = 0.21;

group $P < .01$). To determine whether the baseline difference in intervention sample biomarker composite scores was due to differences in clinical symptom levels, we created a new variable that was coded 1 if the participant met the criteria for clinical PTSD or depression symptoms (or both) and 0 if the participant did not meet the criteria for either condition.

After controlling for standard biomarker composite covariates (age, tobacco use, caffeine use) and neopterin levels, we found a significant interaction ($P = .005$) between group assignment and presence of clinically meaningful symptoms. Those without clinical symptoms had equivalent biomarker composite scores across intervention conditions; among those with clinical symptoms, however, HP group members had significantly elevated biomarker composite scores relative to ES-HIM group members. We also examined whether clinical symptom status influenced neopterin levels. At the 3-month follow-up, participants with clinical symptoms showed elevated neopterin levels (mean = 358.52, SE = 41.36) relative to those without clinical symptoms (mean = 207.08, SE = 64.74; $P < .05$), but there were no differences at baseline or the 6-month follow-up.

In summary, clinically meaningful symptom effects were related to significant intervention group differences in baseline biomarker composite scores and neopterin levels at the 3-month follow-up. The study was not statistically powered to correct for these unexpected failures of the randomization process to equalize intervention groups. Thus, we conducted all subsequent preintervention–postintervention sexual risk and psychological outcome analyses excluding CSA severity, clinical symptom status, or neopterin level. However, we explored outcomes in relation to these factors when any large (detectable) changes were observed in intervention outcome patterns.

## Sexual Risk Behaviors, Number of Partners, and Psychological Distress

Table 1 shows outcomes over time for the variables assessed (sexual risk behaviors, number of partners, psychological symptoms, and biomarker composite scores) as a function of intervention condition.

*Sexual risk behaviors.* The planned analysis showed that both groups reduced unprotected anal insertive sex (time $P < .01$). However,

ES-HIM participants did not show any significant advantage relative to HP participants in reductions in anal insertive sex. Exploratory within-group comparisons indicated a significantly reduced number of episodes of unprotected anal insertive sex ($P < .05$) among ES-HIM participants from baseline (mean = 1.79, SD = 2.99) to the 3-month follow-up (mean = 0.67, SD = 1.59), and that reduction was sustained at the 6-month follow-up (mean = 0.46, SD = 2.01). In the HP group, by contrast, there was no significant reduction in unprotected anal insertive sex from baseline (mean = 1.02, SD = 1.65) to the 3- or 6-month follow-up.

We explored this ES-HIM group advantage in the context of CSA severity. A within-group analysis showed reduced episodes of unprotected anal insertive sex over time among ES-HIM participants with both high and low CSA severity histories (time, $P < .009$; Time × CSA Severity interaction, NS). In contrast, an analysis of HP participants with high and low CSA severity showed that there were no differences at baseline (overall mean = 0.93, SE = 0.34) but that the groups began to diverge at the follow-up assessments (Time × CSA Severity linear interaction, $P < .05$). HP participants with high CSA severity exhibited a dramatic increase in unprotected anal insertive sex after the intervention (6-month mean = 3.14, SE = 0.98), whereas those with low CSA severity showed decreased rates between baseline and the follow-ups (6-month mean = 0.08, SE = 0.51).

Both groups showed significant reductions in unprotected anal receptive sex and unprotected vaginal sex from baseline to the 3-month follow-up, and these reductions were sustained or additional decreases were observed at the 6-month follow-up (time $P < .001$). However, ES-HIM participants showed no significant advantage over HP participants with respect to decreasing unprotected anal receptive intercourse or vaginal sex. There were significantly more episodes of unprotected vaginal sex at baseline among HP participants than among ES-HIM participants ($P < .05$), but controlling for this baseline difference did not alter the significant reductions in vaginal sex over time for either group.

There were significant reductions in both groups in number of male and female sexual

TABLE 1—Intervention Outcomes Over Time as a Function of Intervention Group: ES-HIM Project; Los Angeles County, CA; 2007–2011

| Variable or Group | Baseline Assessment, Mean (SE) | 3-Month Assessment, Mean (SE) | 6-Month Assessment, Mean (SE) |
|---|---|---|---|
| **Sexual risk behaviors** | | | |
| Unprotected anal sex (insertive) | | | |
| ES-HIM | 1.79 (2.99) | 0.67 (1.59) | 0.46 (2.01) |
| HP | 1.02 (1.65) | 0.41 (1.07) | 0.90 (2.96) |
| Unprotected anal sex (receptive) | | | |
| ES-HIM | 1.62 (2.29) | 0.20 (0.90) | 0.29 (0.92) |
| HP | 1.30 (2.78) | 0.34 (1.04) | 0.49 (1.90) |
| Unprotected vaginal sex | | | |
| ES-HIM | 1.70 (1.99) | 0.21 (0.72) | 0.05 (0.31) |
| HP | 2.84 (2.99) | 0.12 (0.45) | 0.02 (0.16) |
| **Number of partners** | | | |
| Male | | | |
| ES-HIM | 1.45 (0.98) | 0.69 (1.00) | 0.88 (1.53) |
| HP | 1.84 (1.95) | 0.74 (1.80) | 0.39 (1.16) |
| Female | | | |
| ES-HIM | 1.43 (1.62) | 0.21 (0.60) | 0.51 (1.62) |
| HP | 1.41 (1.32) | 0.24 (0.48) | 0.10 (0.30) |
| **Psychological symptoms** | | | |
| PTSD symptoms[a] | | | |
| ES-HIM | 10.44 (12.25) | 5.17 (7.76) | 4.61 (5.99) |
| HP | 11.00 (12.17) | 5.10 (8.06) | 2.49 (5.03) |
| Depressive symptoms[b] | | | |
| ES-HIM | 8.18 (10.48) | 4.44 (6.65) | 4.63 (6.20) |
| HP | 4.98 (6.87) | 3.48 (6.34) | 2.39 (5.61) |
| **Biomarker composite[c]** | | | |
| Primary neurohormonal mediators[d] | | | |
| ES-HIM | 0.93 (0.19) | 0.84 (0.18) | 1.62 (0.19) |
| HP | 1.17 (0.21) | 1.00 (0.19) | 0.89 (0.21) |

*Note.* ES-HIM = Enhanced Sexual Health Intervention for Men; HP = health promotion intervention; PTSD = posttraumatic stress disorder. For sexual risk behaviors and number of partners, there is no score range because any number of partners or sex acts can be reported (i.e., no set upper limit).
[a]Score range was 0–51.
[b]Score range was 0–63.
[c]Adjusted for age, tobacco and caffeine use, and neopterin level.
[d]Score range was 0–4.

partners from baseline to the 3-month follow-up, and these effects were sustained or additional reductions were evident at the 6-month follow-up (time $P < .001$). However, ES-HIM participants showed no significant advantage over HP participants in terms of a decreased number of sexual partners.

*Psychological symptoms.* There were significant reductions in both groups in PTSD (time $P < .001$) and depression (time $P < .01$) symptom severity from baseline to the 3-month follow-up, and these effects were sustained

or further reductions were observed at the 6-month follow-up. ES-HIM participants showed no significant advantage over HP participants with respect to reductions in PTSD symptoms; however, within-group exploratory analyses showed significantly decreased depression symptoms among ES-HIM participants from baseline to the 3-month follow-up, and this reduction was sustained from the 3-month to the 6-month follow-up (time $P < .01$). In contrast, HP participants did not exhibit significantly reduced depression symptoms from

baseline to the 3-month follow-up, and they showed only a modest (but significant) reduction from baseline to the 6-month follow-up (time $P < .05$).

*Biomarker composite.* As noted, there were significant between-group differences in biomarker composite scores at baseline, along with a significant interaction between intervention condition and clinically meaningful symptoms (no symptoms vs PTSD, depression, or both). HP participants with clinically meaningful symptoms showed large elevations on the biomarker composite (unadjusted mean = 1.9, SE = 0.36) relative to each of the other groups (HP participants without clinical symptoms and ES-HIM participants both with and without clinical symptoms; mean = 0.64, SE = 0.26). Because of this lack of equivalence at baseline, clinical symptoms were examined together with group assignment in the biomarker composite analyses over time after age, tobacco and caffeine use, and neopterin level had been taken into account. HP participants with clinically meaningful symptoms exhibited reductions in primary biomarkers over time ($P < .05$), whereas there were no significant changes in biomarker composite scores over time in any of the other groups.

## DISCUSSION

Data from our small clinical trial indicate that culturally congruent interventions specifically targeting trauma-exposed groups may be efficacious in reducing psychological symptoms and may contribute to decreasing the spread of HIV. ES-HIM, a brief sexual risk and stress reduction intervention designed for HIV-positive African American MSMW who do not self-identify as gay and who have CSA histories, was effective in helping our participants with histories of both high and low CSA severity reduce episodes of unprotected anal insertive sex. It also helped them achieve reductions in their depression symptoms from baseline to the 3-month follow-up and sustain these symptom reductions at the 6-month follow-up. Reduced episodes of unprotected anal insertive sex among HIV-positive, behaviorally bisexual men may lead to a decreased risk of HIV transmission to their male and female partners. In addition to improvements in mental health, decreases in depression symptoms may

contribute to overall reductions in sexual risk behaviors.[77]

Both ES-HIM and HP participants exhibited reductions in unprotected anal receptive and vaginal sex and in numbers of male and female sexual partners; in addition, they showed decreased PTSD symptoms and sustained these reductions at the 6-month follow-up. The absence of an ES-HIM advantage in all outcomes may be due to the strong effects of CSA severity histories or associated preintervention clinical mental health symptoms and stress-related biomarker levels. Despite standard randomization procedures, ES-HIM participants exhibited a greater prevalence of high-severity CSA at baseline, whereas HP participants had elevated stress biomarker composites. The ES-HIM group advantage with respect to reducing unprotected anal insertive sex was related to the intervention's efficacy over time among participants with high and low CSA severity; by contrast, the HP condition failed to reduce this key sexual risk behavior among participants with high CSA severity.

Furthermore, HP participants with high CSA severity showed dramatic increases in unprotected anal insertive sex over time, suggesting that interventions that do not address CSA histories may exacerbate high-risk sexual behaviors. However, the failure of randomization to control baseline group differences undermines clear interpretation of our findings. Also, the biomarker composite of cortisol and catecholamines showed decreases from baseline to the 3-month follow-up and from the 3-month to 6-month follow-ups among participants with the highest baseline composite scores, that is, HP participants with clinical symptoms. Clinical status at baseline also influenced whether and when decreases in neopterin levels occurred.

These patterns are largely consistent with previous studies showing links between CSA severity and clinically meaningful PTSD or depression symptoms[38,39,74,78] and links between clinically meaningful symptoms and biomarker composite scores among women[65,67,69,74]; they are also consistent with the links between CSA severity and clinical symptom severity reported in the baseline-only analyses of the larger sample of 99 men that included our participants.[40] Nonetheless, replication of this study is needed with a larger sample to increase the probability of effective randomization and provide adequate statistical power to further examine the effectiveness of ES-HIM in improving sexual and mental health outcomes and biomarker composites.

Although a number of interventions have targeted people living with HIV/AIDS in an attempt to decrease sexual risk behaviors and improve psychological outcomes and immune functioning,[43,44,47] few have influenced change across all domains. To our knowledge, this is the first study to show intervention effects on sexual risk taking, psychological symptoms, a biomarker composite of cortisol and catecholamines, and the HIV immune indicator neopterin. That such effects interact with severity of past trauma and current mental health symptoms highlights the need to examine these factors during interventions.

Trauma and mental health symptoms may alter people's psychological motivation to change their sexual risk behaviors. Thus, interventions focusing on readiness to change, a key factor in behavioral intervention outcomes,[79,80] may need to address past trauma experiences that may act as a barrier to risk reduction. In addition, previous research shows that amount and severity of early trauma are not only associated with an increased risk of maladaptive behavioral patterns but are also linked to less effective coping skills as well as lower levels of social support, educational achievement, and subsequent contextual support (e.g., employment and wages).[13] Our study included a population of men with limited education, extremely modest incomes, and histories of being incarcerated. As previously reported, CSA severity was positively correlated with frequency of exchanging sex for money or drugs, and PTSD severity was positively correlated with total number of sexual partners.[40] These links between sexual risk-taking and trauma-related psychological factors require a holistic approach to intervention (i.e., one that addresses both individual challenges as well as environmental or structural ones), and ES-HIM was developed with this need in mind.

There is a dearth of HIV risk reduction interventions targeting African American MSMW. Successful behavior change models must address key components of individual resiliency, including a person's ability to circumvent or resolve the negative effects of risk exposure and develop effective strategies to cope with challenges.[81–86] However, resiliency requires not only personal assets such as coping skills and self-efficacy but also resources to support improvements in health status.[81,82] Although brief interventions such as ES-HIM are extremely valuable in addressing individual change, longer term sources of structural support are needed as well. Only through use of a comprehensive paradigm will significant and sustained changes in health status occur. ∎

## About the Authors

*John K. Williams, Dorie A. Glover, Gail E. Wyatt, Kimberly Kisler, and Muyu Zhang are with the Department of Psychiatry and Biobehavioral Sciences, Semel Institute for Neuroscience and Human Behavior, University of California, Los Angeles. Honghu Liu is with the School of Dentistry, University of California, Los Angeles.*

*Correspondence should be sent to John K. Williams, MD, University of California, Los Angeles, Department of Psychiatry and Biobehavioral Sciences, Semel Institute for Neuroscience and Human Behavior, 760 Westwood Plaza, 38-260, Los Angeles, CA 90024-1759 (e-mail: keoniwmd@aol.com). Reprints can be ordered at http://www.ajph.org by clicking on the "Reprints" link.*

*This article was accepted October 12, 2012.*

## Human Participant Protection

This study was approved by the institutional review board at the University of California, Los Angeles. Before being enrolled, all study participants provided written informed consent.

## References

1. Centers for Disease Control and Prevention. HIV/AIDS among African Americans. Available at: http://www.cdc.gov/hiv/topics/aa/PDF/aa.pdf. Accessed March 1, 2013.

2. Centers for Disease Control and Prevention. Sub-population estimates from the HIV incidence surveillance system—United States, 2006. *MMWR Morb Mortal Wkly Rep.* 2008;57(36):985–989.

3. Johnson WD, Holtgrave DR, McClellan WM, Flanders WD, Hill AN, Goodman M. HIV intervention research for men who have sex with men: a 7-year update. *AIDS Educ Prev.* 2005;17(6):568–589.

4. Operario D, Smith CD, Arnold E, Kegeles S. The Bruthas Project: evaluation of a community-based HIV prevention intervention for African American men who have sex with men and women. *AIDS Educ Prev.* 2010; 22(1):37–48.

5. Peterson JL, Coates TJ, Catania J, et al. Evaluation of an HIV risk reduction intervention among African-American homosexual and bisexual men. *AIDS.* 1996; 10(3):319–325.

6. Wilton L, Herbst JH, Coury-Doniger P, et al. Efficacy of an HIV/STI prevention intervention for black men who have sex with men: findings from the Many Men, Many Voices (3MV) project. *AIDS Behav.* 2009;13(3): 532–544.

7. Mays VM, Cochran SD, Zamudio A. HIV prevention research: are we meeting the needs of African American men who have sex with men? *J Black Psychol.* 2004;30 (1):78–105.

8. Millett G, Malebranche D, Mason B, Spikes P. Focusing "down low": bisexual black men, HIV risk and heterosexual transmission. *J Natl Med Assoc.* 2005;97 (suppl 7):52S–59S.

9. Myrick R. In the life: culture-specific HIV communication programs designed for African American men who have sex with men. *J Sex Res.* 1999;36(2):159–170.

10. Williams JK, Wyatt GE, Wingood G. The four Cs of HIV prevention with African Americans: crisis, condoms, culture, and community. *Curr HIV/AIDS Rep.* 2010;7 (4):185–193.

11. Whiffen VE, Benazon NR, Bradshaw C. Discriminant validity of the TSC-40 in an outpatient setting. *Child Abuse Negl.* 1997;21(1):107–115.

12. Maniglio R. The impact of child sexual abuse on health: a systematic review of reviews. *Clin Psychol Rev.* 2009;29(7):647–657.

13. Green JG, McLaughlin KA, Berglund PA, et al. Childhood adversities and adult psychiatric disorders in the National Comorbidity Survey replication I: associations with first onset of DSM-IV disorders. *Arch Gen Psychiatry.* 2010;67(2):113–123.

14. Greenfield EA. Child abuse as a life-course social determinant of adult health. *Maturitas.* 2010;66(1): 51–55.

15. Hornor G. Child sexual abuse: consequences and implications. *J Pediatr Health Care.* 2010;24(6):358–364.

16. Roche DN, Runtz MG, Hunter MA. Adult attachment. *J Interpers Violence.* 1999;14(2):184–207.

17. Kalichman SC, Benotsch E, Rompa D, et al. Unwanted sexual experiences and sexual risks in gay and bisexual men: associations among revictimization, substance abuse, and psychiatric symptoms. *J Sex Res.* 2001; 38(1):1–9.

18. Rumstein-McKean O, Hunsley J. Interpersonal and family functioning of female survivors of childhood sexual abuse. *Clin Psychol Rev.* 2001;21(3):471–490.

19. Classen CC, Palesh OG, Aggarwal R. Sexual revictimization: a review of the empirical literature. *Trauma Violence Abuse.* 2005;6(2):103–129.

20. Whiffen VE, MacIntosh HB. Mediators of the link between childhood sexual abuse and emotional distress. *Trauma Violence Abuse.* 2005;6(1):24–39.

21. Balsam KF, Lehavot K, Beadnell B. Sexual revictimization and mental health: a comparison of lesbians, gay men, and heterosexual women. *J Interpers Violence.* 2011;26(9):1798–1814.

22. Williams JK, Kisler K, Glover D, Sciolla A. Exploring childhood sexual experiences and vulnerability to intimate partner violence among African American MSMW: was that abuse or love? In: Hynes LE, ed. *Sexual Abuse: Types, Signs and Treatments.* Hauppauge, NY: Nova Science Publishers; 2011:1–22.

23. Feiring C, Simon VA, Cleland CM. Childhood sexual abuse, stigmatization, internalizing symptoms, and the development of sexual difficulties and dating aggression. *J Consult Clin Psychol.* 2009;77(1):127–137.

24. Lovejoy DA. *Neuroendocrinology: An Integrated Approach.* New York, NY: John Wiley & Sons Inc; 2005.

25. McEwen BS, Stellar E. Stress and the individual: mechanisms leading to disease. *Arch Intern Med.* 1993; 153(18):2093–2101.

26. McEwen BS. Stressed or stressed out: What is the difference? *J Psychiatry Neurosci.* 2005;30(5):315–318.

27. McEwen BS. Protection and damage from acute and chronic stress: allostasis and allostatic overload and relevance to the pathophysiology of psychiatric disorders. *Ann N Y Acad Sci.* 2004;1032:1–7.

28. McEwen BS. Effects of adverse experiences for brain structure and function. *Biol Psychiatry.* 2000;48 (8):721–731.

29. Brondolo E, Thompson S, Brady N, et al. The relationship of racism to appraisals and coping in a community sample. *Ethn Dis.* 2005;15(suppl 5):S14–S19.

30. Mays VM, Cochran SD, Barnes NW. Race, race-based discrimination, and health outcomes among African Americans. *Annu Rev Psychol.* 2007;58:201–225.

31. Mays VM, Coleman LM, Jackson JS. Perceived race-based discrimination, employment status, and job stress in a national sample of black women: implications for health outcomes. *J Occup Health Psychol.* 1996;1(3): 319–329.

32. Geronimus AT. The weathering hypothesis and the health of African-American women and infants: evidence and speculations. *Ethn Dis.* 1992;2(3):207–221.

33. Geronimus AT, Hicken M, Keene D, Bound J. "Weathering" and age patterns of allostatic load scores among blacks and whites in the United States. *Am J Public Health.* 2006;96(5):826–833.

34. Briere J, Evans D, Runtz M, Wall T. Symptomatology in men who were molested as children. *Am J Orthopsychiatry.* 1988;58(3):457–461.

35. Weber FT, Gearing J, Davis A, Conlon M. Pre-pubertal initiation of sexual experiences and older first partner predict promiscuous sexual behavior of delinquent adolescent males—unrecognized child abuse? *J Adolesc Health.* 1992;13(7):600–605.

36. Nelson DE, Higginson GK, Grant-Worley JA. Using the Youth Risk Behavior Survey to estimate prevalence of sexual abuse among Oregon high school students. *J Sch Health.* 1994;64(10):413–416.

37. Lenderking WR, Wold C, Mayer KH, Goldstein R, Losina E, Seage GR. Childhood sexual abuse among homosexual men. *J Gen Intern Med.* 1997;12(4):250–253.

38. Holmes W, Foa E, Sammel M. Men's pathways to risky sexual behavior: role of co-occurring childhood sexual abuse, posttraumatic stress disorder, and depression histories. *J Urban Health.* 2005;82(suppl 1):i89–i99.

39. Chartier MJ, Walker JR, Naimark B. Health risk behaviors and mental health problems as mediators of the relationship between childhood abuse and adult health. *Am J Public Health.* 2009;99(5):847–854.

40. Glover DA, Williams JK, Kisler KA. Using novel methods to examine stress among HIV-positive African American men who have sex with men and women. *J Behav Med.* 2012;35(1):27–37.

41. Millett GA, Peterson JL, Wolitski RJ, Stall R. Greater risk for HIV infection of black men who have sex with men: a critical literature review. *Am J Public Health.* 2006;96(6):1007–1019.

42. Antoni MH, Cruess DG, Klimas N, et al. Stress management and immune system reconstitution in symptomatic HIV-infected gay men over time: effects on transitional naive T cells (CD4(+)CD45RA(+)CD29. *Am J Psychiatry.* 2002;159(1):143–145.

43. Brown JL, Vandable PA. Stress management interventions for HIV-infected individuals: reviews of recent intervention approaches and directions for future research. *Neurobehav HIV Med.* 2011;3:95–106.

44. Crepaz N, Passin WF, Herbst JH, et al. Meta-analysis of cognitive-behavioral interventions on HIV-positive persons' mental health and immune functioning. *Health Psychol.* 2008;27(1):4–14.

45. Scott-Sheldon LA, Kalichman SC, Carey MP, Fielder RL. Stress management interventions for HIV+ adults: a meta-analysis of randomized controlled trials, 1989 to 2006. *Health Psychol.* 2008;27(2):129–139.

46. Antoni MH, Cruess DG, Cruess S, et al. Cognitive-behavioral stress management intervention effects on anxiety, 24-hr urinary norepinephrine output, and T-cytotoxic/suppressor cells over time among symptomatic HIV-infected gay men. *J Consult Clin Psychol.* 2000; 68(1):31–45.

47. Carrico AW, Chesney MA, Johnson MO, et al. Randomized controlled trial of a cognitive-behavioral intervention for HIV-positive persons: an investigation of treatment effects on psychosocial adjustment. *AIDS Behav.* 2009;13(3):555–563.

48. Sikkema KJ, Hansen NB, Kochman A, et al. Outcomes from a group intervention for coping with HIV/AIDS and childhood sexual abuse: reductions in traumatic stress. *AIDS Behav.* 2007;11(1):49–60.

49. Sikkema KJ, Wilson PA, Hansen NB, et al. Effects of a coping intervention on transmission risk behavior among people living with HIV/AIDS and a history of childhood sexual abuse. *J Acquir Immune Defic Syndr.* 2008;47(4):506–513.

50. Wirleitner B, Schroecksnadel K, Winkler C, Fuchs D. Neopterin in HIV-1 infection. *Mol Immunol.* 2005;42 (2):183–194.

51. Djoba Siawaya JF, Ruhwald M, Eugen-Olsen J, Walzl G. Correlates for disease progression and prognosis during concurrent HIV/TB infection. *Int J Infect Dis.* 2007;11(4):289–299.

52. Williams JK, Wyatt GE, Rivkin I, Ramamurthi HC, Li X, Liu H. Risk reduction for HIV-positive African

American and Latino men with histories of childhood sexual abuse. *Arch Sex Behav.* 2008;37(5):763–772.

53. Wyatt GE, Longshore D, Chin D, et al. The efficacy of an integrated risk reduction intervention for HIV-positive women with child sexual abuse histories. *AIDS Behav.* 2004;8(4):453–462.

54. Bandura A. *Social Foundations of Thought and Action: A Social Cognitive Theory.* Upper Saddle River, NJ: Prentice-Hall; 1986.

55. Bronfenbrenner U. Toward an experimental ecology of human development. *Am Psychol.* 1977;32(7):513–531.

56. Chin DWG, Carmona JV, Loeb TB, Myers HF. Child sexual abuse and HIV: an integrative risk-reduction approach. In: Koenig LJDL, O'Leary A, Pequegnat W, eds. *Child Sexual Abuse to Adult Sexual Risk: Trauma, Revictimization, and Intervention.* Washington, DC: American Psychological Association; 2004:233–250.

57. *The Wyatt Sex History Questionnaire.* Los Angeles, CA: G. E. Wyatt; 1984.

58. Wyatt GE. The sexual abuse of Afro-American and white-American women in childhood. *Child Abuse Negl.* 1985;9(4):507–519.

59. Foa EB. Trauma and women: course, predictors, and treatment. *J Clin Psychiatry.* 1997;58(suppl 9):25–28.

60. Foa EB, Cashman L, Jaycox L, Perry K. The validation of a self-report measure of PTSD: the Posttraumatic Diagnostic Scale (PDS). *Psychol Assess.* 1997;9(4):445–451.

61. *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision.* Washington, DC: American Psychiatric Association; 2000.

62. Beck AT, Steers RA, Brown GK, eds. *BDI-II, Beck Depression Inventory: Manual.* 2nd ed. Boston, MA: Harcourt Press; 1996.

63. Joe S, Woolley ME, Brown GK, Ghahramanlou-Holloway M, Beck AT. Psychometric properties of the Beck Depression Inventory-II in low-income, African American suicide attempters. *J Pers Assess.* 2008;90(5):521–523.

64. McEwen BS, Seeman T. Protective and damaging effects of mediators of stress: elaborating and testing the concepts of allostasis and allostatic load. *Ann N Y Acad Sci.* 1999;896:30–47.

65. Glover DA, Stuber ML, Poland RE. Allostatic load in PTSD-symptomatic middle-aged mothers of child cancer survivors. *Psychiatry Biol Interpers Processes.* 2006;69(3):191–203.

66. Lanius RA, Hopper JW, Menon RS. Individual differences in a husband and wife who developed PTSD after a motor vehicle accident: a functional MRI case study. *Am J Psychiatry.* 2003;160(4):667–669.

67. Glover DA, Steele AC, Stuber ML, Fahey JL. Preliminary evidence for lymphocyte distribution differences at rest and after acute psychological stress in PTSD-symptomatic women. *Brain Behav Immun.* 2005;19(3):243–251.

68. Lanius RA, Bluhm R, Lanius U, Pain C. A review of neuroimaging studies in PTSD: heterogeneity of response to symptom provocation. *J Psychiatr Res.* 2006;40(8):709–729.

69. Glover DA, Poland RE. Urinary cortisol and catecholamines in mothers of child cancer survivors with and without PTSD. *Psychoneuroendocrinology.* 2002;27(7):805–819.

70. Yehuda R. Post-traumatic stress disorder. *N Engl J Med.* 2002;346(2):108–114.

71. Yehuda R, McEwen BS. Protective and damaging effects of the biobehavioral stress response: cognitive, systemic and clinical aspects: ISPNE XXXIV meeting summary. *Psychoneuroendocrinology.* 2004;29(9):1212–1222.

72. Seplaki CL, Goldman N, Glei D, Weinstein M. A comparative analysis of measurement approaches for physiological dysregulation in an older population. *Exp Gerontol.* 2005;40(5):438–449.

73. Glei DA, Goldman N, Chuang YL, Weinstein M. Do chronic stressors lead to physiological dysregulation? Testing the theory of allostatic load. *Psychosom Med.* 2007;69(8):769–776.

74. Glover DA, Loeb TB, Carmona JV, et al. Childhood sexual abuse severity and disclosure predict posttraumatic stress symptoms and biomarkers in ethnic minority women. *J Trauma Dissociation.* 2010;11(2):152–173.

75. Glover DA, Garcia-Aracena EF, Lester P, Rice E, Rothram-Borus MJ. Stress biomarkers as outcomes for HIV+ prevention: participation, feasibility and findings among HIV+ Latina and African American mothers. *AIDS Behav.* 2010;14(2):339–350.

76. Glover DA, Garcia-Aracena EF, Mohlman J. Peripheral biomarker composite associated with smaller hippocampal volume. *Neuroreport.* 2008;19(13):1313–1316.

77. Alvy LM, McKirnan DJ, Mansergh G, et al. Depression is associated with sexual risk among men who have sex with men, but is mediated by cognitive escape and self-efficacy. *AIDS Behav.* 2011;15(6):1171–1179.

78. Sciolla A, Glover DA, Loeb TB, Zhang M, Myers HF, Wyatt GE. Childhood sexual abuse severity and disclosure as predictors of depression among adult African-American and Latina women. *J Nerv Ment Dis.* 2011;199(7):471–477.

79. Prochaska JO, DiClemente CC, Norcross JC. In search of how people change: applications to addictive behaviors. *Am Psychol.* 1992;47(9):1102–1114.

80. Miller WR, Rollnick S. *Motivational Interviewing: Preparing People to Change Addictive Behavior.* New York, NY: Guilford Press; 1991.

81. Fergus S, Zimmerman MA. Adolescent resilience: a framework for understanding healthy development in the face of risk. *Annu Rev Public Health.* 2005;26:399–419.

82. Gwadz MV, Clatts MC, Yi H, Leonard NR, Goldsamt L, Lankenau S. Resilience among young men who have sex with men in New York City. *Sex Res Soc Policy.* 2006;3(1):13–21.

83. Garmezy N, Masten AS, Tellegen A. The study of stress and competence in children: a building block for developmental psychopathology. *Child Dev.* 1984;55(1):97–111.

84. Masten AS. Ordinary magic: resilience processes in development. *Am Psychol.* 2001;56(3):227–238.

85. Luthar SS, Cicchetti D, Becker B. The construct of resilience: a critical evaluation and guidelines for future work. *Child Dev.* 2000;71(3):543–562.

86. Rutter M. Resilience in the face of adversity: protective factors and resistance to psychiatric disorder. *Br J Psychiatry.* 1985;147:598–611.