Published in final edited form as:

Compr Psychiatry. 2012 October ; 53(7): 901–906. doi:10.1016/j.comppsy.2012.03.009.

Autonomic reactivity and hypothalamic pituitary adrenal axis dysregulation in spouses of Oklahoma City bombing survivors 7 years after the attack

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Abstract

Objective—The objective of this exploratory pilot study was to examine autonomic reactivity and hypothalamic pituitary adrenal axis dysregulation in spouses of highly exposed survivors of the 1995 Oklahoma City bombing.

Methods—This study compared psychiatric diagnoses and biological stress markers (physiological reactivity and cortisol measures) in spouses of bombing survivors and matched community participants. Spouses were recruited through bombing survivors who participated in prior studies. Individuals with medical illnesses and those taking psychotropic medications that would confound biological stress measures were excluded. The final sample included 15 spouses and 15 community participants. The primary outcome measures were psychiatric diagnoses assessed with the Diagnostic Interview Schedule for *DSM-IV* (DIS-IV). Biological stress markers were physiological reactivity and recovery in heart rate and blood pressure responses to a trauma interview and cortisol (morning, afternoon, and diurnal variation).

Results—Compared to the community participants, spouses evidenced greater reactivity in heart rate, systolic blood pressure, and diastolic blood pressure; delayed recovery in systolic blood pressure; and higher afternoon salivary cortisol.

Conclusions—The results support the need for further research in this area to clarify post-disaster effects on biological stress measures in the spouses of survivors and the potential significance of these effects and to address the needs of this important population which may be overlooked in recovery efforts.

1. Introduction

While sparse, the literature on the spouses of trauma victims suggests the potential for adverse effects of trauma in individuals who are not themselves directly exposed to a traumatic event but are the mates of victims. The *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR)* [1] includes learning about the trauma experience of a close associate, such as a spouse, as a qualifying trauma exposure for the diagnosis of posttraumatic stress disorder (PTSD). This suggests that the effects of trauma through another individual's exposure may be associated with the development of PTSD.

Research has examined the spouses of military veterans [2], rescuers, and responders [3–5] and peace makers [6]. The experiences and consequences of trauma in terrorism victims and their spouses may differ from those of military veterans, rescuers and responders, and peace makers in several ways: (1) responders and service personnel have been selected, or have self selected, for involvement in the activities that resulted in their trauma exposure presumably with selection based on certain strengths; (2) these individuals typically undergo training which may enhance their coping and resilience; (3) they are able to anticipate their involvement in such events; and (4) they are likely to see themselves in a helping role rather than as primary victims of an event. On the other hand, responders and peace makers may suffer repeated and prolonged trauma exposure over the course of multiple events or protracted recovery operations. Thus, their experiences and reactions, and those of their spouses, are likely to differ from those of the spouses of terrorism victims.

At the time, the 1995 Oklahoma City bombing was the deadliest act of terrorism on US soil. In their study of the Oklahoma City community after the bombing, Smith et al [7] found that over 60% of a random sample of Oklahoma City residents reported at least 1 of 8 bombing-related experiences: personal injury, time off work, home damage, family member injured or killed, other member of the household injured or killed, knew anyone else injured or killed, attended a funeral or memorial service, or attended any other ceremonies. Family members of victims may have possessed information with which to generate mental impressions of what occurred. For example, spouses may have known that their husband or wife was at the site on the morning of the incident. As the disaster unfolded, spouses may have created their own images and narratives using the accumulated information that circulated throughout the community, which was derived from the physical evidence of the explosion that could be felt, heard, and seen for miles and from media coverage and word of mouth. Speculation about what had occurred was rampant over the days and weeks to follow as details emerged.

Six months after the bombing, North and colleagues [8] studied a representative sample of 182 directly exposed Oklahoma City survivors with follow-up studies conducted 17 months [9] and 7 years [10] after the event. Tucker et al [11, 12] assessed a physically-healthy subsample of the 7-year follow-up study participants to examine physiological reactivity [11, 12] and salivary cortisol [11] relative to a community comparison sample. The physically-healthy but highly exposed Oklahoma City bombing survivors studied by Tucker et al [11, 12] had significantly greater autonomic reactivity than the community group matched for sex and age who were not in the vicinity of the event, not related to deceased victims, and not rescue or recovery workers. The survivors who met criteria for current PTSD had significantly higher morning cortisol levels than both survivors who did not meet criteria for current PTSD and the community participants [12].

Although the biological changes in trauma have been examined in disaster survivors, minimal attention has focused on the families of these individuals who are exposed only through their close relationships with directly-exposed survivors. Thus, in this exploratory pilot study, we examined autonomic reactivity and hypothalamic-pituitary-adrenal (HPA) axis dysregulation in spouses of a subgroup of the directly-exposed survivors assessed by Tucker et al [11, 12] relative to unexposed participants in the community group. We hypothesized that spouses of bombing survivors would experience heightened physiological reactivity and higher morning and afternoon cortisol secretion levels with less diurnal variation (ie, less decline from morning to afternoon) compared to the community comparison group.

2. Methods

We used a sample of Oklahoma City bombing survivors, taken from a longitudinal study [8–10], who had participated in biological assessments at seven-year follow up [11, 12] to generate a group of spouses of directly-exposed bombing survivors. The University of Oklahoma Health Sciences Center and Washington University School of Medicine Institutional Review Boards approved the index and follow-up bombing studies; the University of Texas Southwestern Medical Center Institutional Review Board approved additional data analysis for these studies. Participants for this exploratory pilot study of survivor spouses were recruited through postal mail and/or telephone contacts obtained from the survivors assessed in the seven-year follow-up study. All participants provided written informed consent. They were paid \$75 to compensate for their time and effort.

2.1. Participants

The study participants represented 2 groups—spouses of directly-exposed bombing survivors who participated in the seven-year follow-up study [10] and unexposed community members matched with survivor spouses on sex, race, age, and education. Of 113 bombing survivors who participated in the 7-year follow-up study, 94 survivors were married at the time of the 7-year assessment. Twenty-six (28%) partners of these 94 survivors agreed to participate in this spouse study. The community participants matched to spouses on sex, race, and age were extracted from the original community group ($n = 78$) used in the biological assessments conducted by Tucker et al [11, 12]. Recruitment of participants in the study of biological markers in the survivors excluded individuals with medical illnesses and those taking psychotropic medications that would confound biological stress measures [11, 12]. Likewise, selection of spouses and community participants in this study excluded individuals with medical illnesses and those taking psychotropic medications.

Of 26 participating spouses, we excluded one African American spouse and the matching community participant to eliminate any possible race/ethnic-related effects on blood pressure [13, 14] and diurnal patterns of salivary cortisol levels [15]. Thus, all participants were Caucasian. We also excluded 5 spouses and their matching community participants because one or both of the potential pair had not participated in either the physiological or cortisol assessments. This yielded a final sample size of 15 spouse and 15 community participants.

None of the spouses and none of the community participants were physically present at the bomb site at the time of the incident. The survivor partners of most of the spouses (80%, $n = 12$) were injured in the attack. Of the community group, 7 reported knowing someone injured and/or killed in the bombing though none reported immediate family members injured or killed.

2.2. Procedures and variables

To prevent the possibility that cortisol measures could be affected by biological responses to undergoing psychometric test batteries and physiological assessments, morning salivary cortisol samples were taken first at 8:00 AM, immediately after informed consent was obtained, followed by physiological and then psychometric assessments.

2.2.1. Salivary cortisol measurement—Cortisol was measured with salivary samples which are less invasive and less anxiety-provoking than obtaining serum. Salivary and plasma cortisol levels are highly correlated [16]. Participants were instructed to rinse their mouths with water, avoid eating or drinking fifteen minutes before samples were collected,

and abstain from caffeine and nicotine before the visit. Salivary samples for cortisol levels were collected in Salivette tubes (Sarstedt, Nümbrecht, Germany) from all participants at 8^{AM} for a morning cortisol sample. Participants were given a tube to take home to collect a 4^{PM} afternoon cortisol sample and instructed to refrigerate and return it. All samples were assayed in duplicate using salivary cortisol kits provided by Salimetrics, LLC (Salimetrics, LLC, State College, PA). Samples were centrifuged for 2 minutes and stored at -20°C , with cortisol levels later measured by radioimmunoassay methods (Salimetrics, LLC, State College, PA). Duplicate salivary cortisol assays were averaged, resulting in one data point for the morning and one for the afternoon levels.

Morning (8^{AM}) and afternoon (4^{PM}) salivary cortisol levels were quantified as micrograms per deciliter ($\mu\text{g/dL}$), and diurnal variation was calculated by subtracting the afternoon level from the morning level ($_{\text{AM-PM}}$).

2.2.2. Physiological assessment—Each participant was seated in a comfortable chair in the private testing room, with a blood pressure cuff placed on the left arm and electrocardiogram electrodes appropriately placed. A Lablink Modular Instrument System (Biopac Systems Inc, 1999) recorded heart rate via standard electrocardiogram Lead 2 placement. A Critikon Dinamap Vital Signs Monitor (1846 SX) (GE Healthcare, Milwaukee, WI) recorded systolic and diastolic blood pressures every 30 seconds with a Compaq computer (Hewlett-Packard, Palo Alto, CA) interfaced with all equipment. To reduce potential anxiety about the procedure, participants were shown the physiological equipment before the autonomic assessment. Autonomic measurements were made for four minutes during a resting phase (pre-test phase), for 4 minutes during a semi-structured interview about the bombing (test phase), and for 4 minutes of rest after the interview was completed (post-test phase). The data were reduced by calculating means per phase, yielding 3 data points per measurement. During the interview, participants discussed how they felt after learning about the bombing, whether they knew anyone injured or killed, and other bombing-related issues that occurred to them.

Physiological assessment measures included heart rate quantified as beats per minute (bpm) and systolic and diastolic blood pressure quantified as millimeters of mercury (mm/hg) in 3 time periods—pre-test, test, and post-test. Physiological reactivity scores were calculated by subtracting pre-test from test scores in measures of heart rate, systolic blood pressure, and diastolic blood pressure and physiological recovery (return to pre-test level) scores were calculated by subtracting pre-test from post-test scores in physiological measures. Physiological variables analyzed in this study were pre-test, reactivity, and recovery scores for heart rate, systolic blood pressure, and diastolic blood pressure.

2.2.3. Diagnostic assessment—All participants were administered the Diagnostic Interview Schedule for *DSM-IV* (DIS-IV) [17] and the Disaster Supplement [18], which were used to assess cumulative post-disaster and current PTSD, major depression, generalized anxiety disorder, and panic disorder as well as other aspects of participant experiences and reactions.

Bombing-related PTSD was defined as meeting all *DSMIV-TR* [1] Criteria A through F for the diagnosis of PTSD at any time during the period leading up to the assessment. Spouses of the directly-exposed survivors met Criterion A through the traumatic exposure of a close associate, their spouse, and were considered to meet full PTSD diagnostic criteria if they reported one or more bombing-related Criterion B (intrusion) symptoms, 3 or more new Criterion C (avoidance and numbing) symptoms, and 2 or more new Criterion D (hyperarousal) symptoms related to the traumatic event and also reported that symptoms

lasted more than one month (Criterion E) and were associated with clinically significant emotional distress or functional impairment (Criterion F).

Major depressive disorder was characterized according to *DSM-IV-TR* [1] criteria as experiencing one or more major depressive episode. To meet criteria for a major depressive episode, a participant reported depressed mood or loss of interest for a duration of at least 2 weeks accompanied by at least four additional depressive symptoms including anhedonia, significant weight loss or weight gain, insomnia or hypersomnia, psychomotor agitation or retardation, fatigue or loss of energy, feelings of worthlessness or guilt, concentration problems, and recurrent thoughts of death or suicide or a specific suicide plan or attempt. In addition, the individual also reported clinically significant distress or impairment due to symptoms that were not substance induced or due to a general medical condition.

Generalized anxiety disorder and panic disorder were similarly characterized according to *DSM-IV-TR* [1] criteria for each diagnosis. Participants who met *DSM-IV-TR* [1] criteria for a disorder at some time during the seven years after the bombing were considered to have a cumulative post-bombing disorder. Participants who met diagnostic criteria at some time during the seven years after the bombing and were symptomatic within one month at the time of the seven-year follow-up study were considered to have a current diagnosis.

2.3. Data analysis

Descriptive statistics of variables of interest were calculated for each group of participants—spouse and the community participants. Wilcoxon rank-sum test was used to examine between-group differences—spouses versus the community participants—in physiological and salivary cortisol measures. $P < .05$ was set for statistical significance. SAS 9.2 (SAS Institute, Cary, NC) was used for all statistical analyses.

3. Results

Table 1 displays the descriptive statistics for sex, age, education, marital status, and cumulative and current post-bombing psychiatric disorders this study considered for the spouse and community participants. Although all of the spouses of directly-exposed survivors were married at the time of the study, 4 (27%) of the 15 community participants were not married, 3 were divorced, and 1 had never been married. The 2 groups were matched on sex, race, age, and education and there were no statistically significant differences by marital status in any of the physiological or cortisol measures. Five spouses met diagnostic criteria for one or more post-bombing psychiatric disorders. Only 2 out of these 5 spouses met criteria for a current post-bombing disorder—one for current bombing-related PTSD and one for current major depression. A 51-year-old male spouse with current bombing-related PTSD did not meet criteria for any other post-bombing disorder examined; his survivor mate met diagnostic criteria for cumulative bombing-related PTSD comorbid with cumulative post-bombing major depression but was not currently symptomatic of either disorder at the time of the study. A 52-year-old female spouse who met criteria for current major depression at the time of the study did not meet criteria for any other post-bombing disorder examined; her survivor mate did not meet diagnostic criteria for any of the post-bombing psychiatric disorders examined. See Table 1. No statistically significant differences (at $< .05$ level) were found between spouse and community participants in the prevalence of any of the psychiatric disorders examined.

Table 2 displays results of physiological and cortisol testing. While there were no significant differences in pretest physiological measures of heart rate, systolic blood pressure, or diastolic pressure, spouses had significantly greater physiological reactivity in heart rate and systolic blood pressure than community participants. Relative to community participants,

spouses' recovery from post-test back to pre-test levels for systolic blood pressure was significantly delayed. Spouses also had a significantly higher mean afternoon cortisol level than community participants.

4. Discussion

Prior studies have documented psychological sequelae in the spouses of military veterans [2], rescuers and responders [3–5], and peace makers [6], but only one of these studies examined biological stress measures [5]. Pfefferbaum et al [5] found autonomic reactivity in a small nonrepresentative sample of spouses of firefighters who responded to the Oklahoma City bombing; their study did not use a comparison sample and did not examine HPA axis functioning. Preliminary evidence suggests that the child relatives of disaster victims may also experience physiological reactivity and HPA axis effects [19–21]. Thus, this exploratory pilot study which compared biological reactions of a group of physically-healthy spouses of Oklahoma City bombing survivors and demographically-matched unexposed community participants is one of only a few investigations of biological markers in family members of disaster survivors [5, 19–21].

Our sample was too small to draw conclusions about the etiology or implications of the current post-bombing disorders in spouses seven years after the bombing or about the associations between psychiatric disorders and physiological and HPA axis functioning. Future studies should assess the mutual influence of psychiatric conditions and emotions between couples and the interrelationships with physiological and HPA axis functioning, potential changes in their relationships post disaster, the effects of subsequent trauma and stressors, and other factors that might affect emotional and biological stress measures.

Spouses and the unexposed community participants significantly differed in physiological reactivity and recovery and in afternoon cortisol levels, demonstrating that spouses of terrorism survivors may also experience biological effects of trauma even in the absence of personal physical exposure to an event. Of note, spouses' physiological reactivity was robust when compared to the community participants, and it did not return to pre-test levels during the testing period, suggesting a persistent autonomic response to reminders of their partner's trauma. Diurnal variation in HPA axis functioning was intact in these spouses, not differing from the community comparison group, while their higher afternoon cortisol was consistent with higher morning cortisol levels in direct survivors of the bombing reported in an earlier study of bombing survivors [12]. Studies have found varying abnormalities in cortisol secretion in individuals with PTSD, including higher [12, 22], lower [23], and similar [24] cortisol levels compared to controls. The literature on cortisol in association with major depression is more consistent, generally showing higher evening salivary cortisol secretion levels for those with depressive symptoms than those without [25]. It is unclear the extent to which the physiological reactivity, delayed physiological recovery, and elevated cortisol in the participants we studied were pathologic or predictive of future stress-related health problems. Nonetheless, these results suggest an important area for consideration in future research.

The primary limitation in this exploratory pilot study was the small sample size. A larger sample may have revealed additional associations. Furthermore, recruitment of participants in this study excluded individuals with medical illnesses and those taking psychotropic medications that would confound biological stress measures. This may have resulted in obtaining a physically and/or psychologically healthy sample that was not representative of the survivors in general or of their spouses. We do not know if spouses of more symptomatic or impaired survivors might have exhibited even greater adverse outcomes relative to community participants. In addition, survivor spouses and community participants who

experienced more severe psychological reactions may have elected not to enroll in the study. Seven community participants knew individuals injured and/or killed in the bombing, albeit not close family members. It is unclear how this may have affected the results of the study. If community participants had not known people injured or killed, even greater differences between this group and the spouses might have been observed. Given the cross-sectional design of our study, it is also unclear if the biological differences between spouses and community participants represent late-occurring findings perhaps due to an accumulation of secondary adversities and/or if they represent persistent reactions having onset closer in time to the event. The results are provocative, however, and support the need for further research in this area to clarify post-disaster effects on spouses of survivors and the potential significance of these effects and to address the needs of this important population which may be overlooked in recovery efforts.

Acknowledgments

This research was supported in part by the National Memorial Institute for the Prevention of Terrorism (MIPT) and the Office of Justice Programs, National Institute of Justice, Department of Justice (DOJ) Award Number MIPT106-113-2000-020 (B Pfefferbaum) and the National Institute of Mental Health (NIMH) Grant Number MH40025 (CS North). Dr North discloses employment by VA North Texas Health Care System, Dallas, Texas. Points of view in this document are those of the authors and do not represent the official position of MIPT; DOJ; NIMH; the Department of Veterans Affairs; or the US Government.

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Table 1

Demographics and psychiatric disorders for spouse and community participants

	Spouse (n = 15)	Community (n =15)
Demographic characteristics		
Sex	n (%)	n (%)
Female	11	11
Male	4	4
Age	Mean (SD)	Mean (SD)
Years	47.33 (8.47)	46.00 (8.78)
Education	n (%)	n (%)
High school graduate	5 (33.33)	6 (40.00)
Some college	5 (33.33)	4 (26.67)
College graduate	5 (33.33)	5 (33.33)
Marital status	n (%)	n (%)
Married	15 (100.00)	11 (73.33)
Not married	0 (0.00)	4 (26.67)
Post-bombing psychiatric disorder	n (%)	n (%)
Cumulative bombing-related PTSD	2 (13.33)	0 (0.00)
Current bombing-related PTSD	1 (6.67)	0 (0.00)
Cumulative post-bombing major depression	3 (20.00)	4 (26.67)
Current major depression	1 (6.67)	0 (0.00)
Cumulative post-bombing general anxiety disorder	1 (6.67)	1 (6.67)
Current general anxiety disorder	0 (0.00)	0 (0.00)
Cumulative post-bombing panic disorder	0 (0.00)	1 (6.67)
Current panic disorder	0 (0.00)	1 (6.67)

Table 2

Physiological reactivity, recovery, and cortisol for spouse and community participants

	Spouses (n = 15)	Community (n = 15)	Analysis ^a <i>P</i>
Physiological reactivity	Mean (SD)	Mean (SD)	
Heart rate (bpm)			
Pre-Test	70.17 (12.48)	72.10 (12.15)	<i>P</i> = .5520
Reactivity (test – pre-test)	9.53 (7.38)	2.73 (3.72)	<i>P</i> = .0081
Recovery (post-test – pre-test)	1.10 (4.94)	–0.70 (1.61)	<i>P</i> = .4967
Systolic blood pressure (mmHg)			
Pre-test	121.18 (16.12)	124.79 (16.54)	<i>P</i> = .7733
Reactivity (test – pre-test)	20.07 (12.07)	6.46 (8.20)	<i>P</i> = .0024
Recovery (post-test – pre-test)	4.93 (9.10)	–1.77 (6.05)	<i>P</i> = .0410
Diastolic blood pressure (mmHg)			
Pre-test	71.93 (8.76)	72.67 (9.32)	<i>P</i> = .8532
Reactivity (test – pre-test)	9.57 (6.86)	5.13 (5.10)	<i>P</i> = .0687
Recovery (post-test – pre-test)	2.13 (5.60)	0.47 (4.56)	<i>P</i> = .4481
Salivary cortisol (µg/dL)			
am	0.37 (0.39)	0.24 (0.14)	<i>P</i> = .4135
pm	0.14 (0.06)	0.09 (0.07)	<i>P</i> = .0412
am–pm	–0.23 (0.40)	–0.15 (0.15)	<i>P</i> = .8694

^aWilcoxon rank sum 2-sided test.