*Journal of Traumatic Stress*
June 2013, *26,* 1–7

# Perievent Panic Attacks and Panic Disorder After Mass Trauma: A 12-Month Longitudinal Study

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Panic attacks frequently lead to psychopathological disorders, including panic disorder. Even though panic disorder is a highly comorbid and disabling mental health problem associated with stressful life or traumatic events, perievent panic attacks and their association with panic disorder have hardly been investigated as a central topic after mass trauma. Using data from a longitudinal population-based assessment of Madrid residents after the March 11, 2004 train bombings ($N = 1{,}589$), with assessments conducted 1, 6, and 12 months after the attacks, the rate of perievent panic attacks was 10.9%. Level of exposure, previous life stressors, and negative emotionality were associated with perievent panic attacks ($\beta = .12$, .15, and .10, respectively), which in turn mediated the relationship between exposure to the terrorist event and panic disorder in the following year. Previous life stressors ($\beta = .15$) and low social support ($\beta = -.14$) were directly associated with panic disorder during the subsequent year. The most vulnerable individuals who experienced perievent panic attacks were 3.7 times, 95% confidence interval [CI] = [2.1, 6.4], more likely to suffer from panic disorder in the following year. Results suggest that early identification of perievent panic attacks following mass trauma may be helpful for reducing panic disorder.

A perievent panic attack (PPA), that is, a panic attack that occurs during or shortly after traumatic exposures, has been largely studied as a vulnerability factor that increases the probability of developing different psychopathological sequelae following mass trauma (Galea, Ahern, et al., 2002), but rarely as a risk factor for panic disorder (PD) in relation to trauma. The risk factors that could lead to a PPA after a terrorist attack together with their direct or indirect influence on PD during the following year have not yet been studied.

A panic attack is an intense and sudden feeling of fear accompanied by four or more spontaneous symptoms (e.g., pounding heart, dizziness, trembling, and nausea) that develop abruptly and reach a peak within approximately 10 minutes. When a panic attack occurs in a recurrent manner and is followed by at least 1 month of persistent concern, excessive worry about the potential consequences of the attack, or a significant behavioral change (e.g., avoidance) in relation to the attacks, an individual may begin to suffer from panic disorder (PD) with or without agoraphobia (American Psychiatric Association [APA], 2000). PD is a highly prevalent, chronic, and disabling condition that adversely affects a person's quality of life and increases health care utilization (Birchall, Brandon, & Taub, 2000).

Lifetime and 12-month prevalence of clinical panic attacks in Europe, according to the European Study of the Epidemiology of Mental Disorders (ESEMeD), a cross-sectional survey of the adult general population in six European countries, including Spain, are estimated to be 10.0% and 3.3%, respectively (Fullana et al., 2011). Lifetime and 12-month prevalence of PD in these European countries are 2.1% and 0.8%, respectively; in the United States, lifetime and 12-month PD prevalence are estimated to be 4.5% (Kessler, Berglund, et al., 2005) and 2.7% (Kessler, Chiu, Demler, Merikangas, & Walters, 2005), respectively.

Stressful life events often precede the onset of panic attacks and PD (Scocco, Barbieri, & Frank, 2007). In terms of severe stress, direct and indirect exposure to mass trauma like the September 11, 2001 (9/11) terrorist attacks has been associated with a higher past-year prevalence of panic attacks (between 16.4% and 23.1% in Pentagon personnel and New York adolescents, respectively; Jordan et al., 2004; Pfefferbaum, Stuber, Galea, & Fairbrother, 2006) and PD in the first years following the events (between 2.5% and 5% in World Trade Center non-rescue vs. rescue, recovery, and cleanup workers, respectively; Cukor et al., 2011; Stellman et al., 2008).

This investigation has been partly carried out under the financial sponsorship of the research project "Anxiety Crises: Prevalence and Vulnerability Factors" funded by the Ministry of Science and Innovation MICINN (ref. PSI 2008-05332).

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DOI: 10.1002/jts.21810

In this context, PPAs have become a focus of research related to predicting the mental health of populations (Galea, Ahern, et al., 2002). For example, some longitudinal studies have found that PPAs are strong predictors for the development of posttraumatic stress disorder (PTSD) and depression (Adams & Boscarino, 2011; Salguero, Cano-Vindel, Iruarrizaga, Fernández-Berrocal, & Galea, 2011). Compared to other mental disorders typically present in the aftermath of terrorism (Boscarino, Adams, & Figley, 2004; Salguero, Fernández-Berrocal, Iruarrizaga, Cano-Vindel, & Galea, 2011), very little attention has been paid to PD. An exception is the longitudinal study of a large cohort of World Trade Center rescue and recovery workers carried out for 9 years; the cumulative incidence of PD was 8.4% in police officers and 21.2% in other rescue and recovery workers (Wisnivesky et al., 2011). Nevertheless, little is known about the effects of large-scale terrorist attacks different from the 9/11 events in the general population. In fact, to our knowledge no longitudinal studies have measured the prevalence and course of PD in the general population after a terrorist attack and its specific association with PPAs and level of exposure to the attacks.

The level of exposure to a terrorist attack has been extensively investigated in relation to psychopathological sequelae and a positive dose-response association has been identified between level of exposure and PPAs (Boscarino, Adams, et al., 2004). Research suggests that other variables may increase the risk of PPAs, such as negative emotionality, previous life stressors, and low social support (Galea, Ahern, et al., 2002; Nixon, Resick, & Griffin, 2004; Pfefferbaum et al., 2006; Stellman et al., 2008).

In this study, we prospectively interviewed a representative sample of Madrid citizens three times over a 12-month period following the March 11, 2004 (M-11) train bombings. Our first aim was to analyze the prevalence of PPAs and PD (6 and 12 months after the bombings). Our second objective was to analyze the association between PPAs and PD in the months following the terrorist attacks. The last of our objectives was to study the role of different variables, especially the level of exposure, that have been closely related with PPAs and PD in trauma research.

After conducting a thorough review of the literature, we hypothesized that the prevalence of PPAs and PD after the train bombings in Madrid would be higher than predisaster estimates. We also constructed a model that includes previous life stressors, negative emotionality, level of exposure to the terrorist attacks, and low social support as relevant variables associated with PPAs and PD (see Figure 1). According to this model, the most vulnerable individuals exposed to the events (i.e., those who experience PPAs) will be more likely to suffer from PD in the following months. In this sense, PPAs mediate the relationship between exposure to the event and PD. Given that previous life stressors, negative emotionality, and low social support have been associated with PPAs and PD, we explored their direct and indirect relationship with these variables in the model.

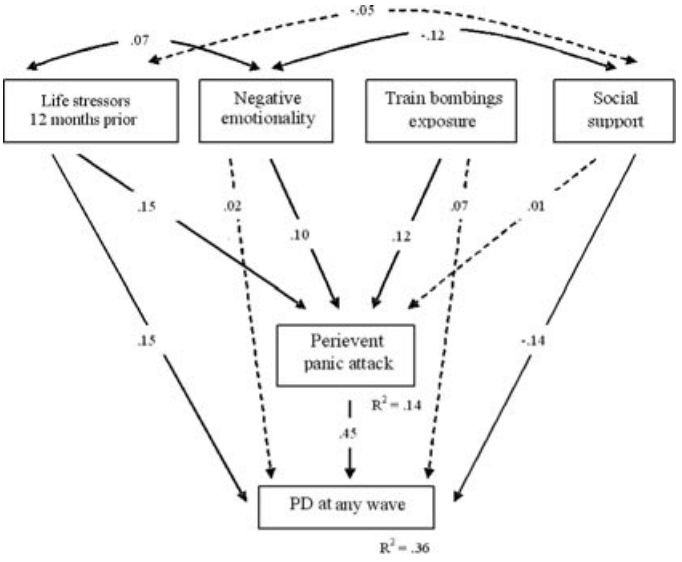


*Figure 1.* Structural equation model for perievent panic attacks and panic disorder (PD) at any wave in the 12 months after the M-11 attacks ($N = 1{,}008$). The $R^2$ shown are pseudo-$R^2$ because of the robust weighted least squares estimator used. They cannot be compared to standard $R^2$. Solid lines represent statistically significant paths; dashed lines represent statistically nonsignificant paths.

## Method

### Participants and Procedure

We carried out a random-digit-dial household survey to recruit baseline respondents living in Madrid approximately 1 month (April 13–June 20, 2004) after the M-11 terrorist attacks. Participants were followed for approximately 6 and 12 months after the event (October 14–December 18, 2004; April 14–June 30, 2005). We interviewed 1,589, 1,192, and 1,008 persons longitudinally in Surveys 1, 2, and 3, respectively. The sampling frame consisted of adults (at least 18 years old) living in households in Madrid with home telephones. Households were screened for eligibility by location. If eligible, up to 10 attempts were made to conduct an interview. One adult per household was randomly selected and was informed that he or she would participate in a survey of a random sample of Madrid city residents regarding their reactions to the M-11 terrorist attacks. The overall response rate until Survey 3 was 38.7%. Compared to the Madrid 2001 Census, there were no differences between our baseline sample or the sample that completed all three waves (hereafter, final sample) in age, sex, and residence distributions (see Miguel-Tobal et al., 2006 and Salguero, Cano-Vindel, et al., 2011 for further details). Table 1 displays the characteristics of the sample and a comparison of the final and excluded samples. Compared to the final sample, dropouts were more likely to have a lower educational level, $\chi^2$ (5, $N = 1589$) = 23.68, $p = .0001$ and less social support, $t$ (1587) = −2.98, $p = .004$; however, no significant differences were found between the final and the excluded samples in sex,

Table 1
*Characteristics of the Sample and Comparison of the Final and Excluded Samples*

| | Baseline sample ($N = 1{,}589$) | Final sample ($n = 1{,}008$) | Excluded sample ($n = 581$) | |
|---|---|---|---|---|
| Characteristics of the sample | % | % | % | $\chi^2$ |
| Perievent panic attack | | | | 2.96 |
| No | 88.3 | 90.3 | 87.1 | |
| Yes | 11.7 | 9.7 | 12.9 | |
| Sex | | | | 3.94 |
| Male | 45.6 | 47.6 | 48.0 | |
| Female | 54.4 | 52.4 | 52.0 | |
| Education | | | | 23.68*** |
| No education | 2.9 | 1.4 | 4.3 | |
| 6–12 years (elementary) | 16.4 | 13.2 | 19.7 | |
| 13–16 (junior high school) | 11.7 | 11.9 | 15.0 | |
| 17–18 (senior high school) | 31.9 | 33.0 | 29.4 | |
| 19–23 (university) | 33.4 | 36.5 | 29.0 | |
| >23 (master's/doctorate) | 3.8 | 4.0 | 2.6 | |
| Annual household income | | | | 4.63 |
| < 12,000 € | 27.6 | 23.4 | 31.5 | |
| 12,001–24,000 € | 35.2 | 34.2 | 33.1 | |
| 24,001–36,000 € | 20.2 | 21.2 | 17.4 | |
| >36,000 € | 17.0 | 21.2 | 18.0 | |
| Life stressors 12 mo prior | | | | 0.19 |
| 0 | 45.8 | 27.4 | 27.1 | |
| 1–2 | 44.3 | 49.0 | 47.6 | |
| 3 or more | 9.9 | 23.6 | 25.3 | |

***$p < .001$.

level of exposure, negative emotionality, life stressors, annual household income, or presence/absence of PPAs. Presence or absence of PD was not included because PD was not assessed in Survey 1. Informed consent was obtained from the respondents and anonymity and confidentiality was guaranteed. The Ethical Committee of the Faculty of Psychology of the Complutense University of Madrid reviewed and approved this study.

## Measures

The survey instrument previously used to assess the psychological sequelae of the 9/11 terrorist attacks in New York (Galea, Ahern, et al., 2002) was specifically modified and adapted for this study to reflect exposure to the M-11 train bombings. The structured interview, administered via telephone by trained interviewers, had an approximate duration of 30 minutes. PPAs and PD were measured using a modified version of the Diagnostic Interview Schedule (DIS) for panic attacks and PD (Centers for Disease Control and Prevention, 1989). The PPA module consisted of 18 items and was phrased to detect symptoms experienced during or shortly after seeing or hearing about the attacks by asking about panic symptomatology "in the first few hours" after the events of M-11 (e.g., "Were you scared?," "Did you have feelings of unreality?," "Did you have shortness of breath?," "Did you feel numbness or tingling sensations?," "Were you scared of dying?"). The two possible answers for each panic attack symptom were yes or no. If a participant reported at least four of the symptoms listed for a panic attack in the the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM-IV-TR*; APA, 2000), he or she was considered to have a diagnosis of a PPA. Cronbach's α for the PPA scale was .76. PD was not assessed until Survey 2 to guarantee that at least 1 month had passed since the attacks. In Surveys 2 and 3, the presence of PD was assessed following *DSM-IV-TR* criteria. Only if respondents had suffered repeated panic attacks during the past 6 months were they asked about at least 1 month of (a) persistent concern about suffering another panic attack, (b) worry about the potential consequences of the attack, and (c) a behavioral change in relation to the attacks. Individuals who answered affirmatively to one or more of these criteria were diagnosed with PD.

We evaluated social support using an abbreviated version of the Medical Outcomes Study Social Support Scale (Sherbourne & Stewart, 1991), which tapped emotional ("having someone to love you and make you feel wanted"), instrumental ("someone to help you if you were confined to bed"), and appraisal

("someone to give you good advice in a crisis") support in the 6 months prior to the Madrid M-11 terrorist attacks (Cronbach's $\alpha = .85$). The interview also included the following domains: demographics (sex, age, educational achievement, annual household income), level of exposure to the attacks (different events that the respondent could have experienced during the terrorist attack, including directly witnessing the events, being injured during the events, having a friend or relative injured or killed, being involved in rescue efforts), recent life stressors (e.g., divorce or separation, family problems, problems at work within the12 months prior to the attacks), and emotional personality traits. Regarding personality traits, we evaluated participants' negative emotionality through a 9-item scale that included three items assessing the tendency to experience anxiety (e.g., "Are you a nervous person?"), three items assessing sadness (e.g., "Do you tend to feel sad?"), and three items assessing anger (e.g., "Are you a hotheaded person?"). Cronbach's $\alpha$ was .81. For further information on the psychometric properties of the rest of the domains, see Boscarino, Galea, et al. (2004); Galea, Boscarino, Resnick, and Vlahov (2003); and Miguel-Tobal et al. (2006).

### Data Analysis

We analyzed the characteristics of our sample in terms of prior demographic characteristics (sex, educational level, and annual household income), stressful events in the past 12 months, social support, level of trauma exposure, and negative emotionality. We also compared the differences in these variables between the final sample and the dropouts. Sampling weights were developed and applied to the data to correct potential selection biases related to the number of household telephones, persons in each household, and oversampling. Poststratification weights were applied to make the survey samples demographically similar to the Madrid population according to the Madrid 2001 Census (Instituto Nacional de Estadística [INE], 2004) to describe the prevalence of panic attacks (in the three waves) and PD at Waves 2 and 3. These analyses were carried out using the SPSS 20 complex samples module.

Mplus version 5 (Muthén & Muthén, 1998) was used to conduct path analysis, a structural equation modeling (SEM) technique that only examines observed variables (Byrne, 1998). Only respondents who completed the three waves were included in this analysis. Robust weighted least squares estimation (WLSMV) was used to specify and test the model because of the combinations of categorical and continuous variables (Flora & Curran, 2004). Data simulation studies indicate that this estimation method produces asymptotically unbiased, consistent, and efficient parameter estimates with ordinal data. WLSMV also corrects $\chi^2$ fit test with dichotomous or ordinal observed variables (Muthén & Satorra, 1995) and is appropriate for use with nonnormal data (Flora & Curran, 2004). In addition to the $\chi^2$ test statistic, overall goodness of fit was evaluated using the following empirically supported fit indices: (a) the comparative fit index (CFI), (b) the Tucker–Lewis index (TLI), (c) the root mean square error (RMSEA), and (d) the weighted root mean square residual (WRMR). Fit index cutoff values for well-fitting models have been suggested as CFI $>$ .95, TLI $>$ .95, RMSEA $<$ .06, WRMR $<$ .90 (Hu & Bentler, 1999; Muthén & Muthén, 1998).

## Results

The prevalence of symptoms indicating a PPA was 10.9%, 95% confidence interval (CI) [8.8, 13.3]. The prevalence of PD during the last 6 months at Waves 2 and 3 was 8.8% and 5.7%. Of those who suffered PPAs, the percentage of PD at Waves 2 and 3 was 26.6% and 13.4%, respectively (i.e., 2.9% and 1.5% of Madrid citizens). Finally, PD at any point during the 12 months following M-11 was 3.7 times higher, 95% CI [2.1, 6.4], in those persons who had versus those who had not experienced a PPA.

### SEM Model of PPAs and PD at Any Wave in the 12 Months After the Attacks

Together with the key variables (life stressors in the 12 months before the attacks, negative emotionality, level of exposure, low social support, PPAs and PD at any wave in the 12 months after the attacks), we included three variables representing demographic status (sex, income, and education level), which were used as covariates. These variables are known as predictors of psychological problems, stressful events, or psychosocial resources (Galea et al., 2004; Schmidt, Kravic, & Ehlert, 2008; Solomon, Gelkopf, & Bleich, 2005). All the variables included in the model were observed variables; thus, our SEM contained nine observed variables. The hypothesized model (see Figure 1) provided a good fit to the data, $\chi^2(3, N = 1{,}008) = 0.89$, $p = .83$, CFI $= 1.00$; TLI $= 1.10$; RMSEA $= 0.001$; WRMR $= 0.14$. The $R^2$ obtained were pseudo-$R^2$ because of the WLSMV estimator used (they cannot be compared to standard $R^2$). Our findings show that the pseudo-$R^2$ for PPAs and PD at any wave in the 12 months after the attacks were .14 and .36, respectively. Figure 1 presents the model with standardized coefficients (for ease of interpretation, covariates were omitted from the figure). Having suffered a PPA was strongly associated with PD at Waves 2 and 3. Other variables that were also related to PD were low social support and life stressors in the year prior to the attacks. With respect to PPAs, participants who in addition to suffering more stressors before the attacks, reported higher levels of negative emotionality and were exposed to a higher level of trauma were more likely as a result to suffer PPAs. Finally, negative emotionality was positively associated with the number of previous stressors and negatively associated with social support.

No direct effects were found between negative emotionality and level of exposure in relation to PD. We inspected the indirect effects of these two variables on PD to examine whether PPAs fully mediated the relation between them and PD. The indirect effect was statistically significant in both cases ($\beta = .05$;

$p = .028$, for negative emotionality; $\beta = .05$; $p = .027$, for level of exposure), indicating that individuals with higher negative emotionality and who were highly exposed to the attacks were more likely to suffer from PD in the year following the attacks, noting that this increase was entirely due to the fact that these participants were also more likely to meet criteria for a PPA.

Further examination of the covariates in the model showed that sex was associated with greater likelihood of PD ($\beta = .16$, $p = .002$). For PPA, educational level lowered the likelihood ($\beta = -.14$, $p = .011$), whereas being female increased the likelihood ($\beta = .22$, $p = .001$) of PAs. All the covariates were associated with social support ($\beta = -.09$, $p = .003$, for sex; $\beta = .12$, $p = .001$, for educational level; $\beta = .07$, $p = .029$, for income). Finally, participants with higher income were less likely to suffer numerous stressors before the attacks ($\beta = -.07$, $p = .018$), and being female and having a lower educational level were associated with higher levels of negative emotionality ($\beta = .07$, $p = .030$; $\beta = -.13$, $p = .001$, respectively).

## Discussion

This investigation is the first longitudinal study carried out in the aftermath of a terrorist attack with a representative and large sample of the population that has analyzed the prevalence of PPAs and PD, the relationship between both and the joint influence of other relevant risk factors for PD.

A growing body of literature suggests that terrorism is a risk factor for the development and chronicity of mental disorders (Stellman et al., 2008) and that experiencing a PPA may be an important indicator of a wide range of psychopathology (Baillie & Rapee, 2005; Marshall, Zvolensky, Sachs-Ericsson, Schmidt, & Bernstein, 2008) including PD (Kinley, Walker, Enns, & Sareen, 2011). One month after the 9/11 terrorist attacks, Galea, Resnick, et al. (2002) found that 12.6% of New York citizens presented PPAs; using the same instrument and time frame, we found that 10.9% of Madrid citizens met the same diagnostic criteria. As we hypothesized, the prevalence of PPAs in Madrid was 3.3 times higher than the 12-month prevalence before the attacks in European population (including Spain; Fullana et al., 2011). The prevalence of PD at Surveys 2 and 3 was also higher (8.8% and 5.7%, respectively) than that found in previous general population surveys carried out in Spain (0.8%; Haro et al., 2006). Although some individuals in the Madrid sample may have already been suffering from PD at the time of the train bombings, the prevalence estimates in Survey 2 still indicated an 11-fold increase compared with predisaster estimates.

Regarding our second objective, we found evidence in favor of a strong association between PPAs and PD. PPAs were significantly associated with PD during the year following the attacks, even after controlling for sociodemographic variables. In addition, PPAs were found to mediate the relationship between level of exposure and PD. Although the level of exposure to the disaster has been recognized in other longitudinal studies as a predictor of PD (Wisnivesky et al., 2011), we found that the level of trauma exposure was only indirectly associated with PD, that is, individuals who were highly exposed to trauma had a higher risk of suffering PD in the year following a terrorist attack probably because they had experienced PPAs.

Other variables that were significantly associated with PPAs in our sample were life stressors before the attacks and negative emotionality, coinciding with the findings of other studies (Coryell, Dindo, Fyer, & Pine, 2006; Moitra et al., 2011). On the other hand, low social support and life stressors in the year prior to the attacks seemed to be associated with PD at Waves 2 and 3, which is also consistent with findings in another study (Batinic, Trajkovic, Duisin, & Nikolic-Balkoski, 2009). Although recent life stressors had a direct effect on PD and an indirect effect through the mediation of PPAs, negative emotionality was only indirectly associated with PD. Last of all, perceived social support was directly related to PD in the long term, but not with PPAs.

In another study carried out with the same sample after the Madrid March 11 train bombings (Salguero, Cano-Vindel, et al., 2011) low social support, negative emotionality, direct exposure to the attacks, recent life stressors, PPAs, and other psychological disorders were identified as risk factors associated with depression. It seems as though all the covariates analyzed in our sample make humans more vulnerable and less capable of coping with a stressful experience without suffering mental disorders like PD, PTSD, and depression. If the presence of other psychological problems (e.g., panic attacks or anxiety disorders) is a risk factor for developing a more prolonged or recurrent depression (Galea, Ahern, et al., 2002; Salguero, Cano-Vindel, et al., 2011), and it is known that anxiety and depressive disorders are often comorbid with each other, it would be of great interest for future investigations to assess different comorbidities (between PD, depression, and PTSD) and analyze how social support, negative emotionality, direct exposure to the attacks, recent life stressors, and PPAs after the M-11 train bombings have an influence on these comorbidities and how these predictor variables are specifically associated with each mental disorder using path analysis. For example, the association we found between PPAs and depression (Salguero, Cano-Vindel, et al., 2011) could be mediated by PD.

Trauma research should also pay more attention to the positive psychological outcomes of mass trauma, such as resilience and posttraumatic growth (Levine, Laufer, Stein, Hamama-Raz, & Solomon, 2009). Just as moderate levels of posttraumatic stress have been found to be associated with the greatest posttraumatic growth (Levine, Laufer, Hamama-Raz, Stein, & Solomon, 2008) and resilience seems to be less common among those individuals who are highly exposed to trauma (Bonanno, Galea, Bucciarelli, & Vlahov, 2006), future investigations should examine whether PD, as well as the other variables analyzed in this study, are positively or negatively associated with resilience and posttraumatic growth.

The present study has a number of limitations including a slight difference in level of social support and educational level between the initial and final sample due to attrition. Second,

there was a lack of information about respondents' psychiatric history prior to the attacks. It could be that some of the participants who reported having experienced a PPA at Wave 1 were already suffering from panic attacks or PD because we did not assess preexisting panic attacks or PD at Wave 1. Third, our assessment was only postevent; hence, we analyzed prevalence, not incidence of disorder. Fourth, the use of self-reports may decrease the validity of our results because of systematic response distortions and memory bias. Last of all, survey nonresponse may also influence inference from the findings shown here. However, the comparability of our sample to expected population demographics and the fact that the response rates obtained in this study are comparable to those in similar work is reassuring in this regard.

Nevertheless, our results could have important practical implications. After the 9/11 events in New York City, there was a significant increase in the prevalence of psychiatric medication use and mental health treatment seeking, especially among those who had suffered PPAs (Boscarino, Adams, et al., 2004; Boscarino, Galea, Ahern, Resnick, & Vlahov, 2003); similar results were reported in Madrid (Miguel-Tobal, Cano-Vindel, Iruarrizaga, González, & Galea, 2004). Although we cannot ascertain the precise number of individuals who were already suffering from PD at the time of the disaster, these findings draw attention to the importance of being alert to PPAs as pertinent variables in relation to subsequent or chronic PD. According to our findings, PPAs mediate the relation between exposure to mass trauma and PD. If individuals who suffer PPAs or who present other risk factors are rapidly detected and treated, future mental health complications and chronic psychoactive drug use may be avoided. Hence, community prevention plans could be designed to identify these highly prevalent presentations after terrorist attacks and effective intervention protocols could be implemented to reduce the probability of PD.

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