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To cite this article: Joyce Zhang, Richard Meiser-Stedman, Bobby Jones, Patrick Smith, Tim Dalgleish, Adrian Boyle, Andrea Edwards, Devasena Subramanyam, Clare Dixon, Lysandra Sinclair-Harding, Susanne Schweizer, Jill Newby & Anna McKinnon (2022) Trajectory of post-traumatic stress and depression among children and adolescents following single-incident trauma, *European Journal of Psychotraumatology*, 13:1, 2037906, DOI: [10.1080/20008198.2022.2037906](https://doi.org/10.1080/20008198.2022.2037906)

To link to this article: <https://doi.org/10.1080/20008198.2022.2037906>



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Published online: 28 Feb 2022.



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Trajectory of post-traumatic stress and depression among children and adolescents following single-incident trauma

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ABSTRACT

Objective: Post-traumatic stress disorder and depression have high comorbidity. Understanding their relationship is of clinical and theoretical importance. A comprehensive way to understand post-trauma psychopathology is through symptom trajectories. This study aims to look at the developmental courses of PTSD and depression symptoms and their interrelationship in the initial months post-trauma in children and adolescents.

Methods: Two-hundred-and-seventeen children and adolescents aged between eight and 17 exposed to single-event trauma were included in the study. Post-traumatic stress symptoms (PTSS) and depression symptoms were measured at 2 weeks, 2 months and 9 months, with further psychological variables measured at the 2-week assessment. Group-based trajectory modelling (GBTM) was applied to estimate the latent developmental clusters of the two outcomes. Logistic regression was used to identify predictors associated with high symptom groups.

Results: The GBTM yielded a three-group model for PTSS and a three-group model for depression. PTSS trajectories showed symptoms reduced to a non-clinical level by 9 months for all participants (if they were not already in the non-clinical range): participants were observed to be resilient (42.4%) or recovered within 2 months (35.6%), while 21.9% experienced high level PTSS but recovered by 9 months post-trauma. The depression symptom trajectories predicted a chronic non-recovery group (20.1%) and two mild symptom groups (45.9%, 34.0%). Further analysis showed high synchronicity between PTSS and depression groups. Peri-event panic, negative appraisals, rumination and thought suppression at 2 weeks predicted slow recovery from PTSS. Pre-trauma wellbeing, post-trauma anxiety and negative appraisals predicted chronic depression.

Conclusions: Post-trauma depression was more persistent than PTSS at 9 months in the sampled population. Cognitive appraisal was the shared risk factor to high symptom groups of both PTSS and depression.

Trayectoria del Estrés Postraumático y la Depresión entre Niños y Adolescentes después de un Incidente único de Trauma

Objetivo: El trastorno de estrés postraumático y la depresión tienen una alta comorbilidad. Comprender su relación es de importancia clínica y teórica. Una forma integral de comprender la psicopatología postraumática es a través de las trayectorias de los síntomas. Este estudio tiene como objetivo observar los cursos de desarrollo del TEPT y los síntomas de depresión y su interrelación en los primeros meses posteriores al trauma en niños/ñas y adolescentes.

Métodos: Se incluyeron en el estudio 217 niños/ñas y adolescentes de ocho a diecisiete años expuestos a un evento traumático único. Los síntomas de estrés postraumático (SEPT) y los síntomas de depresión se midieron a las 2 semanas, 2 meses y 9 meses, con otras variables psicológicas medidas en la evaluación de 2 semanas. Se aplicó un modelo de trayectoria basado en grupos (MTBG) para estimar los grupos de desarrollo latentes de los dos resultados. Se utilizó la regresión logística para identificar predictores asociados con grupos de síntomas elevados.

Resultados: El MTBG arrojó un modelo de tres grupos para SEPT y un modelo de tres grupos para depresión. Las trayectorias de SEPT mostraron síntomas reducidos a un nivel no clínico en 9 meses para todos los participantes (si ellos aún no estaban en el rango no clínico): se observó

ARTICLE HISTORY

Received 1 October 2021
Revised 24 January 2022
Accepted 25 January 2022

KEYWORDS

PTSD; depression; comorbidity; trajectory; GBTM; LCGA; computational phenotyping

PALABRAS CLAVE

TEPT; depresión; comorbilidad; trayectoria; MTBG; LCGA; fenotipado computacional

关键词

PTSD; 抑郁; 共病; 轨迹; GBTM; LCGA; 计算表型

HIGHLIGHTS

- GBTM models estimated trajectories of PTS and depression symptoms from 2 weeks to 9 months post-trauma.
- PTSS trajectories were highly consistent with depression trajectories.
- Cognitive appraisal was the shared risk factor to high symptom groups in PTSS and depression.

que los participantes eran resilientes (42,4%) o se recuperaron en 2 meses (35,6%), mientras que el 21,9% experimentó un SEPT de alto nivel pero se recuperó a los 9 meses después del trauma. Las trayectorias de los síntomas de depresión predijeron un grupo crónico de no-recuperación (20,1%) y dos grupos de síntomas leves (45,9%, 34,0%). Un análisis posterior mostró una alta sincronización entre los grupos de SEPT y depresión. El pánico peri-evento, las evaluaciones negativas, la rumiación y la supresión del pensamiento a las 2 semanas predijeron una recuperación lenta del SEPT. El bienestar pre-traumático, la ansiedad post-traumática y las valoraciones negativas predijeron la depresión crónica.

Conclusiones: La depresión post-traumática fue más persistente que el SEPT a los 9 meses en la población muestreada. La evaluación cognitiva fue el factor de riesgo compartido para los grupos de síntomas altos tanto de SEPT como de depresión.

单次创伤后儿童和青少年的创伤后应激和抑郁的轨迹

目的: 创伤后应激障碍和抑郁的共病率较高。了解它们的关系具有临床和理论意义。了解创伤后精神病理学的一种综合方法是通过症状轨迹。本研究旨在研究儿童和青少年创伤后最初几个月内 PTSD 和抑郁症状的发展过程及其相互关系。

方法: 本研究纳入了 217 名遭受单次创伤的 8 至 17 岁儿童和青少年。在 2 周, 2 个月和 9 个月时测量了创伤后应激症状 (PTSS) 和抑郁症状, 并在 2 周评估时测量了进一步的心理变量。应用基于组别的轨迹模型 (GBTM) 来估计两种结果的潜在发展簇。使用逻辑回归来识别与高症状组相关的预测因子。

结果: GBTM 产生了 PTSS 三组模型和抑郁三组模型。PTSS 轨迹显示所有参与者的症状在 9 个月内减少到非临床水平 (如果他们并非已在非临床范围内): 观察到参与者有韧性 (42.4%) 或在 2 个月内恢复 (35.6%), 而 21.9% 的人经历了高水平 PTSS, 但在创伤后 9 个月内恢复。抑郁症状轨迹预测慢性非恢复组 (20.1%) 和两个轻度症状组 (45.9%, 34.0%)。进一步分析显示 PTSS 和抑郁组之间的高度同步性。2 周时的事件相关恐慌, 负性评价, 反刍和思想抑制预测了 PTSS 恢复缓慢。创伤前的幸福感, 创伤后的焦虑和负性评价预测了慢性抑郁。

结论: 在样本人群中, 创伤后抑郁在 9 个月时比 PTSS 更持久。认知评估是 PTSS 和抑郁高症状组的共同风险因素。

1. Introduction

The co-occurrence of post-traumatic stress disorder (PTSD) and depression has been widely observed. Rytwinski, Scur, Feeny, and Youngstrom (2013) reported that the prevalence of PTSD and major depression disorder comorbidity was 52% (95% CI [48, 56]) in adults. Another meta-analysis estimated the prevalence of depression to be 24.2% (95% CI [20.6–28.0]) in trauma-exposed children and adolescents, and the odds ratio of having a depression diagnosis to be 2.6 (95% CI [2.0, 3.3]) for those exposed to trauma, compared with no or only mild trauma exposure (Vibhakar, Allen, Gee, & Meiser-Stedman, 2019). The high rates of co-occurrence across age groups suggest this is an issue of some clinical and theoretical importance.

PTSD-depression comorbidity is known for being associated with more severe impairments in various domains (Cook et al., 2017) and the key question with regard to their relationship has been ‘is depression part of the PTSD symptoms or are they two independent trauma responses?’ Prior studies investigated the question mainly by looking into hazard ratio, prevalence, risk factors and vulnerabilities (Breslau, Davis, Peterson, & Schultz, 2000; Shalev et al., 1998; Spinhoven, Penninx, van Hemert, de Rooij, & Elzinga, 2014). The consensus is that while the two evidently share common risk factors and vulnerabilities, they are viewed as independent diagnoses because post-traumatic depression is beyond a mere

sharing of common symptoms (Jovanovic et al., 2010; Stander, Thomsen, & Highfill-Mcroy, 2014).

Although these early studies helped to understand how PTSD and depression may relate, Stander et al. (2014) pointed out that most of these studies were limited to examination of associations between PTSD and depression at the macro level. They therefore suggested that future research should consider identifying the time-sensitive mechanisms that facilitate and mediate comorbidity. This point of view echoed Bonanno’s (2004) argument that interpretation of post-traumatic responses would only be meaningful when symptoms were considered in their temporal context. This argument was based on the observations that there are a wide range of individual differences in responding to a potentially traumatic event over time. Bonanno further proposed four prototypical trajectory patterns (Bonanno, 2004), namely: resilient, recovery, delayed and chronic trajectories. In PTSD, these patterns are frequently observed despite diversity in the nature of the traumas (Galatzer-Levy, Huang, & Bonanno, 2018). These trajectories are also found in the youth population. Several children studies have reported that a majority of trauma-exposed children and teenagers may experience elevated distress during the acute phase, but many recover (recovery) while some present persistently low (resilient) or high symptoms (chronic) over time (Hong et al., 2014; La Greca et al., 2013; Lauterbach & Armour, 2016; Punamäki,

Palosaari, Diab, Peltonen, & Qouta, 2015). Late onset (delayed) is relatively less reported (Bonde et al., 2021), however, a comprehensive review of the evidence is only available for adult data.

The implication of recognizing individual differences in trajectories is pivotal. If we are able to describe the developmental patterns of symptoms and to explain what causes the large discrepancies between trajectories after similar trauma exposure, we will have a better understanding of post-trauma psychopathology. With the application of trajectory modelling, a technique specially devised to identify latent longitudinal clusters, more studies exploring PTSD and depression trajectories have emerged. For example, deRoon-Cassini, Mancini, Rusch, and Bonanno (2010) conducted a latent class growth analysis (LCGA) study in adult traumatic injury. They reported four PTSD symptom trajectories (low symptom 59%, chronic 22%, delayed 6% and recovering 13%) and four similar depression groups. Overall, 69.7% of participants were in accordance with the assigned PTSD group (e.g. low PTSD and low depression). Further, they found that individuals in the chronic PTSD and depression group were more likely to have been assaulted, had higher levels of anger and less coping self-efficacy.

Taking the same approach, the present study first aimed to look at the natural trajectories of post-traumatic stress symptoms (PTSS) and depression symptoms in children and adolescents by utilizing group-based trajectory modelling (GBTM). GBTM, equivalent to LCGA, has evinced reliable performance in identifying latent developmental clusters in clinical research (Nagin & Odgers, 2010; Twisk & Hoekstra, 2012). The modelling algorithm analyses the overarching symptom changes over multiple time points and classifies each participant into one particular profile group according to probability. Secondly, we were interested to know whether PTSS and depression symptoms develop in synchrony. The examination was carried out using joint trajectory modelling that returns conditional probabilities linking trajectory groups across two respective outcomes (Jones & Nagin, 2007). The results report the probability of being assigned to a group in PTSS and the chance they would be categorized in the same (or a different) group in their depression trajectory.

Following Hong and colleagues' study (Hong et al., 2014), which also comprised children and adolescents who had been exposed to single incident (mainly non-interpersonal injury), we hypothesized that the trajectory modelling for both PTSS and depression would result in a majority falling into either the low symptoms or recovery groups, and only a small group who would be chronically distressed/depressed. Importantly, in addition to classifying trajectory profiles, we also sought

to identify the potential risk factors associated with the high symptom group in comparison to the low symptom group. By evaluating the risks predicting PTSS with those predicting depression, we aimed to reveal shared processes involved in comorbidity.

The putative risk factors chosen in this study were based on the findings from a risk factor meta-analysis for PTSS in children and adolescents (Trickey, Siddaway, Meiser-Stedman, Serpell, & Field, 2012). Their study examined 25 types of risk that included demographic, trauma characteristics, and post-trauma environmental and psychological factors. They concluded that subjective peri-trauma factors and post-event factors, primarily cognitive processes (e.g. thought suppression, blaming others, perceived threat) are likely to have a major role in the onset of PTSD.

Naturally, the following question would be how much these risks are involved in depression. We hypothesized that the role of age and gender in PTSS might differ from depression. Gender and age are not significant risks for PTSD as per the previous study (Meiser-Stedman et al., 2019), whereas depression tends to be more prevalent in older female adolescents among school-age population (Allgood-Merten, Lewinsohn, & Hops, 1990; Saluja et al., 2004; Thapar, Collishaw, Pine, & Thapar, 2012).

We also predicted that cognitive processes could be the common risks for PTSD and depression post-trauma. Maladaptive appraisal and cognitive coping (e.g. rumination, thought suppression) have been found to be robust in maintaining PTSD symptoms (Ehlers & Clark, 2000; Lavi & Solomon, 2005; Meiser-Stedman, Dalgleish, Smith, Yule, & Glucksman, 2007; Stallard & Smith, 2007). Negative cognitive style (e.g. rumination, self-blaming) is also predictive of depression (Alloy et al., 2000, 2006).

In summary, we hypothesized that PTSS and post-trauma depression are two reactions to trauma which follow matching developmental courses and share certain risks. To examine the elements of the relationship, the study used a trajectory modelling approach, where the differences and similarities were compared in three ways: 1) symptom changes in time (trajectories); 2) the synchronicity of the trajectories; and 3) their predictors.

2. Methods

2.1. Participants

The study used longitudinal data collected by the Acute Stress Programme for Children and Teenagers (ASPECTS), a project set up to study acute PTSD among children and adolescents. Two previous studies have focused on the acute time frame at 2 weeks and 2 months (Meiser-Stedman et al., 2017, 2019); these

studies used the extended data collected at 9 months. Participants were consenting child and adolescent attendees (8–17 years) at four emergency departments (EDs) in the East of England following single event trauma between 3 September 2010 and 30 April 2013. The potentially traumatic events included assault, road traffic accident (RTA) and accidental injury. Participants who did not complete the questionnaires at 2 weeks were not included in the present study. Ten cases with high PTSS measurement scores were referred for treatment after T2, and were therefore excluded from T3 data in the study.

2.2. Symptom measures

All symptom and predictors measures used in the study were child-report. The two key variables of the study were the severity of PTSS and depression symptoms after trauma. These were measured using the Child PTSD Symptom Scale (CPSS; Foa, Johnson, Feeny, & Treadwell, 2001) and the Short Mood and Feelings Questionnaire (SMFQ; Angold, Costello, Messer, & Pickles, 1995).

The CPSS is a self-report questionnaire developed to assess PTSS for school-aged children. It is comprised of 24 items that can be divided into two parts. The first 17 items measure the type and frequency of PTSS (mapping directly on to DSM-IV criteria for PTSD), while the other seven items measure the degree of impairment in functioning. It has shown high reliability and validity across various types of trauma (Foa et al., 2001; Gillihan, Aderka, Conklin, Capaldi, & Foa, 2013; Nixon et al., 2013). A score of 16 was considered a clinical cut-off (Nixon et al., 2013).

The SMFQ is a short version (13 items) of the Mood and Feelings Questionnaire, an inventory that measures depressive symptoms in children and adolescents. Each item is rated on a 3-point scale: 'true', 'sometimes true', and 'not true' with respect to their mood and feelings in the past 2 weeks. It has been shown to be an efficient discriminative tool for school-age children (Cheng, Cao, & Su, 2009; Thabrew, Stasiak, Bavin, Frampton, & Merry, 2018) through to late adolescents (Turner, Joinson, Peters, Wiles, & Lewis, 2014). A total score of eight or higher signifies clinical levels of depression (Angold et al., 1995).

2.3. Predictor measures

We considered eight main factors comprised of three domains controlled at the baseline: demographic (age, gender), peri-event panic, and post-trauma cognitive processes (appraisal, adaptive processing, thought suppression, rumination and self-blame). In addition, anxiety is believed to have a bidirectional relation with depression (Jacobson & Newman, 2017), therefore,

post-trauma anxiety was added to our set of putative predictor measures. Last, it is prudent to have pre-trauma emotional wellbeing controlled as the baseline in the model to eliminate the chance of the observed PTSS and depression symptoms being the result of pre-existing mental health difficulties.

The scores of the ten independent variables were mostly derived from measures developed in previous studies (Meiser-Stedman et al., 2019). Pre-trauma emotional well-being was assessed using the adapted 10 items from the Post-traumatic Adjustment Scale (CPAS) (O'Donnell et al., 2008; $\alpha = 0.81$) that indexes anxiety, low mood and anger. Peri-event panic (CPP) was assessed using a 10-item questionnaire addressing the symptoms associated with a panic disorder diagnosis (Meiser-Stedman et al., 2019; $\alpha = .72$). Post-trauma anxiety was assessed using the Spence Children's Anxiety Scale (SCAS; Spence, 1998; $\alpha = 0.91$). Negative trauma-related appraisals were assessed using the Child Post-Traumatic Cognitions Inventory (CPTCI; Meiser-Stedman et al., 2009; $\alpha = .92$), a 25-item self-report designed to assess dysfunctional trauma-related cognitions. Thought suppression (Children's Thought Suppression Questionnaire, CTSQ) and rumination were assessed using five and three questionnaire items from a previous study that examined thought control strategies and rumination in youths with acute stress disorder (Meiser-Stedman et al., 2014; $\alpha = .85$). Adaptive processing, referring to deliberate efforts to mentally clarify what happened in the traumatic event, was assessed using a five-item measure (Children's Adaptive Processing Questionnaire, CAPQ; Meiser-Stedman et al., 2019; $\alpha = .73$). Self-blame, referring to a cognitive process in which a person attributes the stress event to oneself, was assessed using a two-item measure (i.e. 'I made the event happen', 'it was my fault the event happened'; Meiser-Stedman et al., 2019; $\alpha = .90$).

2.4. Procedure

The study was approved by the UK National Research Ethics Service, Cambridgeshire 1 Research Ethics Committee (10/H0304/11). Parents provided informed consent on behalf of their children, and the child or young person's assent was also required for study entry.

ED research nurses reviewed and screened cases of children attending ED. The parents/caregivers of eligible children were initially contacted by letter 2–4 days post-ED attendance. The nurses excluded cases of chronic trauma exposure, intellectual disability, organic brain damage, significant self-harm and not being a fluent English speaker based on clinical records and parents' report at the initial contact. After T2 (the screening phase), participants with elevated symptoms were referred for intervention. At T3 follow-up, those who sought/received counselling or

treatment were documented while the data were collected as usual. As the current study focused on natural trajectory, we decided to exclude data of participants ($n = 10$) who received multiple sessions of an active psychological intervention for PTSD following T2. Children who had other forms of psychological input, such as one session counselling or treatment for other reasons, were still included.

Consenting participants completed self-report questionnaires at 2 weeks (T1), 2 months (T2) and 9 months (T3) via the telephone or online survey. The survey at T1 and T2 comprised the PTSS and depression measures previously described and the 10 risk variables. The 9-month follow-up only included the PTSS and depression measures. Demographic information, nature of the incident, injury severity and medical treatment were obtained from the ED. PTSS and depression symptoms (assessed by the CPSS and SMFQ, respectively) at T1, T2 and T3 were used for trajectory modelling. Predictive variables were all from T1.

2.5. Statistical analysis

The data analysis followed several steps. First, the distribution of CPSS and SMFQ was checked in order to determine the distribution choice for trajectory modelling. We then used the GBTM program 'Proc Traj' to run modelling for CPSS and SMFQ separately to estimate their candidate models. These candidate models were assessed by their Bayesian Information Criterion (BIC) values along with other interpretive criteria so that we could choose a best fit model for each of the two measures. Next, a joint trajectory model was carried out based on the chosen individual models with a dropout option that compensated for missing data. The joint trajectory returned fine-tuned trajectory probability groups as final results, together with the conditional probability that indexed the connection between the CPSS and SMFQ trajectory groups. Finally, we utilized logistic regression analysis to investigate the link between the predictors and the high symptom groups. Details of each step are as follows.

2.5.1. Data analysis software – Proc Traj

Proc Traj is a SAS/STATA procedure developed by Jones, Nagin, and Roeder (2001). It uses a specialized application of finite mixture modelling to estimate trajectories and does not assume a one size-fits-all model for characterizing symptom onset and progression. Beside the basic modelling function, the package has been extended with functions such as dual-trajectory modelling (Jones & Nagin, 2007). Detailed documentation of the Traj procedure can be found at <https://www.andrew.cmu.edu/user/bjones/>.

2.5.2. Distribution estimation

Estimating the distribution of CPSS and SMFQ variables became necessary so that an appropriate modelling option (CNORM vs. ZIP) could be chosen. An R package, 'fitdistrplus' (<https://cran.r-project.org/web/packages/fitdistrplus/index.html>) was employed to ascertain the distribution of the scores of CPSS and SMFQ at T1. Negative exponential distribution was then considered the best fit for both variables across the three time points (see Appendix B). Therefore, ZIP distribution was chosen for the trajectory modelling.

2.5.3. Single modelling

Given the exploratory nature of modelling, there was no guarantee the procedure would find a successful fit and so determining starting values becomes critical (Jones et al., 2001). Single modelling was used to approximate the parameters of the CPSS and SMFQ trajectories separately before embarking on the joint modelling. Based on previous findings in the literature, potential models with three and four groups were tested. A model of two groups was included as baseline for comparison.

For ZIP distribution, Proc Traj's statistical modelling assumes

$$\ln(\lambda_{it}^j) = \beta_0^j + \beta_1^j Time_{it} + \beta_2^j Time_{it}^2 + \beta_3^j Time_{it}^3 + \beta_4^j Time_{it}^4$$

where λ_{it}^j is the event of interest i at time t , given membership in group j , and $Time_{it}$ is the sampling time point at time t lapsed since the event. The model's coefficients – $\beta_0^j, \beta_1^j, \beta_2^j, \beta_3^j$ and β_4^j – determine the shape of the trajectory. Since Proc Traj allows up to four degrees, our strategy was to probe the possible combinations of a group's polynomial order and to find their highest significant ($p < .05$) degree.

2.5.4. Model selection

Once the single modelling was completed, a best-fit model was selected for each of the outcome measures. Although Jones et al. (2001) recommended an algorithm using two times the change of the BIC values of the adjacent models as the criterion, we argued that it is equally important to realize that depending solely on a statistical figure might fail to identify a model that is clinically meaningful and succinct.

2.5.5. Joint modelling

This was the second step of the modelling analysis. It was undertaken to refine the trajectories and to calculate the conditional probability of group membership based on Bayesian theorem, in order to make immediate linkage between the trajectory groups of PTSS and depression. The configurations of the two selected models produced by the previous steps were entered

into the joint modelling function. False convergence warning was given after the first iteration, therefore a fine-tuning was needed. We used the option ‘detail’ to obtain the parameters returned from the first iteration. We removed insignificant parameters ($p > .05$) and entered the rest into a second iteration as starting values (see complete STATA script in supplementary material Appendix C). The program adjusted well and the model was finalized.

2.5.6. Dropout analysis

Attrition has been a challenge for longitudinal studies and where data is missing careful handling is required or there will be a high risk of bias in the results yielded. Strategies for handling missing data may depend on whether the data are missing completely at random (MCAR), missing at random (MAR) or missing not at random (MNAR). In practice, the difference between MCAR, MAR and MNAR is often too elusive to ascertain (Graham, 2009).

There was an attrition rate of approximately one-third at nine months in our study and the randomness of the missing data was hard to estimate. Fortunately, Haviland, Jones, and Nagin (2011) have extended the Proc Traj package with a dropout option. They demonstrated that non-random attrition, in which the dropout rate is uneven across the latent groups, has a consequential impact on modelling the group size. In an extreme case, a group might completely diminish. They further illustrated that the dropout extension was successfully able to optimize the model by taking account of different dropout rates. Thus, we adopted the dropout option in modelling to estimate a better model and offer informative judgement on the missing data.

2.6. Predictive factor analysis

After the joint modelling, each case was categorized into one PTSS group and one depression group according to the course of their symptoms over the nine months. Membership profiles were labelled as low, medium or high. A multinomial logistic regression analysis was then deployed to calculate the relative risk ratio of falling into the high symptom trajectory group compared to the low symptom trajectory group according to predictor variables of age and gender, measures of pre-trauma emotional well-being (CPAS), peri-trauma panic (CPP), post-trauma anxiety (SCAS), appraisal (CPTCI), rumination, thought suppression and adaptive-processing and self-blaming.

Trauma severity, a common factor that may influence the post-trauma response, was not included in the modelling. The previous study using the same sample revealed that objective indices of trauma severity (number of injuries, sustaining a fracture, being seen in

resuscitation, sustaining an injury with permanent loss of function) were not significantly related to PTSS (Meiser-Stedman et al., 2019). That study suggested, however, that the cognitive processes (peritraumatic panic, post-traumatic rumination, negative appraisals and adaptive processing) played an important role in the onset and maintenance of PTSS; thus, the present study focused on examining the impact of the cognitive elements. Regression analysis also confirmed that trauma severity was not associated with depression (SMFQ) scores; model outputs are listed in supplementary material Table S3.

3. Results

3.1. Descriptive statistics

The data analysis included 217 participants, of whom 124 were males and 93 were females. Participants were aged between 8.01 and 17.97 years ($M = 14.09$, $SD = 2.96$). The traumatic events that participants had been exposed to were RTA ($n = 98$), accidental injury ($n = 71$), assault ($n = 35$), dog attacks ($n = 11$) and other acute medical emergencies ($n = 2$). At T2, there were 13 participants missing CPSS scores and 14 cases missing SMFQ scores. At T3, 58 cases missed both CPSS and SMFQ scores.

3.2. Model selection

As illustrated in supplementary Table S1, for the PTSS (CPSS) trajectories, the BIC criterion favoured models comprising four groups; for depression symptoms (SMFQ) there was no significant difference between the three and four group options (i.e. the BIC difference was less than 10). Figure 1 presents the two proposed models: three groups vs four groups (see Figure 1a for CPSS and Figure 1b for SMFQ). The two models (3-group model vs. 4-group model) were similar in some key regards – they both encompassed a consistently low score group and high score group with broadly equivalent group size (29.3% vs. 26.1% for the low score group and 21.1% vs. 17.1% for the high score group in PTSS; 19.6% vs. 17.6% for the high score group in depression).

The main difference between the three- and four-group models concerned the medium groups. The three-groups model recommended one medium-severity group, whereas two separate groups were proposed by the four-group models. We favoured the more succinct three-group model for several reasons. First, in those two groups, the starting point at T1 of one group is higher than the other and almost reaches the cut-offs for each outcome (16 for CPSS and eight for SMFQ). Although subthreshold symptoms can be of a potential concern, treatment usually will only be considered when symptoms last more than 1 month

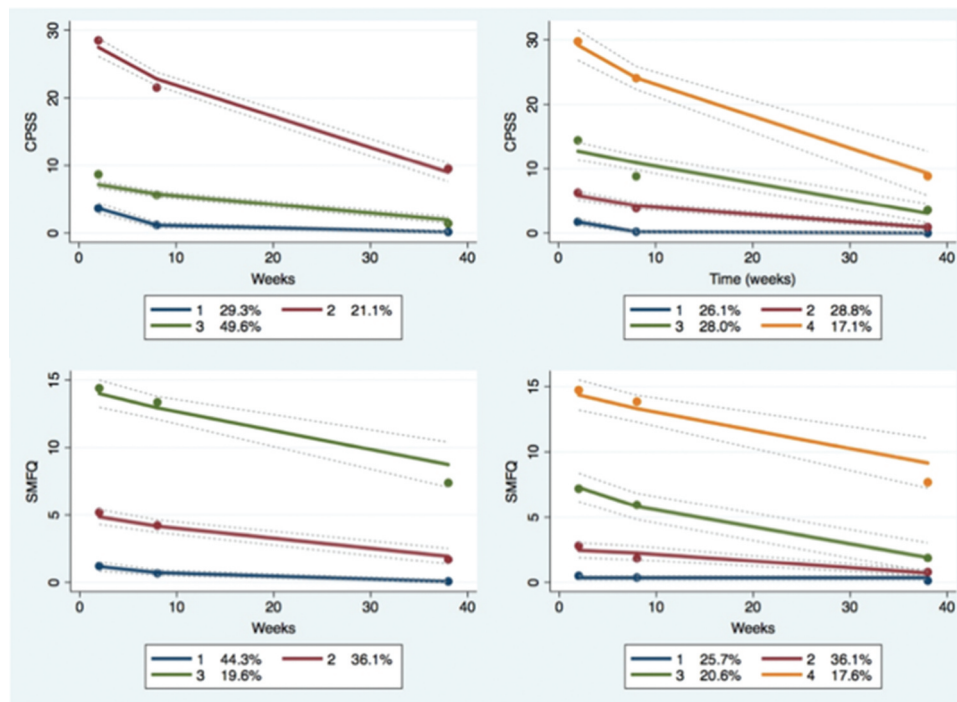


Figure 1. A) Candidate models of CPSS. B) Candidate models of SMFQ.

(NICE, 2018). Since both symptoms dropped further at T2, it will make no material difference between the two middle groups in terms of clinical management. Second, the four-group models also had larger confidence intervals (CI: areas between the dashed lines). Third, the more parsimonious models would be beneficial when it came to joint modelling because, when linking the outcomes of the two trajectories, the proliferation of probability matrices could easily become unmanageable. Specifically, the four PTSS and four depression groups produce 16 combinations while the three-group option only produces nine. Those seven extra combinations are the hybrids from the medium groups, which can be expected to be almost identical.

3.3. CPSS & SMFQ joint modelling

The shape of the trajectory of each group is determined by a vector of coefficients ($\beta_0^j, \beta_1^j, \beta_2^j, \beta_3^j$ and β_4^j). Our model attained significant ($p < .00001$) coefficients for all trajectory groups and drop out polynomials (see complete output in Supplementary Table S1). Conditional as well as joint membership probabilities have been reported.

3.4. PTSS trajectory

The final joint model (Figure 2) yielded three distinct PTSS trajectory probability groups including a low symptom group (42.4% of the sample size) with

persistently low CPSS scores, a group (35.6%) with marginally significant CPSS score at week 2 which dropped below the clinical cut-off at 2 months, and a high symptom group (21.9%) presenting marked distress at 2 weeks and 2 months. At 9 months, the scores of the three groups were all in the non-clinical range.

3.5. Depression trajectory

Similarly, the joint model produced three depression trajectory groups (low, medium and high) comprising 45.9%, 34.0% and 20.1% of the participants, respectively. In contrast to the low and medium groups, whose depression level remained persistently low, the SMFQ score of the high depression group at nine months ($M = 7.96$, 95% CI [7.32, 11.17]) was still around the clinical cut-off.

3.6. Conditional group membership

In probability theory, conditional probability is a measure of the probability of an event occurring given that another event has occurred. If we knew a case was categorized as high PTSS, the probability of its belonging to the low, medium and high depression symptom trajectory groups would be 1.6%, 8.3% and 74.4%, respectively. Conversely, the probability of belonging to the low, medium and high PTSS groups conditional on membership of a high depression group would be 2.5%, 13.1% and 81.8%, respectively.

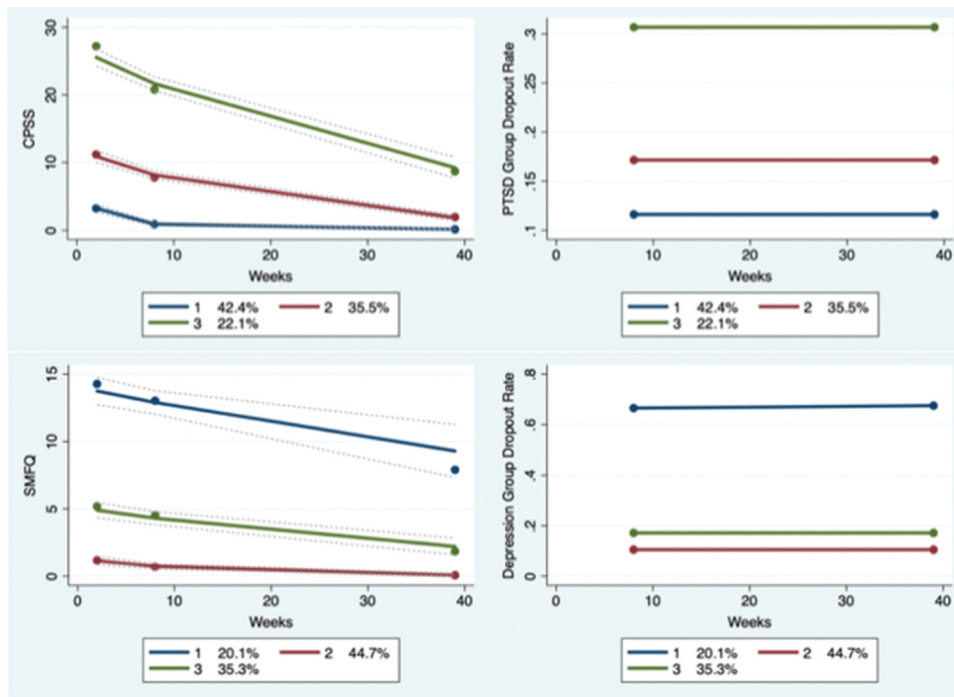


Figure 2. A) Final trajectory and dropout model of CPSS. B) Final trajectory and dropout model of SMFQ.

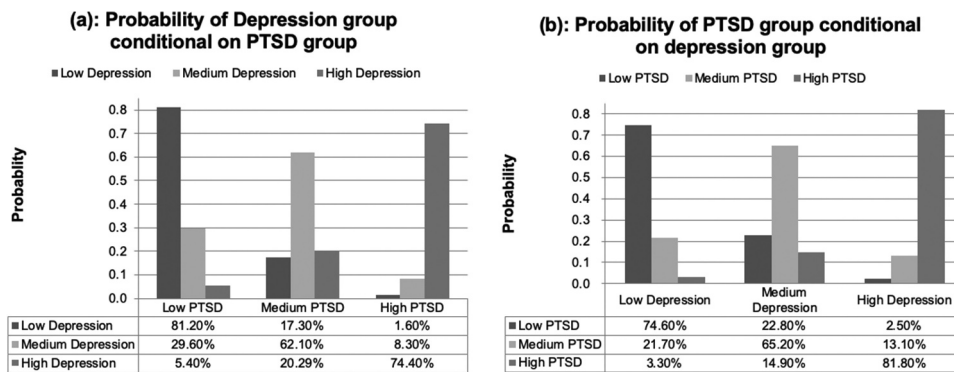


Figure 3. A) Conditional probability of depression if PTSD is known. B) Conditional probability of PTSD if depression is known.

Figure 3 lists the conditional probabilities of all the possible combinations. In addition, the model also reported the joint probability of belonging to the PTSD group and the depression group (Table 1c).

3.7. Dropout model

Dropout model explains the heterogeneity in the dropout pattern within each trajectory group. It also describes the change of attrition rate across the time using the sample size at T1 as baseline. A flat linear model was proposed suggesting an equal rate of attrition at T2 and T3. Estimated dropout rates for the low, medium and high PTSD groups were 14.1%, 15.5%, 30.2%, respectively. Likewise, estimation for the depression groups were 16.3%, 11.9% and 65.1%, respectively. The models indicated that the higher symptoms a child had, either in PTSD or depression, more likely they would dropout.

For the record, the actual dropout rates at T2 were: 3.16%, 3.47% and 6.77% for PTSS groups, and 3.93%, 2.87% and 15.7% for depression groups. At T3, the rates were: 14.9%, 16.2%, 36.7% for PTSS groups, and 11.3%, 10.9% and 67.8% for depression groups.

3.8. Predictive factors

Using low symptom groups as the referent, gender, age, pre-trauma emotional wellbeing, peri-event panic, post-trauma anxiety, trauma-related appraisal, rumination, thought suppression, adaptive processing and self-blaming were entered as independent variables into two multinomial logistic regressions (see STATA scripts in supplementary material Appendix D) to predict the PTSD and depression trajectory outcome, in particular for high symptom groups. The relative risk ratio in the model estimates that for one unit increase in each of the predictive factors the

Table 1. Parameters of trajectory groups and joint probability.

1a: PTSD trajectory groups					
	Group	Mean	Est. Mean	95%CI	Dropout%
2 weeks	Low	3.243	3.238	[2.703, 3.773]	-
	Medium	11.125	10.873	[9.987, 11.759]	-
	High	27.582	25.789	[24.529, 27.048]	-
8 weeks	Low	.879	.875	[.607, 1.142]	14.1%
	Medium	7.780	8.096	[7.498, 8.695]	15.5%
	High	20.824	21.746	[20.742, 22.750]	30.2%
9 months	Low	.127	.127	[0, .258]	14.1%
	Medium	1.921	1.854	[1.467, 2.241]	15.5%
	High	8.709	9.271	[7.718, 10.825]	30.2%
1b: Depression trajectory groups					
	Group	Mean	Est. Mean	95%CI	Dropout%
2 weeks	Low	1.230	1.231	[.9152, 1.546]	-
	Medium	5.220	4.998	[4.438, 5.559]	-
	High	14.351	13.831	[12.762, 14.899]	-
8 weeks	Low	.702	.753	[.5677, .938]	16.3%
	Medium	4.599	4.375	[3.898, 4.852]	11.9%
	High	12.936	12.933	[12.021, 13.845]	65.1%
9 months	Low	.068	.0645	[0, .185]	16.3%
	Medium	1.848	2.248	[1.625, 2.871]	11.9%
	High	7.960	9.246	[7.321, 11.171]	65.1%
1c: Joint probability of combined membership					
		Depression Group			
		Low	Medium	High	
PTSD Group	Low	34.5%	7.3%	0.7%	
	Medium	10.5%	22.1%	3.0%	
	High	1.2%	4.4%	16.3%	

change in the probability of falling into the high symptom group rather than the low symptom group, given that the other variables in the model are held constant.

The statistically significant predictors ($p < .05$), ordered from strongest to weakest, for the high PTSS trajectory group were: peri-event panic (RR = 2.09, 95% CI [1.38, 3.19]), rumination (RR = 1.60, 95% CI [1.07, 2.39]), thought suppression (RR = 1.27, 95% CI [1.04, 1.56]) and negative appraisals (RR = 1.26, 95% CI [1.12, 1.42]). The statistically significant predictors for the high depression trajectory group (also from strongest to weakest) were: negative appraisals (RR = 1.31, 95% CI [1.15, 1.48]), pre-trauma emotional wellbeing (RR = 1.24, 95%CI [1.03, 1.49]), and post-trauma anxiety (RR = 1.20, 95% CI [1.09, 1.34]). Gender and age did not predict PTSS or depression (Table 2).

4. Discussion

The study investigated the natural recovery trajectories of PTSS and depression symptoms for the 9 months period following a single event trauma. Overall, our model suggested that PTSS reduced to non-clinical level for all participants by 9-months. The PTSS trajectories finding was consonant with the prototypical trauma response pattern proposed in 2004 (Bonanno, 2004), although no delayed onset cluster was detected in our sample. The majority (80%) were observed to be consistently displaying low symptoms or able to recover within two months. About one-fifth

of participants experienced high levels of PTSS but managed to reach the recovery range within 9 months. Unlike the other non-interpersonal one-time trauma studies in youth (Hong et al., 2014; La Greca et al., 2013; Punamäki et al., 2015), there was no chronic/increase group.

Another possible reason for the absent chronic group could be that participants with elevated PTSS were referred to intervention and were excluded from the study. It is difficult to be certain how different the PTSS trajectory groups would be if the data of children who received treatment had been included into the modelling. Since their PTSD symptoms were expected to drop at T2 and T3 after treatment, they would likely be merged into the high symptom trajectory group. Meanwhile, we also postulate that in the less ethical counterfactual situation where no intervention is offered, the 10 cases would form a fourth group with a PTSS level higher than the current high symptom group at T1 and possibly with symptoms continuing to deteriorate over T2 and T3. This hypothesis is based on the shared characteristics of the chronic/increase group reported by two similar injury studies (Hong et al., 2014; Punamäki et al., 2015). The shared characteristics were: that the group made up a very small portion (1.8% and 12%); that the initial symptom level at the acute phase was the highest among all groups; and that there was no natural recovery even after periods as long as 30 (Hong et al., 2014) and 11 months (Punamäki et al., 2015). Alternatively, these cases

Table 2. Multinomial logistic regression analysis.

2a: Predictors of high PTSD group using low PTSD symptom group as referent					
Log likelihood = -112.91091			# of obs: 214 LR chi2 (20) = 228.84 Prob > chi2 = 0.000 Pseudo R2 = 0.5033		
	RRR	Std. Err.	Z	P > Z	[95% Conf. Interval]
Age	1.66	1.31	0.64	0.520	[.36, 7.73]
Gender	1.05	.14	0.38	0.704	[.81, 1.37]
Pre-trauma emotional wellbeing	.92	.09	-0.93	0.354	[.76, 1.10]
Peri-trauma panic*	2.09	.45	3.47	0.001	[1.38, 3.19]
Post-trauma anxiety	1.07	.05	1.33	0.182	[.97, 1.17]
Cognitive appraisal*	1.26	.08	3.86	0.000	[1.12, 1.42]
Rumination*	1.61	.33	2.32	0.020	[1.08, 2.39]
Thought suppression*	1.27	.13	2.35	0.019	[1.04, 1.56]
Adaptive processing	.85	.09	-1.47	0.140	[.68, 1.06]
Self blame	.68	.14	-1.83	0.068	[.45, 1.03]
_cons	6.34e-09	2.42e-08	-4.94	0.000	[3.54e-12, .0000114]
2b: Predictors of high depression group using low depression symptom group as referent					
Log likelihood = -110.86479			# of obs: 214 LR chi2 (20) = 225.83 Prob > chi2 = 0.000 Pseudo R2 = 0.5046		
	RRR	Std. Err.	Z	P > Z	[95% Conf. Interval]
Gender	.95	.74	0.07	0.947	[.21, 4.34]
Age	1.30	.18	1.89	0.059	[.99, 1.71]
Pre-trauma emotional wellbeing*	1.24	.12	2.24	0.025	[1.03, 1.49]
Peri-trauma panic	.98	.18	-0.09	0.930	[.68, 1.42]
Post-trauma anxiety*	1.21	.06	3.62	0.000	[1.09, 1.34]
Cognitive appraisal*	1.31	.08	4.22	0.000	[1.15, 1.48]
Rumination	1.28	.24	1.30	0.194	[.88, 1.86]
Thought suppression	1.06	.12	0.61	0.542	[.87, 1.30]
Adaptive processing	.87	.09	-1.20	0.229	[.70, 1.09]
Self blame	1.34	.25	1.54	0.123	[.92, 1.93]
_cons	2.30e-13	1.05e-12	-6.38	0.000	[3.03e-17, 1.75e-09]

RRR: relative risk ratio. * $p < 0.05$.

Note: _cons estimates baseline relative risk for each outcome.

may have increased the predicted depression score of the high symptom group at the T3 assessment.

The depression trajectories were quite different. The three trajectories all described a steady decline but the divergence between the high depression group (20%) and the rest was such that the high depression trajectory group were more likely to have persistently high depression symptoms for nine months, during which time the other two groups demonstrated only mild symptoms. Such a dichotomous pattern has not been apparent in previous trajectory studies in paediatric populations.

In respect of the relationship between the PTSS and depression trajectories, the conditional membership analysis reported high synchronicity: low PTSS participants were highly likely to be classified in the low depression group, while a participant who experienced high PTSS was anticipated to be in the highly depressed group. Similarly, being in the high depression group predicted being in the more severe PTSS group. The finding is consistent with previous studies in injured adults (deRoon-Cassini et al., 2010) and children (Hong et al., 2014). Given that PTSD-depression comorbidity is well established, this finding is not surprising. However, trajectory is a temporal concept and it addresses the dynamic of symptom

change. The synchrony between the two trajectories following the same stressor has more profound implications than a simple indication of symptoms overlapping at some time point. It is reasonable to hypothesize that if PTSS and depression evolve in similar patterns, there should be either a common mechanism underlying their development, or there is/are shared factor(s) driving the mechanisms that determine the symptoms.

The high PTSS and depression trajectory groups shared few predictive factors. Rumination in general is considered a transdiagnostic feature associated with depression and PTSD, and it was strongly related to PTSS in this study although it did not predict depression in our model. This phenomenon suggests that a certain subtype(s) of rumination may maintain PTSD but not depression. Birrer and Michael (2011) conducted a study examining the characteristics of rumination such as duration and content in PTSD and depression; they found that rumination served as a powerful internal trigger for intrusive memories in PTSD, but not in depression. Constructions of various types of rumination (e.g. depressive rumination, stress-reactive rumination) have been suggested and their clinical impact needs further investigation.

In line with the literature, peri-trauma panic (perceived threat), thought suppression and negative appraisal were linked with high PTSS, while only negative appraisal was a factor that was associated with both high symptom groups. This finding confirms that negative appraisal plays a role in maintaining broader post-trauma psychopathology (Hiller et al., 2019). Hamilton et al. (2012) integrated findings from a large body of neuroimaging research and proposed that depression is sustained by the increased salience of negative information leading to biased appraisal. Combined with the heightened sense of threat (e.g. intrusive memory, hypervigilance) in PTSD, which serves as an ongoing source of negative information, appraisal may be central to understanding PTSD-depression comorbidity. The negative appraisals that are proposed to play a major role in the maintenance of PTSD (Ehlers & Clark, 2000) also help to maintain depression.

In summarizing the PTSS-depression relationship observed in the study, we concluded that PTSS and depression are two distinct, but overlapping, responses to a traumatic stressor, and that they are maintained by different processes. This conclusion is based on 1) the high synchronicity in their trajectories, and 2) few mutual predictors. Negative appraisals appeared to play a critical role bridging their mechanisms. Anxiety manifested as the second strongest predictor of depression trajectories. This may be a byproduct of the overlapping presentations of physiological arousal and avoidance in both anxiety disorders and PTSD.

Lastly, our study is the first to examine trajectory and attrition rate in the field and found that the more severe symptoms a participant has, the more likely they will drop out from the study. We hypothesize that this may hold universally in longitudinal research and clinical trials, and the consequences can be serious. The immediate consequence is that, without correction, the averages of the examined measures will be lower than their real means, and other prime parameters of the sampled distribution, such as standard deviation, will be altered. This may make inferred statistical interpretation less accurate. Therefore, this conclusion supports handling missing data with great caution and, if possible, applying appropriate statistical methods (e.g. dropout modelling or imputation) to minimize the impact.

4.1. Clinical implication

In the case of acute post-traumatic psychopathology, depression may be a more lasting condition than PTSD symptoms. In our sample, PTSS tended to diminish over time, whereas depression often persisted. This supports the routine screening of trauma-exposed children and adolescents for depression. Similarly, depression should be included in any

consideration of core post-traumatic symptoms when making clinical decisions such as active monitoring or offering an intervention.

There was a clear correlation between high PTSS and high depression symptoms. Although this study was limited to the non-clinic-referred group (i.e. participants receiving multiple-session interventions were excluded), this relationship is likely to hold in the clinical population given the findings from other studies. This means that patients seeking treatment for PTSD are prone to high levels of depression. Effective intervention should incorporate components addressing both PTSD and depression.

Most importantly, negative appraisal was the only predictor for both high PTSS as well as high depression symptom trajectories; this suggests a possible effective treatment approach, addressing PTSS and depression holistically by focusing on negative appraisal.

4.2. Limitations

The study had several discernible limitations. First of all, the data were limited to children and adolescents, mainly following a one-off, mostly non-interpersonal trauma. Thus, the interpretation of the results may not apply to interpersonal or multiple traumas. Second, for ethical reasons, the dataset was only able to track the natural course of participants with relatively mild symptoms. Ten cases with high PTSS measurement scores were referred for treatment and were therefore excluded from the study. The trajectories that emerged in this study may, therefore, not represent the clinical population. Third, the drop out model predicted equal dropout rates at T2 and T3, which did not fit the actual data perfectly (the missing rate at T2 was much lower (13/14 cases) than at T3 (58 cases)). Consequently, the estimated means at T2 could be higher than their true values as the joint modelling compensates for the missing data by applying the high score, high dropout formulation.

5. Conclusion

Within children and adolescents exposed to single event trauma resulting in minor physical injury, the majority were able to recover without intervention over the following months, although about one-fifth presented with symptoms of lasting depression at 9-month follow-up. PTSS trajectory groups are in high accordance with depression trajectory groups. By examining predictors of high symptom groups, negative appraisals appeared to be a shared risk factor to PTSS and depression.

Disclosure statement

No potential conflict of interest was reported by the authors.

Funding

This research received no specific grant from any funding agency, commercial or not-for-profit sectors.

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Data availability statement

The authors confirm that the data supporting the findings of this study are available within the article and its supplementary materials (also available at <http://c2ad.mrc-cbu.cam.ac.uk/projectsold/aspects/>). This study used existing data acquired by a prior study, no institutional review board approval or patient consent was needed.

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