

Short- and long-term incidence of obsessive-compulsive disorder after objectively recorded potentially traumatic events

Corresponding Author: Dr Josep Pol-Fuster

Parts of this Peer Review File have been redacted as indicated to maintain the confidentiality of unpublished data.

This file contains all reviewer reports in order by version, followed by all author rebuttals in order by version.

Version 0:

Reviewer comments:

Reviewer #1

(Remarks to the Author)

This innovative study sought to evaluate the causal link between potentially traumatic events and OCD in a large sample of 3 million Swedish participants born between 1975 and 2008. The authors found a robust association with assault/victimization exposure, particularly within the first year after exposure, and found no association with transportation accidents. Importantly, the association between assault/victimization exposure and OCD persisted in the presence of genetic influence, suggesting a unique causal pathway not entirely explained by genetics. As the authors explain in their work, existing research has relied heavily on retrospective self-report and/or cross-sectional designs, and few have included genetically informed designs, so although a connection between trauma exposure (particularly those interpersonal in nature) and OCD incidence has been inferred, no study yet has been able to evaluate this direct link. Thus, this represents a monumental study with groundbreaking findings, and I commend the authors highly on their efforts. The study and analytic methodology are robust, offering great confidence in the study's findings. The writing quality is clear, transparent, and follows an intuitive flow. I have only minor recommendations to improve upon this work, and I look forward to seeing it in print:

- Can the authors say more about their decision to focus solely on assault/victimization and transportation accidents? Were there other trauma types available in the data and, if so, why did the authors select only these two? More transparency and clarity on this point would be helpful, particularly in light of research that other trauma types may also uniquely relate to OCD/OC symptoms.
- Are there any subsequent post-hoc analyses that can be run to offer insight into time-to-onset of OCD following trauma exposure? Certainly there is an increased incidence of OCD within 12 months of violence exposure, however if the data allows, it would be interesting to know more precisely when the diagnosis was documented within this timeframe. This would address another existing gap in the literature, which is understanding exactly when OCD is likely to develop following trauma exposure (e.g., within weeks, within months, etc.).
- On pg. 8, the authors state: "This implies that evocative gene-environment correlations could be a potential mechanism, whereby genetically vulnerable individuals may be more likely to enter high risk situations where they may be exposed to interpersonal violence." As this is an important point, it would be helpful if the authors could say more here regarding the ways in which genetics can increase someone's risk for trauma exposure.

Reviewer #2

(Remarks to the Author)

The authors have prepared a well-written manuscript that utilizes population-based data to determine the relation between specific potentially traumatic events and incidence of OCD. The manuscript answers a unique question using original methodology. Critically, much of the work in this area has not utilized prospective data or merely demonstrates associations, which limits causal inference. The authors have utilized prospective methods to uncover this causal link.

There are several suggestions for improvement included below:

- 1) Please include information about the incidence and prevalence of OCD. It would be helpful to contextualize these statistics within the population of interest here, as well as worldwide, if available.
- 2) Because the authors discuss genetics and heritability, it may be helpful to review further literature in this area to address common genetic pathways for internalizing psychopathology and the specificity of genes that have been linked to OCD.
- 3) It would be helpful to include brief background on the context of reporting violent and sexual assaults in the context of Sweden. Are these screened for regularly in health clinics? Would patients have to bring this up on their own? If people would have to bring these events up on their own, is it possible that some siblings would be more likely to disclose to their doctor than others? How reliable is this variable if not all individuals are screened at each visit?
- 4) The authors note that the statistical power of the quantitative genetic modeling is limited. I think this necessitates more information so that readers can interpret with the appropriate amount of caution. How limited was power? Is this something that should result in severe caution when interpreting, or is it the case that these effects are notable because they were found when they were likely underpowered? If an a priori power analysis was conducted, including a sentence or two, in comparison to the sample at hand, would be most helpful.
- 5) The observed findings related to interpersonal, but not transport, trauma aligns with a large body of literature. This literature should be further explored to provide broader context for the findings and how they fit into the literature.
- 6) The authors write: "Interestingly, the familial co-aggregation and quantitative genetic modeling re-198 sults suggested that the association between assault/victimization and OCD was largely 199 attributable to shared genetic factors. This implies that evocative gene-environment cor-200 relations could be a potential mechanism, whereby genetically vulnerable individuals 201 may be more likely to enter high risk situations where they may be exposed to interper-202 sonal violence." Perhaps I am misunderstanding, but this interpretation did not make sense to me. The results indicate that the relation between victimization and OCD may depend heavily on genetic factors. This means that those who have a genetic propensity, combined with the occurrence of this environmental event, are more likely to be diagnosed with OCD. The genetic vulnerability to enter high risk situations seems like a reach given the findings, and it also runs the risk of reading as blaming. I would suggest rephrasing to stick to the findings at hand and then note that future research should explore the genetic/environmental determinants of victimization specifically.
- 7) I would note somewhere in the discussion that this analysis only includes those who chose to seek health care, share with their doctor about life events, and share their symptoms. If individuals never seek healthcare, they would not be included.
- 8) Are there suggested intervals in which Swedish individuals follow with providers? Are annual appointments required/optional/suggested? It would be helpful to know the extent to which Swedish individuals are screened, seen in clinic, or if this is entirely based on patient discretion.
- 9) Figure 1 would benefit from a caption to help to explain to readers how to interpret the graphs.
- 10) Analytic approach could be described in more detail to help increase replicability and validity of results.

Version 1:

Reviewer comments:

Reviewer #1

(Remarks to the Author)

I thank the authors for their thorough response to Reviewer and Editorial Office feedback. Their revisions have improved the transparency and clarity of this work. I am particularly grateful to the authors for conducting the requested post-hoc analysis regarding time-to-onset of OCD. While underpowered (and appropriately couched as such), these data offer important preliminary information about when trauma-exposed individuals may be at heightened risk for developing OCD, which could have a downstream effect on better powered empirical inquiry and more precise intervention efforts. I have no further feedback and am grateful to the authors for their commendable efforts to move this literature forward in a major way. Congratulations!

Sincerely,
Caitlin M. Pinciotti, PhD
Baylor College of Medicine

Reviewer #2

(Remarks to the Author)

Many thanks to the authors for the thoughtful and thorough revision. In particular, thank you for further describing the analytic approach.

The requested areas for evaluation are described below.

- A. Summary of key results - the authors have adequately described the results in detail, and the additional findings added to the manuscript in the revision are interesting findings for future research!
- B. Originality and significance - the authors added information about the prevalence of OCD, which speaks to the significance. I appreciate the originality of this design in investigating causal inference.
- C. Data and methodology - additional information provided in this revision was very helpful.
- D. Appropriate use of statistics and treatment of uncertainties - the uncertainties are better described, and statistics were

used appropriately.

E. Conclusions - conclusions were robust. There may be concerns with the validity of report of traumatic experiences, as the authors noted in the response letter. Those who experience significant injury are most likely to report or seek care. This is likely an underestimate of the true prevalence.

F. Suggested improvements - the authors have improved the manuscript in this revision, and I do not have further recommendations.

G. References - appropriate.

H. Clarity and context - Appreciate the additional context that was added, and this clarified all of my questions.

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Reviewers' Comments:**Reviewer #1 (Remarks to the Author):**

This innovative study sought to evaluate the causal link between potentially traumatic events and OCD in a large sample of 3 million Swedish participants born between 1975 and 2008. The authors found a robust association with assault/victimization exposure, particularly within the first year after exposure, and found no association with transportation accidents. Importantly, the association between assault/victimization exposure and OCD persisted in the presence of genetic influence, suggesting a unique causal pathway not entirely explained by genetics. As the authors explain in their work, existing research has relied heavily on retrospective self-report and/or cross-sectional designs, and few have included genetically informed designs, so although a connection between trauma exposure (particularly those interpersonal in nature) and OCD incidence has been inferred, no study yet has been able to evaluate this direct link. Thus, this represents a monumental study with groundbreaking findings, and I commend the authors highly on their efforts. The study and analytic methodology are robust, offering great confidence in the study's findings. The writing quality is clear, transparent, and follows an intuitive flow. I have only minor recommendations to improve upon this work, and I look forward to seeing it in print:

[We thank the Reviewer for the positive appraisal of our manuscript.](#)

- Can the authors say more about their decision to focus solely on assault/victimization and transportation accidents? Were there other trauma types available in the data and, if so, why did the authors select only these two? More transparency and clarity on this point would be helpful, particularly in light of research that other trauma types may also uniquely relate to OCD/OC symptoms.

[We agree that greater transparency regarding trauma selection is important. Our decision to focus on assault/victimization and transport accidents was based on a systematic evaluation of the availability of potentially traumatic events captured in the Swedish nationwide registers, rather than on an a priori assumption that these were the only relevant trauma types.](#)

Specifically, in addition to assault/victimization and transport accidents, we examined a range of other potentially traumatic events recorded in the registers, including injuries and fall-related accidents. We selected assault/victimization and transport accidents because they represent clearly defined, acute traumatic exposures. By contrast, injuries and fall-related accidents are more heterogeneous and may not necessarily constitute acute exposures suitable for consideration as potentially traumatic events. Many of these incidents are common during childhood and are therefore unlikely to result in lasting mental health problems.

We acknowledge that other trauma types may also be relevant for OCD and OC symptoms, as suggested by prior research. However, given the constraints of register-based data, we focused on the two trauma categories that allowed for the most robust and interpretable analyses in this nationwide setting. Importantly, these two categories also differ in the key aspect of interpersonal violence, providing meaningful contrast for our study.

- Are there any subsequent post-hoc analyses that can be run to offer insight into time-to-onset of OCD following trauma exposure? Certainly, there is an increased incidence of OCD within 12 months of violence exposure, however if the data allows, it would be interesting to know more precisely when the diagnosis was documented within this timeframe. This would address another existing gap in the literature, which is understanding exactly when OCD is likely to develop following trauma exposure (e.g., within weeks, within months, etc.).

We thank the reviewer for raising this interesting point. As an additional post-hoc analysis, and following the Reviewer's suggestion, we have now examined the risk of OCD after the victimization event by defining several follow-up periods within the first year (first month; second to third month; fourth to sixth month; seventh month to one year), as well as additional periods beyond the first year (between the first and fifth year, between the fifth and tenth year, and beyond the tenth year) (see **Figure** below). In these analyses, we observe that the highest risk of OCD occurs shortly after the victimization event, particularly between the second and third month. However, these analyses are considerably underpowered compared with the main analysis (especially for the within full sibling comparisons), and most confidence intervals overlap.

Even if these analyses were sufficiently powered, we believe that the Reviewer's question is difficult to answer with the available data. As noted in our limitations section (page 9), individuals exposed to victimization may receive closer monitoring for psychiatric disorders—particularly immediately after the event—which could contribute to the higher observed risk shortly after exposure. This makes it challenging to determine whether the increased risk reflects the true psychological impact of the event or heightened surveillance. As discussed in the manuscript, a key finding of the study is that the association persists beyond one year, suggesting that increased surveillance alone does not explain the observed findings.

We have now included this additional analysis as a complementary analysis in the supplement, supporting the idea that the association persists at different time points during follow-up. To do this, we have included the following in the methods section (page 13)

“To further explore the temporal dynamics of the association, we conducted a post hoc analysis using more granular follow-up intervals, ranging from the first month to more than ten years after exposure.”

And we have also commented on these new analyses in the results section (page 6)

“The additional post hoc analyses revealed a similar temporal pattern, with the risk of OCD peaking shortly after the victimization event—particularly between the second and third months—and gradually decreasing thereafter. Notably, an elevated risk remained detectable beyond ten years following exposure. However, these post-hoc analyses were substantially underpowered relative to the main analysis, and most confidence intervals overlapped (Supplementary Figure 1).”

Risk of obsessive-compulsive disorder over time after the victimization event

Redacted

Note: Blue is for the population model. Green is for the within full-sibling comparisons model.

Abbreviations: CI, confidence intervals

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• On pg. 8, the authors state: “This implies that evocative gene-environment correlations could be a potential mechanism, whereby genetically vulnerable individuals may be more likely to enter high risk situations where they may be exposed to interpersonal violence.” As this is an important point, it would be helpful if the authors could say more here regarding the ways in which genetics can increase someone’s risk for trauma exposure.

We agree with the Reviewer that this is an important point. As our results indicate, the association between victimization and OCD is partly influenced by genetic factors. For example, relatives of individuals exposed to victimization show a higher risk of OCD themselves, even when they share little or no environment with the proband.

While it is difficult to specify the exact mechanisms for such genetic influences, one plausible explanation is evocative gene–environment correlations (rGE). Previous research has shown that being victimized is to some extent influenced by genetic factors, suggesting that perpetrators may not select their victims randomly, but rather based on certain individual traits shaped by genetic factors (Dahoun et al., 2025). In this framework, individuals with a genetic liability for OCD may display characteristics that potentially make them more likely to be targeted by perpetrators. Although this remains a hypothesis, it is supported by population-based studies showing that people with psychiatric disorders have an increased risk of victimization (Christ et al., 2019; Dean et al., 2024; Sariaslan et al., 2024).

To clarify this concept for the readers, we have slightly modified the Discussion (page 8), also following a recommendation from Reviewer #2, to briefly elaborate on potential mechanisms underlying evocative rGE. We have kept this explanation concise to avoid over-speculation while ensuring that the concept of rGE and its relevance to our findings is clearly conveyed:

*“Interestingly, the familial co-aggregation and quantitative genetic modeling results suggested that the association between assault/victimization and OCD was largely attributable to shared genetic factors. **One potential mechanism for such shared genetic influences is evocative gene-environment correlation, whereby genetically influenced traits associated with OCD, such as emotional dysregulation, may increase the likelihood of encountering high-risk situations, thereby elevating the risk of interpersonal violence.**”³⁶⁻³⁸*

Reviewer #2 (Remarks to the Author):

The authors have prepared a well-written manuscript that utilizes population-based data to determine the relation between specific potentially traumatic events and incidence of OCD. The manuscript answers a unique question using original methodology. Critically, much of the work in this area has not utilized prospective data or merely demonstrates associations, which limits causal inference. The authors have utilized prospective methods to uncover this causal link.

We appreciate the Reviewer's positive remarks.

There are several suggestions for improvement included below:

1) Please include information about the incidence and prevalence of OCD. It would be helpful to contextualize these statistics within the population of interest here, as well as worldwide, if available.

The worldwide prevalence of OCD has been estimated to be around 2% (Fawcett et al., 2020; Ruscio et al., 2010). In Sweden, a country of approximately 10 million inhabitants, this would correspond to roughly 200,000 individuals with OCD, not all of whom will seek or receive clinical care, as prevalence estimates do not necessarily align with help-seeking or treatment utilization. In our cohort, 37,571 out of 3,340,945 individuals had a diagnosis of OCD (1.2%), which is reasonable given that case identification was limited to specialist care (inpatient care from 1973 and outpatient care from 2001). Estimating incidence is more challenging, as this question has been less thoroughly investigated and incidence rates vary by population, healthcare coverage, and time period.

We have now explicitly included information on OCD prevalence in the Introduction, as suggested by the Reviewer (page 4):

*“Obsessive-compulsive disorder (OCD) is a relatively common psychiatric disorder, **with an estimated worldwide prevalence around 2%,^{1,2}** associated with poor quality of life,³ long-term functional impairment,^{4,5} somatic morbidity,⁶⁻⁸ and increased mortality risk.^{9,10”}*

2) Because the authors discuss genetics and heritability, it may be helpful to review further literature in this area to address common genetic pathways for internalizing psychopathology and the specificity of genes that have been linked to OCD.

We appreciate the Reviewer's suggestion. Our intention in briefly mentioning heritability and the main OCD GWAS findings was simply to contextualize that, although genetic factors are relevant, they do not fully explain OCD risk, leaving room for important environmental contributions—which is the primary focus of the present study. To avoid shifting the emphasis of the Introduction toward genetics, we prefer to keep this section concise. Nevertheless, we have slightly expanded the text to briefly acknowledge both shared and disorder-specific genetic influences (page 4):

“The causes of OCD can be broadly understood as a consequence of the complex interplay between genetic and environmental risk factors.^{11,12} The specific genetic underpinnings of the disorder are only beginning to be unraveled, with evidence of substantial genetic correlations with other psychiatric disorders in general, and anorexia nervosa, Tourette syndrome, and internalizing disorders in particular.^{13,14} However, as the heritability of OCD is approximately 50%,^{12,15} it is of vital importance to identify environmental risk factors, particularly if they are modifiable.^{16,17”}

3) It would be helpful to include brief background on the context of reporting violent and sexual assaults in the context of Sweden. Are these screened for regularly in health clinics? Would patients have to bring this up on their own? If people would have to bring these events up on their own, is it possible that some siblings would be more likely to disclose to their doctor than others? How reliable is this variable if not all individuals are screened at each visit?

We thank the Reviewer for this important comment. Because it relates to concerns also raised in comments #7 and #8, we will address them together here.

The Swedish medical registers primarily reflect healthcare-seeking individuals. In the present study, data on victimization and transport accidents come exclusively from ICD codes assigned in specialized inpatient and outpatient care. Consequently, we mainly capture events requiring specialized medical intervention, such as transport accidents involving substantial

injuries, rather than milder cases treated in primary care or events that do not lead to healthcare contact.

In Sweden, routine screening for exposure to violence or sexual assault is not conducted universally across healthcare settings. Documentation of victimization therefore typically occurs when individuals disclose the exposure themselves, when injuries or symptoms prompt clinical inquiry, or when the exposure is clinically relevant to the reason for care. It is reasonable to assume that recordings of transport accidents do not depend heavily on individuals' willingness to disclose these events. In contrast, victimization events pose additional detection challenges.

Reassuringly, although victimization is generally under-detected in the Swedish healthcare system, it is most likely to be identified when it involves physical injuries treated in specialized emergency units or other specialized services, compared with primary care (Simmons & Swahnberg, 2022). In such settings, providers are more likely to inquire about the circumstances leading to the injuries, which reduces the likelihood of systematic differential reporting between siblings. However, not all victimization events captured in our study necessarily involve visible injuries requiring medical attention. Some may be recorded in psychiatric care, where detection is more dependent on help-seeking behavior, willingness to disclose, or clinician inquiry.

This constitutes a limitation of the study and may have resulted in exposure misclassification, with some victimized individuals classified as unexposed. Given the prospective design, it is reasonable to assume that such misclassification is largely non-differential with respect to later OCD onset, which would be expected to bias estimates toward the null rather than inflate them (Oltean et al., 2025). Nevertheless, we cannot entirely rule out the possibility that individuals more likely to seek care or disclose victimization may also be more likely to seek care for OCD at a later stage.

We have modified our limitations to make these points clearer (page 9):

*“Second, our analyses **may not have captured** milder forms of traumatic events, **as exposures were defined based on diagnoses recorded in inpatient and outpatient specialist care. Events for which individuals did not seek medical attention were therefore not included, either because they did not result in physical injuries requiring specialist care or for other***

*reasons (e.g., reluctance to disclose). In the Swedish healthcare context, victimization events are most likely to be detected and documented when they involve injuries treated in emergency units or other specialized care settings, rather than in primary care.*⁴²

4) The authors note that the statistical power of the quantitative genetic modeling is limited. I think this necessitates more information so that readers can interpret with the appropriate amount of caution. How limited was power? Is this something that should result in severe caution when interpreting, or is it the case that these effects are notable because they were found when they were likely underpowered? If an a priori power analysis was conducted, including a sentence or two, in comparison to the sample at hand, would be most helpful.

We thank the Reviewer for this important observation. When we refer to limited statistical power in the quantitative genetic modeling, we mean that the point estimates of genetic (A) and unique environmental (E) contributions should be interpreted with caution, as reflected in the wide confidence intervals. With a larger sample providing greater statistical power, these quantitative estimates might be more precisely determined (i.e. resulting in tighter confidence intervals). However, this limitation does not undermine our core finding. The coaggregation analyses clearly show a genetic contribution to the association between victimization and OCD, as shown by the pattern of results across different types of relatives, and also potentially indicate the contribution of unique environmental factors, as indicated by the estimates for full siblings, which are substantially different from the population estimate. What remains less precisely quantified is the magnitude of these specific contributions.

We have clarified this point further in the Discussion (page 10):

“Finally, our quantitative genetic analyses had limited power, as reflected in the wide confidence intervals of the estimates. Accordingly, the magnitude of the genetic and unique environmental contributions should be interpreted with caution. Nonetheless, our central conclusion, that both genetic and unique environmental factors contribute to the association, remain supported by the overall pattern of results, including consistent findings across several complementary analytical approaches”

5) The observed findings related to interpersonal, but not transport, trauma aligns with a large body of literature. This literature should be further explored to provide broader context for the findings and how they fit into the literature.

Thank you for this comment. This point is addressed in the Discussion section (page 8), where we situate our findings within the broader literature showing that interpersonal trauma is associated with more severe psychopathological consequences than non-interpersonal trauma. Specifically, we discuss how trauma involving intentional harm confers elevated risk of subsequent psychopathology, consistent with prior research across a range of mental disorders, including OCD. The revised text reads as follows (page 8):

“The observed associations were specific to assault/victimization, as we did not observe any meaningful associations with transport accidents. These categories differ meaningfully in that the first involves interpersonal trauma, characterized by intentional harm inflicted by another person, whereas the second typically reflects non-interpersonal or accidental trauma, lacking deliberate intent. This distinction is important, as interpersonal trauma is consistently associated with greater psychological impact, and may differentially influence the development of psychopathology, including OCD.”⁴⁰

6) The authors write: "Interestingly, the familial co-aggregation and quantitative genetic modeling results suggested that the association between assault/victimization and OCD was largely attributable to shared genetic factors. This implies that evocative gene-environment correlations could be a potential mechanism, whereby genetically vulnerable individuals may be more likely to enter high risk situations where they may be exposed to interpersonal violence." Perhaps I am misunderstanding, but this interpretation did not make sense to me. The results indicate that the relation between victimization and OCD may depend heavily on genetic factors. This means that those who have a genetic propensity, combined with the occurrence of this environmental event, are more likely to be diagnosed with OCD. The genetic vulnerability to enter high risk situations seems like a reach given the findings, and it also runs the risk of reading as blaming. I would suggest rephrasing to stick to the findings at hand and then note that future research should explore the genetic/environmental determinants of victimization specifically.

We agree with the Reviewer that these findings should be interpreted with caution.

Our results indicate that there are shared genetic factors between OCD and victimization. Although the underlying mechanisms remain uncertain, the implications of these findings are: (1) OCD has a genetic component, (2) victimization has a genetic component, and (3) both share genetic influences, to some extent. While the genetic component of OCD is relatively straightforward, genetic influences on victimization may be less obvious, as victimization appears to be an “environmental” exposure. However, consistent with our analyses, existing literature indicates that victimization also shows genetic influences (Dahoun et al., 2025). Given that this exposure appears to be environmental on the surface, evocative gene-environment correlations (rGE) are the most plausible mechanism for such influences (Dahoun et al., 2025; Jaffee & Price, 2007). In other words, some individuals may present traits/characteristics that increase their vulnerability to being victimized, meaning that perpetrators may not select victims randomly.

As we have mentioned, it is difficult to determine the specific mechanisms behind the shared genetic influences between OCD and victimization. Nevertheless, rGEs seem a reasonable hypothesis. In this scenario, individuals with a genetic liability for OCD may present characteristics that make them appear more vulnerable to potential perpetrators. Although this remains a hypothesis, it is supported by population-based studies showing that people with psychiatric disorders have an increased risk of being victimized (Christ et al., 2019; Dean et al., 2024; Sariaslan et al., 2024).

As the reviewer noted, these findings should not be understood as blaming individuals for being victimized—just as we do not attribute personal blame for developing OCD. The responsibility for the assault lies entirely with the perpetrator, who may select victims based on characteristics that themselves have some genetic influence. We have revised this section of the manuscript to improve clarity accordingly. See page 8 in the Discussion section:

*“Interestingly, the familial co-aggregation and quantitative genetic modeling results suggested that the association between assault/victimization and OCD was largely attributable to shared genetic factors. **One potential mechanism for such shared genetic influences is evocative gene-environment correlation, whereby genetically influenced traits associated with OCD, such as emotional dysregulation, may increase the likelihood of encountering high-risk situations, thereby elevating the risk of interpersonal violence.***^{36-38”}

7) I would note somewhere in the discussion that this analysis only includes those who chose to seek health care, share with their doctor about life events, and share their symptoms. If individuals never seek healthcare, they would not be included.

Please see our response to comment #3 above.

8) Are there suggested intervals in which Swedish individuals follow with providers? Are annual appointments required/optional/suggested? It would be helpful to know the extent to which Swedish individuals are screened, seen in clinic, or if this is entirely based on patient discretion.

As above, we refer the Reviewer to our response to comment #3.

9) Figure 1 would benefit from a caption to help to explain to readers how to interpret the graphs.

Thank you for your suggestion, we have included a note to the Figure to help with the interpretation of the graph, as follows:

“Note: Models were adjusted for birth year (categorized in 6-year increments) and sex. Color indicates type of comparison: population (blue) and within full sibling (green). Shape indicates follow-up period analyzed: whole follow-up period (circle), within the first year after the potentially traumatic event (triangle), and beyond one year after the potentially traumatic event (square).”

10) Analytic approach could be described in more detail to help increase replicability and validity of results.

We thank the Reviewer for this comment. We have re-examined the Methods section and have added several details to improve replicability, particularly regarding the coaggregation analysis. We have made a series of additions to the Methods section:

1. We have included more details on how the clusters of relatives were identified (pages 11-12):

“For the coaggregation and quantitative genetic analysis, we used individuals from the main cohort as probands and identified proband-relative pairs to construct four sub-cohorts: full siblings, maternal half siblings (i.e., siblings with the same mother but different father), paternal half siblings (i.e., siblings with the same father but different mother), and cousins (i.e., individuals that share two grandparents). All relatives were drawn from the main cohort and linked using family identification numbers.”

2. We have indicated that we used cluster-robust standard errors in our Cox regression analyses (page 13):

*“All models were adjusted for sex and birth year (categorized in 6-year increments). **Robust standard errors were applied in all analyses to address familial clustering.**”*

3. We have provided more details about the coaggregation analyses (page 13):

“All three models were adjusted for sex and birth year (categorized in 6-year increments) of both the proband and the relative. Robust standard errors were used in all models to account for clustering within families. Estimates are presented as odds ratios (ORs) with 95% CIs.”

4. We have specified the version of the R packages used (page 14):

“Analyses were conducted using SAS version 9.4 (SAS Institute Inc.) and the R packages Survival 3.5-5, drgee 1.1.10, and OpenMx 2.21.8. Data were analyzed from September 2024 to April 2025.”

Editorial Office comments:

In addition to the reviewers' comments, please address the following points:

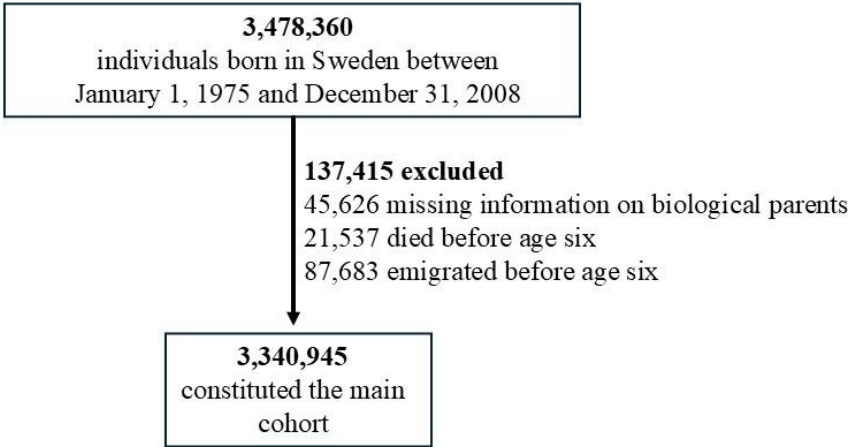
- Please fill out the STROBE checklist providing text excerpts from the manuscript for each point.

We have completed the STROBE checklist.

- Please add the flowchart as the main figure reporting numbers of individuals at each stage of study – i.e. numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analyzed. Please indicate the study design (cross-

sectional, case-control or cohort) in the Abstract or title according to the STROBE requirements.

We have included the flow chart as the main figure (now Figure 1), reporting the number of individuals at each stage of study (see below).



“Note: Flow chart depicting the selection of the main cohort. Some individuals may have been excluded for more than one reason (e.g. missing information on biological parents and emigrating before age six). From this cohort, we subsequently identified clusters of relatives for coaggregation and quantitative genetic modeling, which are presented separately in Supplementary Table 2.”

Page 5:

*“The characteristics of the main cohort are presented in Table 1. **After excluding 137,415 individuals**, the main cohort included 3,340,945 individuals (Figure 1).”*

We have also indicated the study design in the Abstract (page 3), in accordance with STROBE requirements. The revised sentence now reads:

“In this Swedish population-based cohort study, we identified 3,340,945 individuals born between 1975-2008 and prospectively examined the associations of objectively (...)”

- To have more information on how representative the study sample is, please include data on study participants' ethnicity in Methods and Table describing demographic data. If ethnicity is

not reported, the reason should be stated in Methods. The Methods section should include an explanation of who identified participant ethnicity and the source of the classifications used (e.g. self-report or selection, investigator, database, electronic health record, survey instrument). Specific ethnic categories are preferred over collective terms, when possible. Categories included in groups labeled as “other” should be defined.

We are unable to report ethnicity because information on race/ethnicity is not collected in the Swedish national registers due to legal and ethical restrictions. We have added a clarifying sentence to the Methods section (page 12):

Ethnicity is not reported because Swedish national health and population registers do not collect this information for legal and ethical reasons.

- Please add Code Availability sections in the end of the manuscript. If any custom code was created for the data analysis, please add a link to the code in the open repository according to the Nature Mental Health's reporting

standards: <https://www.nature.com/natmentalhealth/editorial-policies/reporting-standards#availability-of-computer-code>

We have included a Code Availability statement in the manuscript (page 16). See below:

*“**Code availability:** No custom code was developed for this study; all analyses were conducted using publicly available resources. Data management was performed in SAS version 9.4 (SAS Institute Inc.), and data analyses were carried out in R (packages: survival 3.5-5, drgee 1.1.10, and OpenMx 2.21.8) within RStudio version 2025.9.0.387. Scripts for the data analysis are available at Zenodo (<https://doi.org/10.5281/zenodo.18258343>)”*

Please note that the scripts for the data analysis will be publicly available at Zenodo upon acceptance of the paper.

References

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Reviewers' Comments:

Reviewer 1

Remarks to the Author:

I thank the authors for their thorough response to Reviewer and Editorial Office feedback. Their revisions have improved the transparency and clarity of this work. I am particularly grateful to the authors for conducting the requested post-hoc analysis regarding time-to-onset of OCD. While underpowered (and appropriately couched as such), these data offer important preliminary information about when trauma-exposed individuals may be at heightened risk for developing OCD, which could have a downstream effect on better powered empirical inquiry and more precise intervention efforts. I have no further feedback and am grateful to the authors for their commendable efforts to move this literature forward in a major way. Congratulations!

Reviewer 2:

Remarks to the Author:

Many thanks to the authors for the thoughtful and thorough revision. In particular, thank you for further describing the analytic approach.

The requested areas for evaluation are described below.

- A. Summary of key results - the authors have adequately described the results in detail, and the additional findings added to the manuscript in the revision are interesting findings for future research!
- B. Originality and significance - the authors added information about the prevalence of OCD, which speaks to the significance. I appreciate the originality of this design in investigating causal inference.
- C. Data and methodology - additional information provided in this revision was very helpful.
- D. Appropriate use of statistics and treatment of uncertainties - the uncertainties are better described, and statistics were used appropriately.
- E. Conclusions - conclusions were robust. There may be concerns with the validity of report of traumatic experiences, as the authors noted in the response letter. Those who experience

significant injury are most likely to report or seek care. This is likely an underestimate of the true prevalence.

F. Suggested improvements - the authors have improved the manuscript in this revision, and I do not have further recommendations.

G. References - appropriate.

H. Clarity and context - Appreciate the additional context that was added, and this clarified all of my questions.

[We thank the reviewers for their thorough evaluation of our manuscript. There are no further reviewer comments to address at this stage.](#)