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# Childhood sexual abuse and early substance use in adolescent girls: the role of familial influences

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## ABSTRACT

**Aim** To assess the extent to which the association between childhood sexual abuse (CSA) and early use of alcohol, cigarettes and cannabis in adolescent girls is mediated by risk factors that tend to cluster in families where CSA occurs. **Design** An abridged version of the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA) was administered by telephone. **Participants** A total of 3761 female twins aged 18–29 (14.6% African American, 85.4% European American). **Measurements** CSA experiences and history of substance use were queried in the SSAGA-based interviews. **Findings** After controlling for familial influences on early substance use by including co-twin early use status in models, separate Cox proportional hazards regression analyses predicting onset of alcohol, cigarette and cannabis use revealed a significant association with CSA. The effect was observed to age 19 years for cigarettes and to age 21 years for cannabis, but was limited to age 14 years or younger for alcohol, with the most pronounced risk before age 10 [hazard ratio (HR) = 4.59; confidence interval (CI): 1.96–10.74]. CSA-associated risk for initiation of cigarette and cannabis use was also highest in the youngest age range, but the decline with age was much more gradual and the hazard ratios significantly lower (HR: 1.70; CI: 1.13–2.56 for cigarettes and HR: 2.34, CI: 1.57–3.48 for cannabis). **Conclusions** Childhood sexual abuse history is a distinct risk factor for use of cigarettes and cannabis, and a very strong predictor of early age at first drink.

**Keywords** Alcohol, cannabis, cigarettes, sexual abuse, twins, women.

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## INTRODUCTION

An estimated 20% of women in the United States report experiencing childhood sexual abuse (CSA) [1–3], a well-documented risk factor for a range of substance-related outcomes. CSA has been linked to elevated rates of alcohol abuse and dependence [4–8], adolescent alcohol use [9,10] regular smoking [6,11], nicotine dependence [6,7], cannabis use [6,9] and cannabis use disorders [5,8]. Fewer investigations have focused on its association with early onset substance use, but the pattern is the same. The risks for early age at first drink [9,10,12], first cigarette [12,13], first cannabis use [9,12], regular drinking before age 10 [12] and heavy episodic drinking before age 15 [14] are greater in girls who have experienced CSA than in those who have not. Early initiation of substance use is a major public health concern because early use typically means longer than average life time exposure to substances of abuse and, even more importantly, exposure during a critical period of brain development, with the potential for serious and long-lasting consequences [15,16]. Additionally, risk for abuse or dependence is substantially higher in early initiators of alcohol [17–19], nicotine [20,21] and cannabis [22,23] use. Thus, substance use in childhood or early adolescence is an early warning sign of possible substance-related problems later in life.

CSA occurs frequently in combination with other adverse events or conditions that contribute to psychological problems. CSA is more common in families in which one or both parents have a history of problem drinking [1,2,10,24] or psychopathology [24,25]; children are physically or emotionally abused or neglected [10,24,25]; and/or familial conflict, including violence

between parents or parental separation, occurs [10,25]. Depression [4,7] and post-traumatic stress disorder (PTSD) [26,27] are also common among girls who have been sexually abused. Each of these co-occurring conditions is also associated with early and problem substance use. Parental alcohol problems have been linked to early age at first drink and first cigarette [17,28], as well as regular smoking [29] and cannabis dependence [30,31]. Exposure to adverse events such as physical abuse has been linked to early use of alcohol [9,10], cigarettes [12,13] and cannabis [9,12], as have both depression [32,33] and PTSD [8,21]. This clustering of risk factors makes it difficult to determine whether the observed elevation in risk for substance use outcomes associated with CSA is attributable to exposure to sexual abuse specifically or to the constellation of risk factors that tend to co-occur with CSA. Making this determination is important with respect to the interpretation of CSA history as a distinct marker of risk for early initiation of substance use, yet few investigations have applied the methodological approaches needed to do so.

The aim of this study was to assess the extent to which the association between CSA and early use of alcohol, cigarettes and cannabis in adolescent girls is mediated by risk factors that cluster in families where CSA occurs. Given the challenge of measuring and adjusting statistically for the many risk factors associated with both CSA and early substance use, other approaches for attempting to isolate the effects of CSA on initiation of substance use are needed. In the current study, we modeled CSA as a predictor of initiation of alcohol, cigarette and cannabis use (separately), then added co-twin early use status into models to control for genetic and shared environmental influences common to early use and co-occurring psychiatric and psychosocial risk factors (e.g. depression, physical abuse). The contribution of familial influences to the association between CSA and early use could be inferred by observing changes in the estimated risk attributed to CSA after adding co-twin early use status.

## METHODS

### Participants

The sample was composed of 3761 female twins who completed the fourth wave of data collection for the Missouri Adolescent Female Twin Study (MOAFTS). Female twins born in Missouri to Missouri-resident parents between 1975 and 1985 were identified through birth records and recruited between 1995 and 1999 for the MOAFTS wave 1 assessment. Cohorts of 13-, 15-, 17- and 19-year-old female twin pairs and their families were ascertained in the first 2 years. New cohorts of 13-year-old twins and their families were added in the subsequent 2 years. Parent interviews were completed by 78% of targeted families (see Heath *et al.* [34] for details on ascertainment). Wave 3 retest interviews were administered to a subset of wave 1 participants 2 years after wave 1 assessments. (Data were not drawn from wave 2, which covered only the previous 24 months.) Wave 4 interviews were conducted from 2002 to 2005. Wave 5 assessments were conducted from 2005 to 2008. Of the 4638 twins identified from birth records, 80% completed the wave 4 interview ($n = 3787$), the primary source of data for the current study. Of these participants, CSA status was available for 3761 (926 monozygotic twin pairs, 772 dizygotic twin pairs and 365 individuals whose co-twins did not participate in the study). Mean age at wave 4 was 21.7 [standard deviation (SD) = 2.8; range 18–29] years; 14.6% of participants self-identified as African American, 85.4% as European American.

### Procedure and assessment battery

Data were collected by trained interviewers through an interview modified for telephone administration from the Semi-Structured Assessment for the Genetics of Alcoholism [35,36]. Verbal consent was obtained prior to starting the interview. Interviews queried substance use history, DSM-IV psychiatric disorders and related psychosocial domains. MOAFTS was approved by the Washington University Human Research Protections Office.

### Predictors and outcomes

Data from wave 4 were available for all participants. Data from waves 1, 3 and/or 5 were available for 95.5% of participants. In cases where age at first CSA experience or age at first use of alcohol, cigarettes or cannabis was reported in more than one assessment, the first report was used.

#### *CSA*

CSA was queried in waves 1, 3 and 4 using the questions listed in Table 1. Items relevant to sexual abuse history appeared in three different sections of the interview under different wording. A cut-off of age 15 was chosen, as questions in the Parental Discipline and Childhood Experiences section of the interview referred to the period 'before you turned 16'. Age at first occurrence was queried for all items endorsed. Criteria for CSA were met if any sexual abuse question in any wave of data collection was endorsed and reported to have occurred at age 15 or younger. Thirteen per cent of participants ($n = 487$) reported experiencing CSA. Mean age at first CSA experience was 8.4 (SD = 4.1) years. There were 1749 complete twin pairs in which CSA status was available for both members of the pair: 111 concordant for CSA (6.5%),

211 discordant for CSA (12.1%) and 1427 in which neither twin endorsed CSA (81.6%).

*Age at first use*

Age at first use was queried if the respondent reported having consumed at least a full alcoholic drink (alcohol), trying a cigarette (cigarette) or using marijuana (cannabis). Use/age at first use was broken into four categories for each substance: never, early (lowest quartile of age at first use distribution), average (middle 50%), and late (highest quartile). (See Table 2 for substance-specific age ranges.) As there is no standard definition of early initiation of substance use, we chose the lowest quartile of the age distribution to represent early use relative to same sex peers in the same birth cohort.

*Familial risk for substance use*

Familial risk for substance use was operationalized as a dichotomous variable representing co-twin early use of the corresponding substance, which was derived from the categorization of the co-twin's reported use/age at first use of the substance as early, average, late or never. An interaction term between co-twin early use and zygosity, which is an indicator of the extent to which familial influences are attributable to genetic factors, was also included in analyses.

**Table 1** Items used to define childhood sexual abuse (CSA) by section of the interview.

| |
|---|
| Traumatic events (waves 1, 3 and 4) |
| Endorsement of event 1 or 2, with reported age at first experience 15 years or younger |
| 1. Raped |
| 2. Sexually molested |
| Health problems and health habits (waves 1, 3 and 4) |
| Endorsement of item, with reported age at first experience 15 years or younger |
| Has anyone ever forced you to have sexual intercourse? |
| Parental discipline and early childhood experiences (wave 4) |
| Endorsement of item 1 or 2 |
| 1. Before you turned 16, was there any forced sexual contact between you and any family member, such as a parent or step-parent, grandfather, etc? By sexual contact I mean their touching your sexual parts, your touching their sexual parts or intercourse. |
| 2. Before you turned 16, was there any forced sexual contact between you and anyone who was 5 or more years older than you (other than a family member)? |

## Data analysis

*Use/age at first use by CSA history*

Chi-square tests of association were conducted to test for differences by CSA status in use/age at first use.

*Predicting use of alcohol, cigarettes and cannabis with CSA history*

Three sets of Cox proportional hazards (PH) regression analyses were conducted to predict onset of alcohol, cigarette and cannabis use (i.e. first use). Cox PH regression analyses are commonly used with time-to-event data to account for the possibility that some participants who have not yet experienced the event of interest (here, use of alcohol, cigarettes or cannabis) may do so in the future. The advantage of this approach is that data until the time of censorship (most recent interview) are used in the calculation of hazard ratios (HRs). In addition, tests of the PH assumption that risk remains constant over time can reveal the extent to which risk associated with CSA varies across the period of risk, i.e., whether CSA is a predictor specifically of early initiation of substance use. CSA was modeled as a time-varying covariate by creating a 'person-year' data set using SAS [37]. Data were constructed such that each line of data represented a single year of life for each case. CSA was coded as absent in each year prior to age of onset and present for each subsequent year. Zygosity was coded with dizygotic twin pairs as the reference group. Analyses were conducted in Stata [38] using the Huber–White correction to adjust for the non-independence of observations in twins. Model 1 included only CSA. Co-twin early use and the interaction between co-twin early use and zygosity were added in model 2.

**Table 2** Age at first use of alcohol, cigarettes, and cannabis by childhood sexual abuse (CSA) status[a].

| | *Alcohol* | | | *Cigarettes* | | | *Cannabis* | | |
|---|---|---|---|---|---|---|---|---|---|
| | | *Prevalence (%)* | | | *Prevalence (%)* | | | *Prevalence (%)* | |
| | *Age range (years)* | *CSA* | *No CSA* | *Age range* | *CSA* | *No CSA* | *Age range (years)* | *CSA* | *No CSA* |
| Early | ≤14 | 32.7 | 20.0 | ≤12 | 35.5 | 19.0 | ≤15 | 29.6 | 12.9 |
| Average | 15–17 | 41.3 | 44.5 | 13–16 | 39.6 | 36.0 | 16–18 | 30.6 | 26.3 |
| Late | ≥18 | 20.9 | 28.6 | ≥17 | 12.6 | 18.0 | ≥19 | 8.4 | 11.7 |
| Never | | 5.1 | 6.9 | | 12.3 | 27.0 | | 31.4 | 49.1 |

[a]CSA: $n = 487$; no CSA: $n = 3274$.

The proportional hazards assumption was tested with the Grambsch and Therneau test of the Schoenfeld residuals [39]. Violations were observed for the variables representing CSA in all models, co-twin early use in model 2 for alcohol, cigarettes and cannabis, and co-twin early use by zygosity in model 2 for cigarettes. Violations indicated that the association of these variables with risk for initiation of substance use varied by period of risk (i.e. age), so hazard ratios were reported by risk period. Division of the risk period was based on the distribution of age at first use for each of the three substances. For alcohol, the four categories were 5–9, 10–14, 15–19 and ≥20 years; for cigarettes: 4–9, 10–14, 15–19 and ≥20 years; and for cannabis: 5–13, 14–17, 18–21 and ≥22 years.

## RESULTS

### Use/age at first use by CSA history

Rates of alcohol, cigarette and cannabis are shown by CSA status in Table 2, with lifetime users categorized as early, average and late initiators. Chi-square tests of association revealed significant differences in use/age at first use by CSA status for all three substances: alcohol ($\chi^2_{(3)} = 43.23$, $P < 0.001$), cigarettes ($\chi^2_{(3)} = 100.30$, $P < 0.001$) and cannabis ($\chi^2_{(3)} = 114.18$, $P < 0.001$). In every case, women who had experienced CSA were over-represented in the early use category.

### Predicting use of alcohol, cigarettes and cannabis with CSA history: results of Cox proportional hazards regression analyses

*Alcohol*

As seen in Table 3, in model 1, CSA was associated with a greater than four-fold increase in risk for initiation of alcohol use before age 10 [HR = 4.59; confidence interval (CI): 1.96–10.74] that dropped to a 76% increase in risk for use between ages 10 and 14 (HR = 1.76; CI: 1.46–2.14) and 15% for ages 15–19. The addition of co-twin early age at first drink and co-twin early use × zygosity in model 2 did not reduce the HR for use before age 10, but HR estimates for the later periods of risk were slightly attenuated, significantly at ages 10–14 (i.e., the HR did not fall within the model 1 CIs) and at the trend level for 15–19. There was also evidence for a peak period in risk associated with co-twin early use: HR = 4.51 (CI: 3.62–5.62) for ages 10–14. The HR for co-twin early use by zygosity (1.25; CI: 1.04–1.51) is suggestive of a modest degree of genetic influence on the timing of first drink.

*Cigarettes*

As seen in Table 4, in model 1, CSA was associated with a significant increase in risk for cigarette use to age 19. The

**Table 3** Results of Cox proportional hazards regression analyses predicting alcohol use.

| | *Model 1* | *Model 2* |
|---|---|---|
| | *CSA only* | *CSA + co-twin early use and co-twin early use × zygosity* |
| | *HR (95% CI)* | *HR (95% CI)* |
| CSA | | |
| 5–9 years | 4.59 (1.96–10.74)* | 4.75 (1.83–12.31)* |
| 10–14 years | 1.76 (1.46–2.14)* | 1.42 (1.19–1.70)* |
| 15–19 years | 1.15 (1.02–1.31)* | 1.10 (0.96–1.26) |
| ≥20 years | 0.79 (0.57–1.11) | 0.76 (0.53–1.08) |
| Co-twin early use | | |
| 5–9 years | – | 2.26 (0.92–5.56) |
| 10–14 years | – | 4.51 (3.62–5.62)* |
| 15–19 years | – | 1.40 (1.20–1.62)* |
| ≥20 years | – | 1.02 (0.75–1.38) |
| Co-twin early use × zygosity | – | 1.25 (1.04–1.51)* |
| Zygosity | – | 0.95 (0.87–1.03) |

*$P < 0.05$. HR: hazard ratio; CI: confidence interval. CSA: childhood sexual abuse.

**Table 4** Results of Cox proportional hazards regression analyses predicting cigarette use.

| | *Model 1* | *Model 2*[a] |
|---|---|---|
| | *CSA only* | *CSA + co-twin early use and co-twin early use × zygosity* |
| | *HR (95% CI)* | *HR (95% CI)* |
| CSA | | |
| 4–9 years | 2.61 (1.79–3.80)* | 1.70 (1.13–2.56)* |
| 10–14 years | 1.94 (1.65–2.28)* | 1.34 (1.12–1.59)* |
| 15–19 years | 1.40 (1.16–1.68)* | 1.35 (1.11–1.66)* |
| ≥20 years | 1.33 (0.77–2.29) | 1.02 (0.54–1.92) |
| Co-twin early use | | |
| 4–9 years | – | 6.38 (4.12–9.87)* |
| 10–14 years | – | 2.67 (2.15–3.30)* |
| 15–19 years | – | 1.24 (0.94–1.63) |
| ≥20 years | – | 2.44 (1.18–5.04)* |
| Co-twin early use × zygosity | | |
| 4–9 years | – | 1.32 (0.83–2.09) |
| 10–14 years | – | 2.14 (1.60–2.86)* |
| 15–19 years | – | 1.19 (0.80–1.78) |
| ≥20 years | – | 0.52 (0.14–1.96) |
| Zygosity | – | 0.79 (0.68–0.92)* |

*$P < 0.05$. [a]Interactions added to adjust for proportional hazards (PH) violations for zygosity: zygosity × risk periods 15–19 and ≥20. CSA: childhood sexual abuse; HR: hazard ratio; CI: confidence interval.

highest HR was observed for the period of risk from ages 4–9 (2.61; CI: 1.79–3.80), but the decrease in HR estimates across periods of risk was more gradual than for alcohol; point estimates of 1.94 and 1.40 were found for ages 10–14 and 15–19, respectively. The addition of co-twin early use and its interaction with zygosity led to a significant drop in HRs for ages 4–9 and 10–14, but CSA remained a significant predictor of cigarette use (HR = 1.70; CI: 1.13–2.56 for ages 4–9, HR = 1.34; CI: 1.12–1.59 for ages 10–14). The HR for ages 15–19 in model 2 was nearly identical to that for ages 10–14, and was not significantly lower than in model 1. Co-twin early cigarette use was associated with a greater than six-fold increase in risk for use at ages 4–9, and a more than 2.5-fold increase for ages 10–14. Genetic influences were evident at ages 10–14 (HR for co-twin use by zygosity was 2.14, CI = 1.60–2.86).

*Cannabis*

As seen in Table 5, in model 1, CSA was associated with a 3.5-fold increase in risk for cannabis use at ages 5–13 (HR = 3.56; CI: 2.44–5.20), an 80% increase for ages 14–17 and a marginally significant increase at ages 18–21. The HRs remained significant following the addition of co-twin early use and its interaction with zygosity. The estimates differed little for ages 14–17 and 18–21, but the drop to 2.34 (CI = 1.57–3.48) was statistically significant for the youngest group [5–13]. Co-twin early use was associated with a nearly eight-fold elevation in risk for use before age 14 and a 3.5-fold increase in risk between 14 and 17. Familial contributions to cannabis use were attributable in part to genetics (HR for co-twin early use by zygosity = 1.84; CI: 1.43–2.37).

**Table 5** Results of Cox proportional hazards regression analyses predicting cannabis use.

| | *Model 1* | *Model 2* |
|---|---|---|
| | *CSA only* | *CSA + co-twin early use and co-twin early use × zygosity* |
| | *HR (95% CI)* | *HR (95% CI)* |
| CSA | | |
| 5–13 years | 3.56 (2.45–5.17)* | 2.34 (1.57–3.48)* |
| 14–17 years | 1.80 (1.54–2.11)* | 1.60 (1.36–1.88)* |
| 18–21 years | 1.30 (1.01–1.69)* | 1.37 (1.05–1.79)* |
| ≥22 years | 0.72 (0.33–1.56) | 0.84 (0.38–1.82) |
| Co-twin early use | | |
| 5–13 years | – | 7.84 (5.07–12.13)* |
| 14–17 years | – | 3.40 (2.76–4.20)* |
| 18–21 years | – | 1.38 (0.98–1.93) |
| ≥22 years | – | 1.62 (0.65–4.04) |
| Co-twin early use × zygosity | – | 1.84 (1.43–2.37)* |
| Zygosity | – | 0.84 (0.74–0.94)* |

*$P < 0.05$. HR: hazard ratio; CI: confidence interval; CSA: childhood sexual abuse.

## DISCUSSION

The current study extends the literature on childhood sexual abuse and substance use by examining the contribution of familial influences to this association and comparing the degree to which familial risk accounts for the co-occurrence of CSA and early initiation of alcohol, cigarette and cannabis use. Across substances, women with a history of CSA reported a younger age at first use, and this association persisted after controlling for genetic and shared environmental influences that contribute to early use.

### Familial influences

The fact that CSA remained a significant predictor of early initiation of use after accounting for familial influences on early use indicates that the association between CSA and early substance use is only partially accounted for by common risk factors such as genetic liability to substance use disorders, parental separation and depression. The significant association between co-twin early use and respondent substance use onset found for each of the three substances provides further support for the contribution of genetic and shared environmental influences to initiation of alcohol, cigarette and cannabis use reported in the larger literature [40–42].

Only one other known investigation using a twin design to control for common familial influences has reported on the association between CSA and lifetime substance use: a study by Nelson and colleagues [7], in which women exposed to CSA were significantly more likely than their unexposed co-twins to report using cannabis. The majority of co-twin control studies of CSA have focused on substance use disorders. Our results also parallel their findings of elevated rates of alcohol use disorders [5–7], nicotine dependence [6,7] and cannabis dependence [5,6] after controlling for genetic and shared environmental influences. Among them is Kendler *et al.*'s [5] study of psychiatric and substance use disorders in women, in which the authors concluded that the observed pattern of results was consistent with a causal relationship between CSA and psychiatric and substance use disorders.

### Differences across substances: alcohol versus cigarettes and cannabis

There are three striking distinctions between alcohol and the other two substances with respect to their

associations with CSA. First, the decrease with age in CSA-associated risk for use was much more gradual for cigarettes and cannabis. Second, the estimated risk associated with CSA in the youngest age range, after adjusting for familial influences, was more than twice as high for alcohol than for cigarettes or cannabis. Third, there was a significant degree of overlap between the contributions of CSA and familial influences on early use of cigarettes and cannabis, as indicated by the significant decreases in HRs from model 1 to model 2 for the youngest age ranges. There was no change in the estimate of CSA-associated risk for very early use of alcohol after controlling for genetic and shared environmental influences, indicating that familial risk factors that co-occur at elevated rates in families affected by CSA did not account for the association. The relative influence of familial risk (represented by co-twin early use in the models) on initiation of use was also notably lower for alcohol compared to cigarettes and cannabis.

The distinctions between alcohol versus cigarettes and cannabis may be explained in part by the fact that some alcohol use is nearly universal, but the use of cannabis and, increasingly, cigarette smoking, at any age is somewhat deviant. Ninety-three per cent of our sample reported using alcohol, compared with 75% for cigarettes and only 47% for cannabis. With such a large proportion of individuals using alcohol, there would be little variance to account for if the outcome of interest were ever versus never using alcohol. By splitting the risk period we were, in essence, predicting age at first drink. Given that alcohol consumption before age 10 is so rare, it was not surprising that CSA-associated risk was concentrated in this young age range, before peer influences on drinking have emerged. As for why the association between CSA and early use was so much stronger for alcohol than for cigarettes or cannabis, this may be a reflection of easier access to alcohol versus cannabis and/or greater perceived effectiveness of alcohol versus cigarettes for dampening negative affect associated with experiencing CSA, but these hypotheses have yet to be tested.

### Limitations

The findings from this study should be interpreted with certain limitations in mind. First, the proportion of women endorsing CSA in our study was somewhat lower than prevalence estimates of CSA in women found in the larger literature [1–3]. The lower degree of specificity in our CSA assessment (e.g., we did not query vaginal, oral and anal sexual abuse acts separately) and relatively young age cut-off compared to other studies may have contributed to this difference. Second, in discordant pairs the unaffected twin may be influenced indirectly by her co-twin's CSA history, such that the affected twin seeks out a substance at a young age and uses with her unaffected co-twin [43,44]. Co-twin early use status is therefore not a very conservative indicator of familial influences. Third, the greater degree of overlap between early use and CSA in concordant than discordant twins results in a more modest drop in the estimated HR for CSA for a sexually abused twin whose co-twin was also abused, thus leading to an overestimate of CSA-associated risk in these twins. Fourth, using a time-varying covariate to represent CSA avoids the issue of CSA being counted as a risk factor for alcohol use when the first drink is consumed at the same time as the first CSA experience, but it is still possible that the observed elevated risk for very early alcohol use in women with a history of CSA reflected alcohol use in the context of later sexual abuse experiences.

## CONCLUSIONS

The results indicate that CSA is a distinct risk factor for use of cigarettes and cannabis, especially at a young age, and a potent predictor of very early use of alcohol, and thus support the use of CSA history as a marker of high risk for substance-related problems. In addition, our findings highlight the importance of integrating substance abuse prevention into psychological support services for girls who have experienced CSA.

### Future directions

Identification of the specific familial influences, such as parental monitoring, quality of parent–child relationships and parental psychopathology, that jointly contribute to or reduce risk for CSA and early substance use, is an important next step in this line of research. Additional research is also needed to determine the generalizability of findings to ethnic groups not included in the current sample, and also to males, who have substantially lower rates of CSA [1,3,4].

### Declarations of interest

None.

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