Does the association of child maltreatment with adult cardiovascular disease differ by gender?

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Childhood maltreatment (abuse and neglect) is a preventable risk factor associated with a range of health outcomes. There is now extensive evidence on links between childhood maltreatment and cardiovascular diseases (CVD) in adulthood (1). It is known that the occurrence of maltreatment and distributions of some CVD outcomes differ by gender, raising the question as to whether the association of childhood maltreatment with adult CVD differs by gender, and more importantly, the reasons and potential mechanisms for such differences in association. Yet few studies have explicitly compared the association between men and women. Understanding whether gender modifies the relationship will have important implications on deciding whether intervention aiming to reduce the risk of CVD should be tailored differently for maltreated men and women.

Main findings from the UK Biobank

The study by Soares and colleagues (2) using the large UK Biobank cohort adds to our understanding on the impact of gender on CVD risk in adults who have experienced maltreatment in childhood. The authors investigated the associations of individual type of maltreatment (also number of types) with three CVD outcomes (hypertensive disease, ischaemic heart disease and cerebrovascular disease) and tested gender differences in the associations. The outcomes were derived from both self-reported and objective measures of CVD, the latter of which included hospital and death register data. The large sample size provides statistical power and enables the investigation of the gender-specific risk for each CVD outcome, especially for infrequent maltreatment types, such as sexual abuse. This study found consistent associations between childhood maltreatment and adult CVD. The associations were generally stronger in women than men, although the gender differences were mostly modest. In particular, the associations of physical abuse with ischaemic heart disease (relative risk 1.47 for women vs 1.19 for men) and with any CVD (1.14 vs 1.07), and emotional neglect with ischaemic heart disease (1.41 vs 1.18) were significantly stronger in women (2).

Potential mechanisms for maltreatment–CVD associations and gender differences

Several mechanisms by which child maltreatment confers risk for CVD later in life have been postulated. Firstly, maltreatment may lead to unhealthy behaviours, which impact on the risk of CVD. Secondly, the influence of maltreatment on mental health problems could link to CVD via increased physiological response to stressors or unhealthy behaviours. Thirdly, maltreatment may influence adult socio-economic position (SEP), which in turn affects adult cardio-metabolic health. Fourthly, childhood maltreatment is associated with dysregulation of biological stress response systems such as the hypothalamus–pituitary adrenal (HPA) axis (3), and elevated inflammatory markers (4), which have been shown to link to CVD risk.

Little work has been done so far to establish the rationale for gender difference in the maltreatment-CVD relationship, although some potential mechanisms have been found to vary by gender. For instance, child maltreatment is associated with alcohol problems (5) and lower likelihood of quitting smoking in women (6), but not in men, suggesting that there might be gender differences in the health behavioural pathway. Gender difference has also been noted in the maltreatment association with the risk of developing obesity across the life course, with a stronger association in women (7). It has been suggested that psychosocial stress may be a more important risk factor for CVD in women, who may be exposed to more psychosocial stress or may be more vulnerable to the mental health consequences of maltreatment (8). Moreover, psychosocial stress may be more strongly associated with cortisol patterns or inflammatory markers in women. For example, among those experiencing childhood neglect in the 1958 British birth cohort, lower morning cortisol levels and slower morning decline were found in women but not in men, reflecting possible dysregulation of the HPA axis in women alone (3).

Evidence on gender differences in maltreatment-CVD associations

Gender-specific mechanisms may lead to differential associations for men and women. However, few studies have formally investigated gender-related differences in the association of maltreatment with adult cardio-metabolic outcomes, such as coronary heart disease and stroke, and among studies that have, there are inconsistent gender-related patterns (1). More recently, in the 1958 British Birth Cohort, we found that physical abuse and neglect in childhood were associated with lower HDL-C, a risk factor for CVD only in females, and no gender difference for other cardio-metabolic biomarkers (9). These data indicate large gaps in our knowledge. Therefore, a more rigorous investigation of gender differences in maltreatment association with CVD and its mechanisms is warranted.

The findings from Soares et al 2020 are important, although we recommend some care when interpreting them for the following reasons. First, the participant cohort is highly selective. Participants are predominantly white, more likely to live in less deprived areas, and heathier with lower disease rate than the general British population. Second, the retrospective self-reports of childhood maltreatment could be affected by recall error or reporting bias associated with current emotional or health status. However, retrospective measures of adversity are widely used in population studies when prospective measures are not available, although they should not be assumed to be interchangeable. Third, maternal smoking around birth and current Townsend scale

(which could be on the maltreatment to CVD pathway) are used as proxy indicators of childhood SEP. Many effects found here are of small/moderate sizes but statistically significant. This could be partly due to the large sample size and uncontrolled confounders such as adverse environmental and social factors in childhood.

As pointed out by the authors, more work is needed to better understand the life course pathways that might explain any gender differences in the associations. Large administrative databases with high statistical power such as UK Biobank are valuable to study gender-specific associations. Nonetheless, prospective longitudinal studies (e.g. birth cohorts) with data collected across the life course can account for early life confounders and life course pathways for the associations and thus would be ideal to assess the complex associations.

In summary, this current study adds important knowledge to the so-far limited literature on gender difference in the life course influence of childhood maltreatment on cardio-metabolic health. If the association indeed differs by gender, it will be crucial to further explore the mechanisms and life course pathways that may contribute to the gender-specific associations and also the timing of their emergence, which may indicate a sensitive period during which intervention could lead to improvements in adult cardio-metabolic health, especially for women.

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