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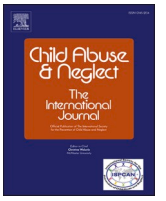

Gender differences in the association between adverse childhood experiences and premature mortality: A prospective population study

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ABSTRACT

Background: Birth cohort studies have shown that adverse childhood experiences (ACEs) are associated with all-cause mortality. The effect of ACEs on premature mortality among working-age people is less clear and may differ between the genders.
Objective: In this prospective population study, we investigated the association of ACEs with all-cause mortality in a working-age population.
Participants and methods: In a representative Finnish population study, Health 2000, individuals aged 30 to 64 years were interviewed in 2000, and their deaths were registered until 2020. At baseline, the participants ($n = 4981$, 2624 females) completed a questionnaire that included 11 questions on ACEs and questions on tobacco smoking, alcohol abuse, self-reported health and sufficiency of income. All-cause mortality was analysed by Cox regression analysis.
Results: Of the ACEs, financial difficulties, parental unemployment and individual's own chronic illness were associated with mortality. High number (4+) of ACEs was significantly associated with all-cause mortality in females (HR 2.11, $p < 0.001$), not in males. Poor health behaviour, self-reported health and low income were the major predictors of mortality in both genders. When the effects of these factors were controlled, childhood family conflicts associated with mortality in both genders.
Conclusions: Among working-age people, females seem to be sensitive to the effects of numerous adverse childhood experiences, exhibiting higher premature all-cause mortality. Of the individual ACEs, family conflicts may increase risk of premature mortality in both genders. The effect of ACEs on premature mortality may partly be mediated via poor adult health behaviour and low socioeconomic status.
What is already known: In birth cohort studies, adverse childhood experiences (ACEs) have been associated with all-cause mortality. In working-age people, the association of ACEs with premature mortality is less clear and may differ between the genders.

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What this study adds: In working-age people, high number of ACEs associate with all-cause premature mortality in females, not in males. The effect of ACEs on premature mortality may partly be mediated via poor adult health behaviour, self-reported health and low socioeconomic status.

1. Introduction

Adverse childhood experiences (ACEs) affect individuals' subsequent physical and mental health as well as well-being and competence in profound and long-lasting ways (e.g., Hughes et al., 2017). ACEs are associated with poor overall health, various chronic diseases and risk factors for poor health and increased mortality (Brown et al., 2009; Felitti et al., 1998; Petruccelli et al., 2019). A systematic review suggested that childhood adversities were leading contributors to morbidity and mortality (Grummitt et al., 2021).

In a British birth cohort study, males and females who experienced numerous childhood adversities had a higher risk of death compared to individuals with no or few ACEs (Kelly-Irving et al., 2013). In a Swedish birth cohort, the involvement of child welfare services, household dysfunction, and disadvantageous family socioeconomic conditions associated with premature all-cause mortality between the ages of 19 and 65 years (Jackisch et al., 2019). Meanwhile, a Danish cohort study found that, compared with children with low adversity trajectory, those who experienced early-life material deprivation, persistent deprivation, or loss or threat of loss within the family had a moderately higher risk of premature mortality (Rod et al., 2020). In an American birth cohort assessing thirteen adverse childhood experiences clustered into 5 clusters, compared with the low ACE cluster, the clusters of family instability, poverty and crowded housing, and poverty and parental separation clusters were associated with premature mortality (Yu et al., 2022). In a population sample of 3646 young individiuals aged 18 to 30, who were followed for a median of 31 years, high number of childhood adversities were associated with all-cause mortality, even after controlling for age, sex, race, recent unemployment, smoking status, participant's education level, and highest parental education level (Pierce et al., 2020).

Previous long-term birth cohort studies have demonstrated a positive association between ACEs and all-cause mortality. However, the contribution of ACEs to premature mortality among working-age people, in the presence of other risk factors, is less clear. Interventions targeting at individuals' ACEs are expected to be more effective in working-age adults than in older people, as there is more time for corrective measures to mitigate possible detrimental effects of ACEs. Moreover, individuals older than 64 years may exhibit more ACE recall errors than younger individuals (Pirkola et al., 2005). Furthermore, considering that the effects of ACEs on mortality rates may vary between genders, separate analyses on women and men are necessary.

In the present study, we investigated the effect of ACEs on all-cause mortality in a nationally representative sample of adults aged 30 to 64 years, followed for 20 years. Because we were interested in premature death, individuals aged 65 years or more were excluded. Analyses were conducted for all participants and for males and females separately, with and without controlling for previously known mortality risk factors (e.g., Carlsson et al., 2014; Desalvo et al., 2006; Giovino et al., 2012; Mansson & Rastam, 2001; Müezzinler et al., 2015; Walter et al., 2012), including smoking, alcohol abuse, self-reported health, and economic situation (defined by family income relative to expenditure, hereafter referred to as income), at baseline examination.

2. Methods

This study was based on a multidisciplinary epidemiological survey in Finland, Health 2000, carried out by the Finnish Institute for Health and Welfare (THL, previously National Public Health Institute; https://thl.fi/en/web/thlfi-en/research-and-development/research-and-projects/health-2000-2011, https://www.julkari.fi/bitstream/handle/10024/78185/2008b26.pdf?sequence=1&isAllowed=y). In the Health 2000 study, a regionally stratified sample representing the Finnish population aged 30 years or older ($n = 8028$) was selected, with 88 % of these individuals participating in the interview. As we were interested in premature mortality among working-age people, participants aged 65 years or older were excluded. Following this exclusion, 5871 subjects were included in the sample.

All subjects received a basic questionnaire with questions about childhood adverse experiences (ACEs). For ACEs, response options were "no" (=0), "yes" (=1), or "cannot say" (=2). The questionnaire included the following questions: "When you think about your growth years i.e. before you were aged 16....

1. Did your family have long-term financial difficulties?, 2. Was your father or mother often unemployed although they wanted to work?, 3. Did your father or mother suffer from some serious disease or disability?, 4. Did your father have alcohol problems?, 5. Did your mother have alcohol problems?, 6. Did your father have any mental health problem, e.g., schizophrenia, other psychosis, or depression?, 7. Did your mother have any mental health problem, e.g., schizophrenia, other psychosis, or depression?, 8. Were there serious conflicts within your family?, 9. Did your parents divorce?, 10. Were you yourself seriously or chronically ill? and 11. Were you bullied at school?" The sum of "yes" answers (0−11) represented the degree of childhood adversity. Retrospective reports of adverse childhood experiences have been found to have good test–retest reliability (Dube et al., 2004). Previously, using the Health 2000 data, Kestilä et al. (2006) and Pirkola et al. (2005) examined the association between ACEs and adult self-reported health and distress and adult mental disorders.

Of the 5871 subjects aged 30 to 64 years, 4981 (85 %) returned the questionnaires, including the ACE questions. These respondents constitute the sample for the present study.

All-cause mortality data for all participants were obtained from the national cause-of-death register (Official Statistics of Finland: Causes of death). The follow-up period lasted from study entry (2000) to each participant's death or to the end of the follow-up period

(2020), whichever came first. We considered gender and age as potential confounders while tobacco smoking (never, earlier, incidentally/daily), alcohol abuse (number of hangovers during the previous year), self-reported health (1 = good to 5 = poor), and family income (1 = more than enough to 5 = we do not manage with our own income) were potential mediators - all of which have been associated with mortality. The original questions are included in the Supplementary Material.

2.1. Statistical analyses

First, we calculated the proportion of deaths during the 20-year follow-up (%) according to gender, age and other background characteristics. We used Chi-square (χ^2) tests to compare ACE items and their sum.

In the Cox regression analysis, the hazard rate for mortality was calculated based on the number of ACEs. These were classified in two categories: 0–3 and 4–11. The analysis was conducted both with and without controlling for gender, age, smoking, alcohol abuse, self-reported health and income. Age, alcohol abuse, self-reported health and income were analysed as continuous variables. Data analysis was conducted using SPSS (Statistical Package for the Social Sciences) software (28.0 for Windows). *P* values below 0.05 (two-tailed) were considered statistically significant.

3. Results

In total, 597 (12.0 %) deaths were registered. Male gender, age, smoking, alcohol abuse, low income and poor self-reported health were associated with a higher proportion of deaths (Table 1). Among the individual ACEs, financial difficulties, parental unemployment and chronic illness, and the total number of ACEs were associated with mortality during the follow-up (Table 2). Females reported a higher number of ACEs (mean: 1.28, SD: 1.42) ($p = 0.003$) compared to males (mean: 1.16, SD: 1.42). Specifically, females more frequently reported parents' unemployment and alcohol problems, and family conflicts than males (Supplementary Material, Table 1).

The death rate did not increase monotonously with the number of ACEs. There was a clear increase in death rates between 3 and 4 ACEs. Therefore, in further classifications, the participants were classified into two categories: 0–3 vs. 4–11 ACEs. The prevalence of smoking, alcohol abuse, poor self-reported health and low income were associated with the number of ACEs, indicating that risk factors for death increased with the number of ACEs (Supplementary Table 2 and Fig. 1).

In Cox regression analyses, both the number of ACEs and dichotomized ACEs (0-3/4-11) were significantly associated with

Table 1
Proportion of deaths among the 30 to 64-year-old participants during the follow-up period by background characteristics.

	N	Deaths	%	P
Gender				<0.001
Male	2357	383	16.2	
Female	2624	214	8.2	
Age				<0.001
30–34	708	16	2.3	
35–39	756	36	4.8	
40–44	742	41	5.5	
45–49	822	84	10.2	
50–54	834	136	16.3	
55–59	574	111	19.3	
60–64	545	173	31.7	
Smoking[a]				<0.001
No/Earlier	1361	124	9.1	
Incidentally/Daily	3601	469	13.0	
Alcohol abuse (Number of hangovers/12 months[a]				<0.001
No	2582	321	12.4	
1–2	1040	90	8.7	
3–6	783	93	11.9	
7–12	352	50	14.2	
13+	224	43	19.2	
Sufficiency of income[a]				<0.001
More than enough	821	64	7.8	
Enough	2076	220	10.6	
Some problems with income	1296	160	12.4	
Considerable problems with income	569	97	17.0	
Do not manage with income	157	45	28.7	
Self-reported health[a]				<0.001
GoodRather	1898	125	6.6	
Rather good	1554	161	10.4	
Moderate	1130	185	16.4	
Rather poor	285	89	31.2	
Poor	90	32	35.6	

[a] Original questions in Supplementary Material.

Table 2
Proportion of deaths among the 30 to 64-year-old participants during the follow-up period by adverse childhood experiences (ACEs) and significance (p) for risk of all-cause mortality in cox-regression analysis.

	N	Deaths	%	P
Adverse childhood experiences				
1. Financial difficulties	1070	161	15.0	0.001
2. Parents' unemployment	313	53	16.9	0.006
3. Parents' diseases	1146	151	13.2	0.162
4. Father's alcohol problems	810	102	12.6	0.565
5. Mother's alcohol problems	79	7	8.9	0.425
6. Father's mental problems	154	20	13.0	0.722
7. Mother's mental problems	166	16	9.6	0.326
8. Family conflicts	841	98	11.7	0.743
9. Parents divorced	506	65	12.8	0.548
10. Subject's chronic illness	251	55	21.9	<0.001
11. Subject bullied at school	764	89	11.6	0.757
Sum of ACEs				
0	2134	240	11.2	0.013
1	1225	141	11.5	
2	764	95	12.4	
3	416	48	11.5	
4+	442	73	16.5	
All	4981	597	12.0	

Original ACE questions: 1. Did your family have long-term financial difficulties?, 2. Were your father or mother often unemployed although they wanted to work?, 3. Did your father or mother suffer from some serious disease or disability?, 4. Did your father have alcohol problems?, 5. Did your mother have alcohol problems?, 6. Did your father have any mental health problem e.g. schizophrenia, other psychosis or depression?, 7. Did your mother have any mental health problem e.g. schizophrenia, other psychosis or depression?, 8. Were there any serious conflicts within your family?, 9. Did your parents get divorced?, 10. Were you seriously or chronically ill? and 11. Were you bullied at school?

mortality in all participants (Table 3 and Fig. 1) and among females when analysed separately. However, when adjusting for potential mediators, the association between the cumulative number of ACEs and mortality was not significant, suggesting that the effect of ACEs was largely mediated via these mediators. Nonetheless, the effect of dichotomised number of ACEs remained significant. In males, ACEs had no significant association with mortality. Further analyses showed that of the potential mediators, most strongly self-reported health and income, reduced the effect of ACEs on mortality. Interestingly, income was stronger mediator in females than in males (Supplementary Material, Table 3).

ACEs frequently co-occurred, one third of all participants reporting two or more ACEs (Table 2). In stepwise Cox regression analyses, among the individual ACEs, parental divorce (for all participants and males), chronic illness (for females), mother's alcohol problems (for males) and family conflicts (for females) were associated with mortality (Supplementary Material, Table 4). When the effects of potential mediators were taken into account, chronic illness (for females) and parental divorce (for all participants and males separately) were significantly associated with mortality. In all participants, the effect of childhood chronic illness was no longer significant ($p = 0.056$) due to the effects of self-reported health and income.

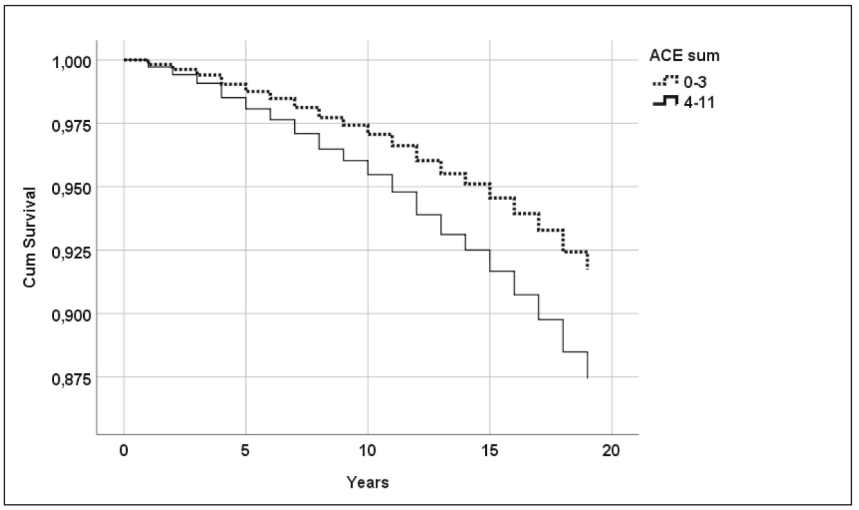

Fig. 1. Survival curve for classified adverse childhood experiences (ACE sum: 0-3/4-11 events). Effects of gender and age controlled.

Table 3
Cox regression analyses for the effect of adverse childhood adversities (ACEs) on mortality risk for the total sample and males and females separately.

	All			Male			Female		
A 1.	HR	CI95%		HR	CI95%		HR	CI95%	
ACEs	**1.080**	**1.026**	**1.136**	1.056	0.988	1.130	**1.114**	**1.032**	**1.203**
Gender (male)	**2.166**	**1.832**	**2.561**						
Age	**1.091**	**1.080**	**1.101**	**1.090**	**1.077**	**1.103**	**1.092**	**1.074**	**1.109**
A 2.	HR	CI95%		HR	CI95%		HR	CI95%	
ACEs	1.020	0.968	1.076	1.001	0.935	1.073	1.047	0.965	1.137
Gender (male)	**1.834**	**1.534**	**2.193**						
Age	**1.089**	**1.078**	**1.101**	**1.089**	**1.075**	**1.103**	**1.089**	**1.070**	**1.108**
Smoking	**1.351**	**1.097**	**1.662**	**1.744**	**1.258**	**2.417**	1.056	0.792	1.408
Alcohol abuse	**1.050**	**1.025**	**1.075**	**1.039**	**1.012**	**1.066**	**1.104**	**1.046**	**1.166**
Self-reported health	**1.271**	**1.173**	**1.377**	**1.229**	**1.114**	**1.357**	**1.354**	**1.179**	**1.555**
Income	**1.266**	**1.167**	**1.373**	**1.303**	**1.178**	**1.442**	**1.189**	**1.037**	**1.362**
B 1.	HR	CI95%		HR	CI95%		HR	CI95%	
ACE 4-11/0-3	**1.554**	**1.217**	**1.986**	1.255	0.898	1.755	**2.108**	**1.466**	**3.030**
Gender (male)	**2.160**	**1.827**	**2.554**						
Age	**1.091**	**1.080**	**1.101**	**1.090**	**1.077**	**1.103**	**1.092**	**1.075**	**1.110**
B 2.	HR	CI95%		HR	CI95%		HR	CI95%	
ACE 4-11/0-3	**1.303**	**1.017**	**1.670**	1.097	0.782	1.537	**1.642**	**1.128**	**2.388**
Gender (male)	**1.844**	**1.542**	**2.204**						
Age	**1.089**	**1.078**	**1.101**	**1.089**	**1.075**	**1.103**	**1.089**	**1.070**	**1.108**
Smoking	**1.346**	**1.094**	**1.656**	**1.742**	**1.257**	**2.414**	1.051	0.788	1.402
Alcohol abuse	**1.049**	**1.025**	**1.074**	**1.038**	**1.012**	**1.066**	**1.096**	**1.038**	**1.157**
Self-reported health	**1.266**	**1.169**	**1.372**	**1.226**	**1.112**	**1.353**	**1.350**	**1.176**	**1.549**
Income	**1.265**	**1.167**	**1.371**	**1.303**	**1.179**	**1.441**	**1.180**	**1.030**	**1.351**

A1: analyses for the effect of the number of ACEs when the effects of gender and age are controlled; A2: analyses for the effect of the number of ACEs when the effects of gender, age, smoking, alcohol abuse, self-reported health and income are controlled. B1: analyses for effect of the classified number of ACEs when the effects of gender and age are controlled; B2: analyses for the effect of classified number of ACEs when the effects of gender, age, smoking, alcohol abuse, self-reported health and income are controlled.
B2: analyses for the effect of classified number of ACEs when the effects of gender, age, smoking, alcohol abuse, self-reported health and income are controlled.
Bolded significant ($p < 0.05$).

4. Discussion

Consistent with long-term follow-up birth cohort studies (Jackisch et al., 2019; Kelly-Irving et al., 2013; Rod et al., 2020; Yu et al., 2022), we found that retrospectively reported ACEs also predicted all-cause mortality among the working-age population during a 20-year follow-up period. However, this association was considerably reduced after accounting for common mortality predictors, including self-reported health, tobacco smoking, alcohol abuse, and financial situation (family income) (Carlsson et al., 2014; Desalvo et al., 2006; Giovino et al., 2012; Mansson & Rastam, 2001; Müezzinler et al., 2015; Walter et al., 2012). This suggests that the effect of ACEs on mortality was partly mediated through poor health behaviour, self-reported health and, particularly in females, though socioeconomic conditions in adulthood. The strong associations of childhood financial difficulties and parents' unemployment with mortality became non-significant when age and potential mediators were included in the Cox regression model. In a comparable study with a younger population (Pierce et al., 2020), the association of ACEs with cardiovascular mortality became nonsignificant after adjusting for cardiovascular risk factors, while the association of ACEs with all-cause mortality remained significant.

However, in this study, the association of ACEs with mortality was significant only among females. In males, the direct effects of potential mediators, such as tobacco smoking and socioeconomic status (income), were more pronounced, whereas in females, the effect of ACEs on mortality was strongly mediated through income. This aligns with previous studies (Chen et al., 2016; Lee & Ryff, 2019), which found that females were more sensitive to the long-term detrimental effects of multiple ACEs. Contrarily, Lee and Ryff (2019) found that besides material and psychological resources, an unhealthy lifestyle, indicated by tobacco smoking, high body mass index and lack of physical exercise, was significantly associated with mortality in both sexes. The discrepancy between these two studies may be attributed to differing definitions of health behaviour. In our study, low socioeconomic status and tobacco smoking were the primary factors explaining premature mortality, especially in males. Tobacco smoking has been the biggest cause of adult death in developed countries (Boyle, 1997).

In line with the impact of chronic illness on mortality in the adults (Walter et al., 2012), we found that childhood chronic illness was associated with premature death in our adult population, particularly in females. Childhood chronic illness strongly associated with poor self-reported health in adulthood, which, in turn, predicted increased mortality. However, its effect on mortality was also partly mediated through lower income. Thus, childhood chronic illness seems to associate with premature death both directly and indirectly

via reduced income in adulthood.

Additionally, other ACEs such as various family problems, including parental divorce and family conflicts (shortly family conflicts), were linked with premature death. These findings highlight the importance of a stable and emotionally supportive family environment in the development of children and adolescents, influencing their education, relationship formation and mortality risk (Bereczkei & Csanaky, 2001). Such an environment forms the basis of individuals' mental and physical health in adulthood and may even enhance their survival.

Gender is well known to be strongly associated with mortality, with male mortality rates typically higher than female mortality rates (i.e., Wang et al., 2020). Although females reported more ACEs compared to males, their mortality was lower. In terms of mortality, females seemed to be more sensitive to the long-term effects of multiple ACEs than males. It is noteworthy that of the individual ACEs, females reported more parental unemployment and alcohol problems. Since it is unlikely that these particular childhood adversities were dependent of children's gender, this finding may indicate some sort of bias towards observation, recall, or reporting between genders, and emphasises the need for analysing also males and females separately.

In contrast, in males, a poor childhood socioeconomic status, coupled with adult unhealthy behaviours and a low socioeconomic status seemed to have a stronger effect on premature mortality. However, the significant association between childhood financial difficulties and mortality in males was reduced after controlling for the effect of age, as older males more frequently reported childhood financial difficulties. Thus, socioeconomic disadvantages during both childhood and adulthood may aggravate age-dependent premature mortality, particularly in males.

Self-reported health was also associated with all-cause mortality. Previous studies have shown that self-reported health independently predicts mortality (Desalvo et al., 2006) and has an additive effect with objective health in mortality prediction (Viljanen et al., 2021). In our study, ACEs predicted self-reported health (Supplementary Material, Table 2) indicating that the effect of ACEs on mortality was partly mediated via self-reported health.

The relationship between ACEs and premature mortality is most likely complex and spans multiple generations. ACEs may trigger a cascade of stress-mediated effects on increased systemic inflammation, hypothalamic–pituitary–adrenal (HPA) axis dysfunction (e.g., glucocorticoids and vasopressin-oxytocin), neurotransmitters, neurological, neurodegenerative, inflammatory, and autoimmune diseases and epigenetic modifications, such as increased methylation, and shorter telomere length in adulthood (Jiang et al., 2019; McGowan et al., 2009; Morris et al., 2019; Soares et al., 2021; Teicher et al., 2002). These factors are associated with poor health outcomes and premature mortality. For example, telomeres, which are located at the ends of chromosomes and act as a molecular clock for cellular ageing, have been studied in this context. A prospective study showed that children exposed to two or more types of violence exhibited increased telomere erosion, a marker of premature cellular ageing, compared to those not experienced violence (Shalev, Entringer, et al., 2013; Shalev, Moffitt, et al., 2013). Pre- and perinatal stress, such as that experienced by pregnant women who suffered intimate partner violence, has been also associated with the telomer shortening in their newborns (Chan et al., 2019). Therefore, telomere erosion may partially explain why individuals with ACEs exhibit increased premature mortality (Brown et al., 2009).

Interestingly, gender differences exist in the effects of ACEs on HPA axis function. In non-symptomatic subjects, ACEs were more strongly associated with corticotrophin-releasing factor levels in males than in females (DeSantis et al., 2011). In physically maltreated girls, exposure to a speech stressor correlated with increased oxytocin levels and blunted cortisol levels. However, in boys or non-maltreated girls, no such changes were found in oxytocin levels were observed (Seltzer et al., 2014). Therefore, severely maltreated females may be more sensitive to adult stress-situations, while in males, poor health behaviour, partly influenced by childhood adversities, may have a more significant role in premature mortality.

Our study has both strengths and limitations. The Health 2000 sample is representative of the Finnish population aged 30 to 64 years, and the response rate of the ACE inquiry was substantial (85 %). Thus, our sample represents well the working-age population. The prospective study design and large sample size are notable strengths.

The ACE items represent objective events, which typically have a weaker association with outcome factors than subjective experiences. This aspect could be seen as a limitation or an advantage, depending on the perspective. An objective list may provide more reliable information than a subjective questionnaire on self-reported childhood experiences. However, we were unable to study all ACEs, such as emotional, physical or sexual abuse or emotional neglect, potentially limiting our findings on the effect of ACEs on mortality. Moreover, the duration of ACEs was not available for analysis, preventing us from evaluating its effect in later life. Future studies focusing on subjective ACEs across difference life stages are needed.

5. Conclusions

In our working-age population study, ACEs predicted all-cause mortality on 20 years follow-up in females, but not in males. These results suggest that females may be more vulnerable to the effects of multiple adverse childhood experiences, exhibiting higher rate of premature all-cause mortality. The effect of ACEs on premature mortality may partly be mediated through poor adult health behaviour and low socioeconomic status. Considering these factors, childhood family conflicts may increase risk of adult mortality in both genders.

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None.

Ethical standards

The study had approval of the Ethics Committee of the Hospital District of Helsinki and Uusimaa. Participants provided written informed consent.

CRediT authorship contribution statement

Raimo K.R. Salokangas: Writing – review & editing, Writing – original draft, Methodology, Formal analysis, Data curation, Conceptualization. **Henri R.W. Salokangas:** Writing – review & editing, Writing – original draft, Methodology, Conceptualization. **Tiina From:** Writing – review & editing, Software, Project administration, Data curation. **Lara Lehtoranta:** Writing – review & editing. **Anne Juolevi:** Writing – review & editing, Methodology, Data curation. **Jarmo Hietala:** Writing – review & editing, Writing – original draft, Supervision, Methodology. **Seppo Koskinen:** Writing – review & editing, Resources, Conceptualization.

Declaration of competing interest

The authors declare no conflict of interest.

Data availability

The authors do not have permission to share data.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.chiabu.2024.106838.

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