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Adverse Childhood Experiences, Insomnia, and Depressive Symptoms in Later Life: Moderation Effect of Loneliness but Not Hair Cortisol

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ABSTRACT

Objectives: Adverse childhood experiences (ACEs) are established risk factors for developing depression in adulthood, although the mechanisms of this association are yet to be fully elucidated. In this study, we tested whether insomnia (i.e., difficulties in sleep onset and maintenance) can mediate the association between ACEs and adult depressive symptoms, and whether loneliness and hair cortisol, reflecting hypothalamic-pituitary-adrenal axis activity, can act as moderators.

Methods: We analyzed data of 1593 participants (64.7% female) aged 65.25 ± 8.15 from the English Longitudinal Study of Ageing (ELSA) across three waves of data collection. ACEs were retrospectively assessed in 2008–2009 (wave 4), insomnia symptoms, loneliness, and hair cortisol in 2012–2013 (wave 6), while depressive symptoms were assessed in 2014–2015 (wave 7).

Results: After accounting for health-related confounders and baseline values, conditional process analysis showed that insomnia symptoms exerted a mediating role between ACEs (ACEs total, parental bonding, and household dysfunction) and depressive symptoms, with a stronger effect in lonely older adults. Hair cortisol did not moderate the association between ACEs and insomnia symptoms.

Conclusion: Results are consistent with the view of insomnia as a mechanism linking ACEs to depressive symptoms later in life. Elderly experience of loneliness may further increase the mediatory role of insomnia between ACEs and depression.

1 | Introduction

Adverse childhood experiences (ACEs) include exposure to several types of abuse and neglect during early life and adolescence, including physical, emotional, and sexual abuse and physical and emotional neglect (Bernstein et al. 1998; Iob et al. 2020a). Forty-seven to 64% of Western adults report at least one ACE, while 9%–17% report four or more ACEs (Bellis et al. 2014; Swedo et al. 2023). The detrimental effects of ACEs on psychological outcomes can linger throughout the lifespan (Petruccelli et al. 2019). Particularly, meta-analytic estimates suggest a risk of adult depression between 1.34 and 3.17 OR (e.g., Tan and Mao 2023). The detrimental impacts of ACE can also persist into later years of life (Sachs-Ericsson et al. 2011; Dias et al. 2017; Lin and Chiao 2020; Ernst et al. 2021; Fu and Chen 2023), as suggested by the life cycle model of stress (Lupien et al. 2009), which essentially indicates that late-onset disorders might reflect "delayed" effects of early life stress. These effects are particularly significant during critical periods of life such as the elderly (Clark and Manini 2010) which bring various challenges related to psychophysical health (e.g., income changes, cognitive decline, and heightened awareness of mortality; Charles and Carstensen 2010). These factors contribute to increased frailty and vulnerability, potentially triggering the onset of psychopathology in later life for individuals who experienced ACEs (Van Assche et al. 2020; Ní Mhaoláin et al. 2012).

Cumulative evidence relates depression in the elderly to various forms of early-life adversities (e.g., emotional and physical neglect; Ege et al. 2015) with a dose-dependent trend (e.g., Raposo et al. 2014), but the underlying mechanism of these associations remains unclear. A range of mediators have been hypothesized to play a role in the ACEs-depression association, including psychological mechanisms such as dysfunctional beliefs and maladaptive interpersonal schema (e.g., Boyda et al. 2018; Aafjes-van Doorn et al. 2020), social factors such as lack of social support (Kobrinsky and Siedlecki 2023), and biological factors such as immune (e.g., Zagaria et al. 2024) and autonomic (Winzeler et al. 2017) dysregulation.

Another putative psychophysiological mediator between ACEs and depression is insomnia, that is, difficulties in sleep onset and/or maintenance (Kajeepeta et al. 2015; Liu et al. 2023). Up to 46% of adult patients with insomnia report history of ACEs (Bader et al. 2007). ACEs at 9 years-old have been associated with difficulties falling asleep at 15 (Rojo-Wissar et al. 2021), and the number of ACEs showed a dose–response association with insomnia symptoms in adolescence (Desch et al. 2023). Similar results have been reported in middle adulthood samples (e.g., Geng et al. 2021). Moreover, it is well-known from meta-analyses of longitudinal epidemiological literature that the presence of insomnia prospectively increases the risk of developing mood disturbances, and particularly depression in adult (Baglioni et al. 2011; Hertenstein et al. 2019) and elderly samples (e.g., Perlis et al. 2006). Mediation studies in this field suggested that insomnia may be a potential mechanism linking ACEs to adult depression. In fact, in several cross-sectional studies insomnia has emerged as a mediating factor in the association between ACEs and depressive symptoms (e.g., Conway et al. 2020; Luo et al. 2022; Laskemoen et al. 2021). Furthermore, in longitudinal studies (e.g., Guo et al. 2023), specific sleep parameters, such as sleep duration, have been identified as mediators between ACEs and the onset of depressive symptoms at 1-year follow-up. Consistently, in the China Health and Retirement Longitudinal Study (CHARLS), involving middle-age and older adults, ACEs measured in 2014 were significantly associated with depressive symptoms at 4-year follow-up, with insomnia symptoms significantly mediating this association (Yin et al. 2023).

Regardless, several points still require clarification. Previous mediation studies on ACEs, insomnia, and depressive symptoms have been mostly focused on cross-sectional data, which does not allow to properly estimate the temporal dynamics between the variables under study (Zagaria et al. 2023). Moreover, available evidence was mainly collected in predominantly young samples (Conway et al. 2020; Luo et al. 2022; O'Neill et al. 2024; Laskemoen et al. 2021; Li, Wang et al. 2023), despite a possible increase in insomnia (Klimt et al. 2023) and depressive symptoms (e.g., Sutin et al. 2013; Lee 2020) in older adulthood. If specific ACEs may influence depressive symptoms through the mediation of insomnia more than others, also remain obscure. Also, whether ACEs may have a stronger impact on insomnia in individuals with abnormalities in hypothalamic-pituitary-adrenal (HPA) axis functioning is also yet to be determined. In fact, it has been proposed that early-life adversities may dysregulate HPA axis and predispose to the development of insomnia (Palagini et al. 2015; Reffi et al. 2022) and psychiatric symptoms (Drake et al. 2017) later in life.

Finally, the interaction of ACE-related insomnia with other frequently recognized predictors of depression remains to be explored. A variable that often co-occur with both insomnia and depression is loneliness (Hom et al. 2017), which is defined as the negative perception of personal deficient social relationships (Singh and Srivastava 2014). A recent pooled analysis of nine longitudinal studies revealed that loneliness trajectory across the lifespan follows a *U*-shaped curve, decreasing from young adulthood to midlife and increasing in older adulthood (Graham et al. 2024). Meta-analyses confirmed significant moderate to large associations of loneliness with insomnia (Hom et al. 2020) and depression (Park et al. 2020). Individuals experiencing higher loneliness may also be at higher risk of developing depressive symptoms in future compared to those with lower loneliness (Erzen and Çikrikci 2018), especially in those aged > 55 years (McClelland et al. 2020).

The aims of this study were to: (1) test the longitudinal mediation of insomnia symptoms in the relationship between ACEs and depressive symptoms in older adults; (2) test the potential moderating role of hair cortisol, reflecting HPA activity, on the association between ACEs and insomnia symptoms; and (3) test the potential interaction between insomnia symptoms and loneliness in influencing depressive symptoms.

2 | Materials and Methods

2.1 | Procedure

We analyzed data from the English longitudinal study of ageing (ELSA), a panel study of older adults aged 50 years and older living in England which began in 2002 (Zaninotto and Steptoe 2019; Banks et al. 2019). For the purpose of this study, three waves of data collection were considered: ACEs were retrospectively assessed in 2008/9 (wave 4); insomnia symptoms, hair cortisol, and loneliness were measured in 2012/2013 (wave 6); self-reported depressive symptoms were assessed in 2014/2015 (wave 7). These three waves were a priori selected to ensure consistency with the theoretical model under investigation, as hair cortisol data were only available at ELSA wave 6. Particularly, insomnia symptoms can be experienced in mid-life adults with ACEs history (e.g., Geng et al. 2021). Also, meta-analysis showed that insomnia was a risk factor for depression during the following 2 years (Baglioni et al. 2011).

2.2 | Participants

A total of 1593 participants with complete data for the variables of interest were included in the present investigation. Attrition analyses revealed small or negligible differences at baseline in age (Cohen's $d = 0.013$, $p = 0.619$), sex (φ coefficient = 0.076, $p < 0.001$), BMI (Cohen's $d = -0.010$, $p = 0.727$), depressive symptoms (Cohen's $d = -0.216$, $p < 0.001$), insomnia symptoms (Cohen's $d = -0.047$, $p = 0.083$), and ACEs (Cohen's $d = 0.035$, $p = 0.240$) between participants included in the analyses and those who were excluded due to missing data (i.e., Cohen's d and φ coefficient < |0.5| and < |0.3 | , respectively; Cohen 2013). The main reasons for missing data in ELSA include non-respondents (i.e., those who lack contact, refuse to participate,

or cannot be traced) and non-eligibility (i.e., deaths or relocation outside of Great Britain) (see Chapman et al. 2011).

The analytical sample was predominantly composed of females (i.e., 64.7%), with a mean age of 65.25 years (SD = 8.15). On average, participants reported a BMI value of 28.22 kg/m (SD = 5.12). Smoking was reported by 10.4% of the sample, while 9.1% reported having avoided drinking alcohol during the year before baseline assessment. Moreover, according to a cut-off score of ≥ 3 on the 8-item version of the CES-D (Steffick 2000), 15.3% of participants reported clinically significant depressive symptoms at baseline. This cut-off has been shown to yield a sensitivity of 71% and a specificity of 79% in identifying probable cases of depression, as defined by the Short Form Composite International Diagnostic Interview (CIDI-SF; Steffick 2000).

2.3 | Measures

2.3.1 | ACEs

ACEs were operationalized following Iob et al. (2020a), who identified four components of adverse childhood experiences based on ELSA data: threat (including sexual and physical abuse, and physical assault); household dysfunction (including parental arguments, parental mental illness or substance abuse, and parental separation or divorce); low parental bonding (including maternal and paternal bonding); and loss (including separation from mother, parental death, foster care or adoption, and institutionalization). The Life History Interview (Ward et al. 2009) was administered at wave 4 to assess ACEs experienced up to the age of 16 years. The interview is not characterized by a fixed number of items as it is administered using a technique called "feeding forward" data. It is a technique that reintroduces responses given by individuals in earlier interviews to either aid memory recall and/or improve consistency of responses across interviews. Moreover, based on the participant's previous answers some questions were asked, allowing the interviewer to explore relevant topics in more depth. The interview includes items that investigate sexual abuse, physical assault, physical abuse by parents, parental arguments, parental mental illness or substance abuse, parental separation or divorce, maternal bonding, paternal bonding, separation from the mother for more than 6 months, parental death, foster care or adoption, and institutionalization. Several studies provided evidence on the validity of the retrospective data collected with Life History Interview in ELSA study, stressing the quality and the importance of such data to study adverse childhood experiences (Banks et al. 2020). For all items, except for parental bonding, participants reported whether or not they had ever experienced that particular event during childhood. Child-parent relationships were assessed using the seven-item Parental Bonding Instrument (PBI) (Parker et al. 1979). This questionnaire is designed to retrospectively assess adults' perceptions of their parents' parenting styles. Total bonding scores were calculated separately for each parent figure and ranged from 0 (*highest bonding*) to 7 (*lowest bonding*).

Based on the dimensions of ACEs identified by Iob et al. (2020a), namely *Threat*, *Household Dysfunction*, *Parental Bonding*, and *Loss*, we derived an overall ACEs index consistent with a formative measurement approach (i.e., "constructs conceived as explanatory combinations of indicators that are determined by a combination of variables"; Fornell and Bookstein 1982, 292). More specifically, we applied principal component analysis (PCA) to the four-dimension scores, conceptualizing them as distinct contributors to a broader composite index of childhood adversity (Diamantopoulos and Winklhofer 2001). The first principal component, which explained 41.7% of the total variance, was retained. The resulting component scores—a weighted linear combination of the ACE dimensions—were extracted and used as a predictor in subsequent regression models (e.g., Vyas and Kumaranayake 2006). The weights assigned to each ACE dimension in the PCA-derived score are reported in Table S1. Compared to a traditional cumulative score, that is, an unweighted count variable ranging from 0 to 4, the PCA-derived index yielded a truly continuous variable, which aligns more closely with the assumptions of general linear models underlying further regression-based analyses. Moreover, as shown in Table S1, the weights assigned to the four ACE dimensions differed, reflecting their unequal contributions to the PCA-derived component. In contrast, cumulative scoring assumes equal weights across dimensions, potentially overlooking meaningful differences in how each domain contributes to overall childhood adversity.

2.3.2 | Insomnia Symptoms

Insomnia symptoms were evaluated at wave 6 using three items from the Jenkins Sleep Problems Scale (Jenkins et al. 1988). Items addressed the most frequent insomnia symptoms: difficulty falling asleep, frequent awakenings during the night, and feeling tired upon waking in the morning. Participants rated these items on a 4-point Likert scale ranging from "not at all during the past month" to "three or more times per week." The questions referred to the previous month, and the scores were summed so that higher scores indicate greater insomnia symptoms.

2.3.3 | Depressive Symptoms

Depressive symptoms at wave 7 were assessed using the 8-item version of the Centre for Epidemiological Studies-Depression scale (CES-D, Radloff 1977). The psychometric properties of the eight-item reduced version have demonstrated to be comparable to the original 20-item version (Steffick 2000). This includes questions about the occurrence (yes/no) of eight depressive symptoms such as feeling depressed or feeling sad for much of the time over the previous week to give a possible score range from 0 (*no symptoms*) to 8 (*all eight symptoms*). Higher scores in the CES-D indicate more severe depressive symptoms. Since the CES-D includes a single item measuring sleep disturbance (i.e., "Whether felt their sleep was restless during past week"), a sensitivity analysis was also performed by excluding this item from the CES-D total score calculation (e.g., Ballesio, Zagaria, Ottaviani et al. 2022).

2.3.4 | Loneliness

Subjective feelings of general loneliness were assessed using the brief version of the University of California Los Angeles

(UCLA) Loneliness Scale at wave 6. This scale consists of three questions: "How often do you miss company?" "How often do you feel isolated from others?" and "How often do you feel left out?" Each item is rated on a 3-point scale ranging from 0 (*Hardly ever or never*) to 3 (*Often*). The total UCLA score ranges from 0 to 9, with higher scores indicating greater loneliness. This measure has been validated among older adults (Neto 2014) and has demonstrated high internal consistency within ELSA (Cronbach's $\alpha = 0.83$) (Hawkley et al. 2020).

2.3.5 | Hair Cortisol Concentration

Hair cortisol was assessed at wave 6. A lock of hair at least 2 cm in length and weighing at least 10 mg was collected from the posterior vertex of all consenting participants, cut as close to the scalp as possible. Hair sampling was excluded for individuals with certain scalp conditions, those who were pregnant or breastfeeding, those unable to sit still with their head in a fixed position, and those with less than 2 cm of hair in the posterior vertex area. Hair washing and steroid extraction were performed using high-performance liquid chromatography-mass spectrometry (HPLC-MS), as outlined by Gao et al. (2013). Given that hair grows approximately 1 cm per month (Kintz et al. 2006), the 2 cm segment closest to the scalp represents the average cortisol levels over the 2 months preceding the sample collection.

2.3.6 | Covariates

Several covariates measured at baseline (i.e., wave 4) were included in the model due to their potential association with insomnia and/or depressive symptoms: gender, age, body mass index (BMI), alcohol use (ranging from "Not at all in the last 12 months" to "Almost every day"), frequency of vigorous sport activity (ranging from "Hardly ever or never" to "More than once a week"), and smoking (i.e., 0 = no, 1 = yes). Moreover, to disentangle the longitudinal component of the mediation effects (Jose 2016) and to effectively study overtime change, baseline insomnia and depressive symptoms were considered as control variables.

2.4 | Data Analysis

Data were analyzed using *Jamovi* version 2.3.18 (https://www.jamovi.org/).

Initially, descriptive statistics and bivariate correlations among the main variables under study were calculated. Subsequently, a conditional process analysis was conducted following three steps (see Figure 1). First, we investigated whether adverse childhood experiences (ACEs) predicted depressive symptoms at wave 7 through the mediating role of insomnia symptoms assessed at wave 6. Second, we examined whether hair cortisol levels moderated the relationship between ACEs and insomnia symptoms. Third, we evaluated whether loneliness moderated the association between insomnia and depressive symptoms.

Before creating the interaction terms, predictors were mean-centered following the recommendations of Aiken et al. (1991). The Johnson and Neyman (1936) technique was then employed

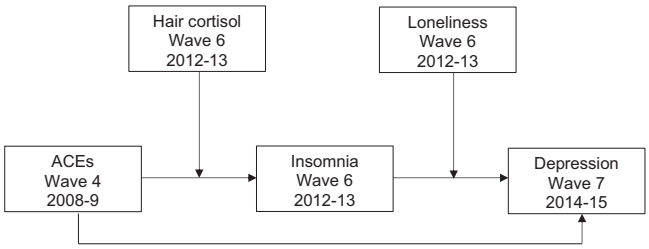

FIGURE 1 | The proposed model. Covariates were not depicted to avoid clutter, that is, sex, BMI, smoking, alcohol use, physical activity, age, baseline insomnia, and baseline depressive symptoms. ACEs, adverse childhood experiences.

to identify the specific values of the moderator at which the effect of insomnia on depressive symptoms turned statistically significant. In addition, simple slope analyses were conducted at the 25th and 75th percentiles of the moderator to further probe the nature of the interaction. Mediated effects were formally tested by calculating 95% bias-corrected bootstrap confidence intervals (BCI; 5000 replications) as suggested by MacKinnon et al. (2002). Effect sizes were expressed as standardized regression coefficients (β).

All models were controlled for baseline insomnia and depressive symptoms (i.e., wave 4), as well as for health-related covariates (see Paragraph "Covariates"). Further exploratory analyses were conducted by considering each ACE subtype (i.e., threat, loss, parental bonding, household dysfunction) separately as predictors in the conditional process modeling.

3 | Results

3.1 | Descriptive Statistics and Bivariate Correlations

Descriptive statistics and bivariate correlations among the main variables are reported in Table 1. ACEs, loneliness, insomnia and depressive symptoms significantly correlated in the expected direction (r range 0.163–0.382, $p < 0.001$). The absolute values of the correlation coefficients indicated no meaningful concerns regarding multicollinearity among the main variables (Shrestha 2020). Multicollinearity was further assessed using Variance Inflation Factor (VIF) values for all focal predictors and covariates, which ranged from 1.015 to 1.740, substantially below the conservative threshold of 5 (e.g., Kim 2019).

3.2 | Conditional Process Analysis

As a first step, we examined the mediating role of insomnia symptoms in the relationship between ACEs and depressive symptoms. Findings showed that ACEs were positively related to insomnia symptoms at wave 6 ($\beta = 0.067$, $p = 0.001$), which in turn were associated with depressive symptoms at wave 7 ($\beta = 0.162$, $p < 0.001$). ACEs were also directly related to depressive symptoms at wave 7 ($\beta = 0.063$, $p = 0.003$). Importantly, insomnia symptoms exerted a mediating role in the relationship between ACEs and depressive symptoms ($\beta = 0.011$, 95% BCI 0.007–0.032).

TABLE 1 | Descriptive statistics and bivariate correlations between main study variables.

	Mean (SD)	1	2	3	4	5	6	7	8
1. ACEs total (wave 4)	0 (1)								
2. Parental bonding	25.90 (6.01)	0.888**							
3. Household dysfunction	0.32 (0.61)	0.651**	0.370**						
4. Threat	0.15 (0.43)	0.485**	0.238**	0.210**					
5. Loss	0.20 (0.50)	0.401**	0.181**	0.180**	0.117**				
6. Hair cortisol (wave 6)	0.89 (0.57)	0.014	0.009	−0.003	0.030	0.009			
7. Insomnia symptoms (wave 6)	6.89 (2.53)	0.163**	0.138**	0.121**	0.085**	0.060*	0.029		
8. Loneliness (wave 6)	4.07 (1.46)	0.196**	0.169**	0.125**	0.125**	0.065*	0.010	0.274**	
9. Depressive symptoms (wave 7)	1.14 (1.63)	0.165**	0.116**	0.152**	0.084**	0.096**	0.029	0.348**	0.402**

Note: To normalize the shape of its distribution, hair cortisol was log-transformed (see Tabachnick and Fidell 2007). ACEs total was a weighted score derived from a principal component analysis on the ACEs subtypes.
*$p < 0.05$; **$p < 0.01$.

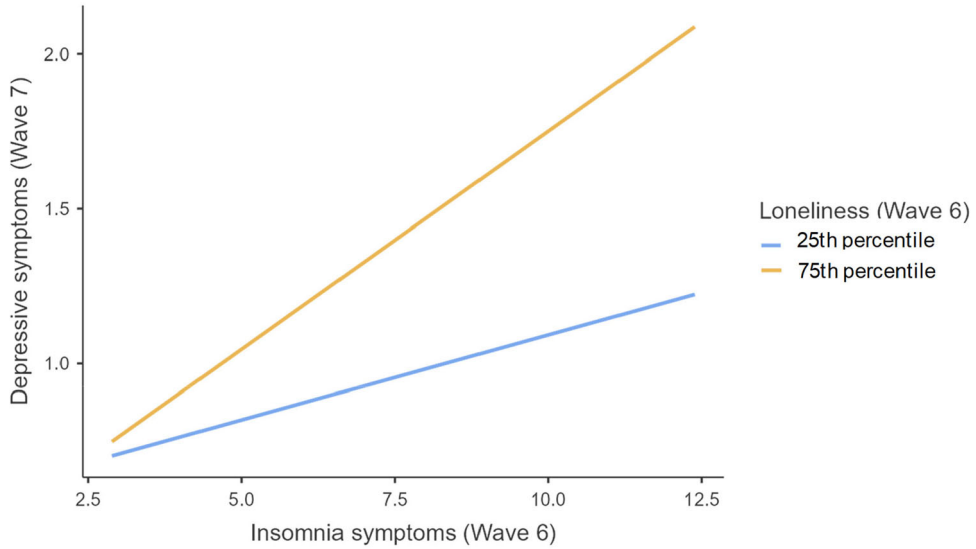

FIGURE 2 | Plot of the relationship between insomnia and depressive symptoms conditioned at the 75th (i.e., 5) and 25th (i.e., 3) percentiles of loneliness.

In the second step, we examined the moderating role of hair cortisol in the relationship between ACEs and insomnia symptoms. Findings showed that hair cortisol did not moderate such a path ($\beta = -0.024$, $p = 0.258$); therefore, the interaction effect was trimmed in further analyses to maintain parsimony.

In the third step, we examined the moderating role of loneliness in the relationship between insomnia and depressive symptoms. Findings showed that loneliness significantly moderated such a path ($\beta = 0.097$, $p < 0.001$). Specifically, the effect of insomnia on depressive symptoms increased by 0.097 standardized units for each one standard deviation increase in loneliness. The Johnson-Neyman plot (see Figure S1) revealed that the effect of insomnia on depressive symptoms reached statistical significance when loneliness exceeded a hypothetical value of 2.68. However, because the minimum observed score in the data set was 3, this value fell outside the actual data range. Therefore, the effect of sleep on depression could be considered significant across all observed values of loneliness. To avoid extrapolation to unobserved values, we conducted simple slopes analysis at the 25th and 75th percentiles of loneliness (i.e., scores of 3 and 5, respectively; see Figure 2). This approach allowed us to probe the interaction within the observed distribution of the moderator. Although we reported simple slopes to illustrate the nature of the interaction, it is important to note that the significance of the interaction term itself provided the primary evidence for moderation, whilst the simple slopes were secondary probes and should be interpreted accordingly. Specifically, simple slopes showed that the effect of insomnia on depression strengthened as loneliness increased, with a modest effect at the 25th percentile ($\beta = 0.085$, $p = 0.005$) and a stronger effect at the 75th percentile ($\beta = 0.219$, $p < 0.001$).

Additionally, we further tested the indirect effect linking ACEs to depressive symptoms through insomnia conditioned at high and low values of loneliness (i.e., simple mediated effects). Specifically, the mediating effect of insomnia symptoms was stronger at high (75th percentile; $\beta = 0.015$, 95% BCI [0.0095, 0.0429]) compared to low (25th percentile; $\beta = 0.006$, 95% BCI [0.0024, 0.0198]) levels of loneliness. Overall, the model

explained 36.1% of the variance of insomnia (wave 6) and 32.6% of the variance of depressive symptoms (wave 7). These estimates reflect the combined contribution of the main predictors, covariates, and the rank-order stability of the outcomes overtime. Significant covariate effects are reported in the Supplementary Document.

A further sensitivity analysis, conducted by excluding the sleep item of the CES-D from the total score calculation, replicated the moderation effect. Full results are detailed in the Supplementary Document.

3.3 | Exploratory Analyses on ACEs Subtype

Four conditional process analyses were further conducted considering each ACE subtype as a predictor (i.e., *threat*, *loss*, *parental bonding*, *household dysfunction*). Findings were replicated when examining parental bonding and household dysfunction, but not when considering threat and loss subdimensions.

In detail, both *parental bonding* ($\beta = 0.052$, $p = 0.012$) and *household dysfunction* ($\beta = 0.065$, $p = 0.002$) were significantly associated with insomnia symptoms. The interaction effects between insomnia symptoms and loneliness in predicting depressive symptoms were also significant (*parental bonding*: $\beta = 0.098$, $p < 0.001$; *household dysfunction*: $\beta = 0.096$, $p < 0.001$). Conditional indirect effects revealed that parental bonding was indirectly associated with depressive symptoms via sleep disturbance at high levels of loneliness ($\beta = 0.012$, 95% BCI [0.0006, 0.0059]) and, to a lesser extent, at low levels ($\beta = 0.005$, 95% BCI [0.0002, 0.0032]). Similarly, household dysfunction was indirectly associated with depressive symptoms via sleep disturbance at both high ($\beta = 0.014$, 95% BCI [0.0123, 0.0663]) and low ($\beta = 0.005$, 95% BCI [0.0031, 0.0340]) levels of loneliness, with a stronger effect observed at higher levels.

When considering the *threat* and *loss* subdimensions, neither *threat* ($\beta = 0.018$, $p = 0.394$) nor *loss* ($\beta = 0.028$, $p = 0.178$) was significantly associated with insomnia symptoms. In contrast, the interaction effects between insomnia and loneliness in predicting depressive symptoms were significant (*threat*: $\beta = 0.098$, $p < 0.001$; *loss*: $\beta = 0.097$, $p < 0.001$). Conditional indirect effects were not statistically significant at either level of loneliness. Specifically, for *threat*, the indirect effect on depressive symptoms via insomnia was nonsignificant at both high ($\beta = 0.004$, 95% BCI [−0.0218, 0.0517]) and low ($\beta = 0.002$, 95% BCI [−0.0072, 0.0286]) levels of loneliness. Similarly, *loss* did not show a significant indirect effect at either high ($\beta = 0.006$, 95% BCI [−0.0115, 0.0510]) or low ($\beta = 0.003$, 95% BCI [−0.0027, 0.0248]) levels of loneliness.

4 | Discussion

Childhood adversity is a key modifiable risk factor for psychopathology including depressive disorders and suicide attempts (Zatti et al. 2017). Only recently, however, research has employed longitudinal designs and sophisticated statistical tools to identify psychological and physiological mechanisms linking traumatic experiences in childhood to adult psychopathology (Conway et al. 2020; Iob et al. 2020a; Liu et al. 2023; Zagaria et al. 2024). In this context, this study found that the association between retrospective ACEs and late life depressive symptoms was mediated by insomnia symptoms experienced at an intermediate time-point, and that feelings of loneliness increased the magnitude of this mediation. Specific ACEs associated with depressive symptoms through the mediation of insomnia were parental bonding and household dysfunction (i.e., parental arguments, parental mental illness or substance abuse, and parental separation or divorce). To the best of our knowledge, there is no previous data in older adults testing the associations between ACEs subtypes, insomnia and depression, strengthening the unique contribution of the present study. To further contextualize these findings, it is important to consider the distinct yet overlapping mechanisms through which specific categories of ACEs—particularly those involving early relational environments—can influence long-term mental health outcomes. Among these, household dysfunction, which encompasses parental mental illness, substance abuse, and family conflict (Felitti et al. 1998), has been associated with chronic exposure to stress and impaired emotional regulation. These dysregulations are known to contribute to sleep disturbances and increase susceptibility to depression in adulthood (Chapman et al. 2011; Kessler et al. 2010). Similarly, deficits in parental bonding, often conceptualized as impairments in early attachment and caregiving responsiveness, may hinder the development of neurobiological systems involved in stress regulation and sleep architecture. Insecure or disrupted parental bonding has been linked to both insomnia and affective disorders later in life (Kidd et al. 2022; Rojo-Wissar et al. 2020). This body of evidence supports the notion that socio-relational forms of childhood adversity may exert a particularly enduring impact, shaping mental health trajectories through mechanisms rooted in early emotional development. Our findings converge with findings from previous research (Li, Wang et al. 2023), who identified insomnia as an underlying mechanism between childhood maltreatment and suicidal ideation (i.e., a clinically salient symptom of depression; American Psychiatric Association 2013) in adolescents, supporting the broader hypothesis that sleep disturbances may represent a core mechanism linking early-life adversity to depression across the lifespan. Previous authors have suggested that the onset of depressive symptoms in older adults may imply a traumatic reactivation, arguing that the pathway that mediates this process is still unclear (Comijs et al. 2013). The present results are relevant insofar as they suggest insomnia as a possible mechanism explaining how the effects of ACEs during the first decades of life can extend to mental health problems later in life. This result shed new light on the literature on life course (Elder 1998) which posits that adverse life events are interconnected, with experiences in the early stages of life influencing the occurrence of later psychopathology (Yin et al. 2023). The effect sizes observed in this study ranged from small ($\beta = 0.011$) to moderate ($\beta = 0.254$) across the tested pathways, which is consistent with longitudinal research on psychological determinants of mental health in older adults (Davies et al. 2021; Lin and Chiao 2020; Prather 2019). Small effects emerged for the associations between ACE, insomnia and depressive symptoms, in line with the distal nature and cumulative influence of childhood exposures over the lifespan on these disturbances (Chau et al. 2024; Sheffler et al. 2023). Conversely, moderate effect

sizes were found for proximal relationships, such as those between insomnia and depressive symptoms in the context of high loneliness, confirming that psychosocial conditions exert a strong impact on mental health outcomes in late life (McClelland et al. 2020). These patterns are consistent with life course epidemiological theories of health, where even modest associations, especially when persistent, can have meaningful implications for long-term psychological well-being (McLaughlin and Sheridan 2016).

The association between loneliness and health is a topic of great contemporary scientific relevance and social concern. Experiencing loneliness was estimated as impacting on mortality as commonly recognized health behavior such as smoking (Baarck and Kovacic 2022). Loneliness has been associated with a range of poor sleep indicators in older age, such as worse subjective sleep quality, shorter sleep duration (Benson et al. 2021), and insomnia severity (Qi et al. 2023). A recent meta-analysis (Deng et al. 2023) reported that older adults who were lonely were significantly more likely to suffer from low sleep quality than those without loneliness (OR = 1.750), and longitudinal evidence suggested the existence of a bidirectional association between loneliness and sleep in older age (Griffin et al. 2020). To the best of our knowledge, no study to date tested the combined effect of ACEs-associated insomnia and loneliness on depressive symptoms.

Several paths may link loneliness to depressive symptoms in older adults such as accelerated cognitive decline (Donovan et al. 2016), increase in ruminative thinking (Zawadzki et al. 2013), and negative self schema (Ypsilanti et al. 2019). Further neurobiological processes linking loneliness to depression may include morphological (e.g., volume reduction) or functional changes of brain areas relevant for cognitive-affective regulation such as the prefrontal cortex (Rosenbaum et al. 2024), the amygdala and the hippocampus (Düzel et al. 2019) and altered default and attention brain network features (Li, Huang et al. 2023). Downregulation of neurotrophic factors (Dabiri et al. 2024) and neuroimmune pathways (Smith et al. 2020) may also play a role. In ELSA, the onset of loneliness has also been associated with increase in concentrations of inflammatory markers (Vingeliene et al. 2019), and lonely older adults displayed significant greater concentrations of inflammatory markers in response to stress (Steptoe et al. 2004), which may be associated with the onset of physical symptoms of depression (Frank et al. 2021; Ballesio 2023). Some of these factors may also explain part of the association between insomnia and depression. For instance, insomnia is associated with poor cognitive function (Altena et al. 2010; Ballesio et al. 2020), more frequent use of maladaptive emotion regulation strategies (Cerolini 2015; Meneo et al. 2023), lower cortical top-down regulation of the amygdala (Kweon et al. 2023), lower concentration of neurotrophic factors (e.g., Ballesio et al. 2023), and upregulation of inflammatory biomarkers (e.g., Zhang et al. 2023), which have all been associated with the onset of depression in elderly in ELSA (Ballesio, Zagaria, Ottaviani et al. 2022) and other samples (Li et al. 2015; Sroykham and Wongsawat 2019).

Contrary to our hypothesis, the association between ACEs and insomnia symptoms in our sample was independent of the moderation of hair cortisol. Perceived stressful experiences are considered precipitating factors of insomnia (e.g., Perlis et al. 2011; Ballesio, Vacca et al. 2022b). Prenatal stress and traumatic childhood experiences may interact with genome to trigger epigenetic modification in the regulation of stress and arousal systems (e.g., affecting the expression of glucocorticoid receptors in the brain) ultimately sensitizing the reactivity of HPA axis (Lo Lo Martire et al. 2020). Consistently, we hypothesized that the impact of ACEs on insomnia may vary at different levels of vulnerability in the HPA axis activation. Only a few studies investigated HPA axis dynamics and ACEs in older adults. Within the Longitudinal Aging Study Amsterdam, Gerritsen et al. (2010) found a hyposecretion of morning cortisol and flattened diurnal cortisol variability in older adults with a history of childhood trauma. In the Netherlands Study of Depression in Older Persons, Wielaard et al. (2017) showed a higher cortisol reactivity during the hour after awakening only in nondepressed participants, which authors interpreted as a depression-driven modification of the effect of childhood abuse on the HPA axis. Another possibility is that the activation of the HPA axis may be a mediator, rather than a moderator, of the association between ACEs and insomnia (Simon and Admon 2023). Hyperarousal via dysregulation of HPA axis is a putative pathophysiological marker of insomnia (e.g., Dressle et al. 2022), and exposure to 3+ ACEs was related to a steeper increase in hair cortisol with age (Iob et al. 2020b). Exposure to repeated stress activates the hypothalamus-pituitary-adrenal (HPA) axis (Pruessner et al. 2010) and leads to the chronic production of glucocorticoids, possibly resulting in insomnia (Wang et al. 2019). Increased cortisol is also frequent in individuals with depressive symptoms, particularly in response to psychological stress (Burke et al. 2005). Longitudinal data also suggests that cortisol may predict depressive symptoms (Hsiao et al. 2013), although negative findings were also reported (e.g., Carnegie et al. 2014). The ELSA protocol precluded a multiple assessment of cortisol reactivity and sleep variables to empirically test the mediation effect of hair cortisol. Future studies are needed to corroborate the mediation hypothesis of HPA dysregulation between ACEs and insomnia. The effects of ACEs on insomnia may be also serially mediated by recent stressful life events and perceived stress (Benham et al. 2025) and anxiety symptoms (Ashour et al. 2024). Furthermore, it has been hypothesized that ACEs may influence the expression of chronotype (i.e., time of day preference for wake-up, activity and sleep) towards a circadian phase delay (McCarthy et al. 2023). Consistently, insecure attachment styles were associated with evening chronotype (Işik and Kirli 2022), which may predispose individuals to insomnia and depression (Kivelä et al. 2018). To the best of our knowledge, however, the influence of ACEs on chronotype trajectories during late-life is yet to be investigated.

Finally, the current results should be interpreted in light of the bidirectional associations existing between insomnia, loneliness, and depression. For instance, in a meta-analysis of longitudinal studies in older adults, Bao et al. (2017) found significant bidirectional associations between insomnia and depressive symptoms. Indeed, a longitudinal study with multiple assessments found that depressive symptoms were predictive of insomnia, but the latter was not predictive of depressive symptoms (Zhou et al. 2024). Additionally, while lonely individuals may be prone to develop insomnia (e.g., Hom et al. 2020), it is also possible that trouble sleeping may limit social interactions and increase loneliness (Gunn et al. 2014).

Furthermore, while loneliness is a strong predictor of depression in older adults (Ward et al. 2023), symptoms of anhedonia may limit interpersonal interactions and increase loneliness and insomnia in a vicious cycle. Alternative paths should be explored in future studies to detect the stronger targets for clinical interventions.

5 | Limitations

This study has several strengths, including a large sample size and the use of a three-wave longitudinal mediation model. However, there are also several limitations to acknowledge. Although we controlled for depressive symptoms at wave 4, this does not fully account for changes in depression that may have occurred between waves 4 and 6. As such, the observed association between insomnia at wave 6 and depressive symptoms at wave 7 may partially reflect unmeasured changes in depression during that interim period. This limitation should be considered when interpreting the mediation findings. Insomnia symptoms were assessed using the Jenkins Sleep Scale (Jenkins et al. 1988), which captures only the most common symptoms of insomnia in older adults, such as difficulties falling asleep and maintaining sleep, but does not consider daytime consequences except for feeling tired at awakening. Daytime impairment due to insomnia is included in other validated and widely used measures, such as the Insomnia Severity Index (ISI; Bastien 2001). Additionally, this study did not include measures of chronotype. Therefore, future studies would benefit from including more comprehensive measures of insomnia symptoms and incorporating chronotype measures to further enhance the understanding of these relationships. Related to this, ELSA did not include objective sleep measures, which prevented us from exploring the relationship between specific physiological sleep parameters, ACEs, depressive symptoms, and activation of the HPA axis. Future research should incorporate objective sleep measures such as polysomnography or actigraphy. The inclusion of objective measures of sleep may provide objective data on specific sleep characteristics that could not be assessed with self-reported questionnaires such as sleep onset latency (SOL) or wake after sleep onset (WASO). This might contribute to a more comprehensive understanding of the phenomenon. Another limitation is the lack of distinction between loneliness and social isolation. While loneliness is a subjective state, social isolation is the objective condition of having few social relationships or infrequent social contact (House et al. 1988). Although interrelated, these constructs reflect different concepts (Routasalo et al. 2006), and individuals might feel lonely without being socially isolated. Thus, future studies should integrate both loneliness and social isolation measures to better understand the unique contributions of these constructs. Also, this study examined depressive symptoms in a sample of healthy older adults. Future studies should explore these relationships in individuals with clinical depression for a more comprehensive understanding. Furthermore, understanding the history of emotional and social-cognitive development in future studies (i.e., through the implementation of cross-lagged models) would also enrich the knowledge in the field, pointing out a potential causal link between ACEs, insomnia and depressive symptoms. Finally, although attrition analyses indicated small or negligible differences between participants with and without missing data, relying on complete case analysis may still introduce potential biases and should be considered when interpreting the findings.

6 | Conclusions

The risk of insomnia and depressive symptoms for individuals with a history of childhood adversity seems to persist throughout the lifespan. Our analysis suggests that insomnia may act as a mediator between ACEs and depressive symptoms in older adults. Importantly, the impact of ACEs-related insomnia on depression was stronger in elderly with high feelings of loneliness, suggesting a cumulative effect of these conditions on mental health. It is important that older adults with a history of ACEs be screened for both insomnia and loneliness, and referred for evidence-based treatments accordingly. Such preventive interventions may limit the long-term impact of ACEs on depressive symptoms. Regardless, it is important to note that insomnia, depression, and loneliness may be bidirectionally related. Also, insomnia may mediate the association between loneliness and depression in older age (Gyasi et al. 2022). Therefore, testing of opposite paths seems also plausible.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are openly available in UK Data Service at https://beta.ukdataservice.ac.uk/datacatalogue/studies/study?id=5050, reference number 10.5255/UKDA-SN-5050-31. Data is deposited at http://ukdataservice.ac.uk/.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.
Supplementary Document.