Received: 1 March 2023 | Revised: 19 June 2023 | Accepted: 3 July 2023

DOI: 10.1002/smi.3290

WILEY

RESEARCH ARTICLE

Life stress and adiposity in mothers: A 14-year follow-up in the general population

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Funding information
ZonMw; Ministerie van Volksgezondheid, Welzijn en Sport; Nederlandse Organisatie voor Wetenschappelijk Onderzoek

Abstract

Exposure to specific stressors has been found to associate with higher adiposity in adulthood. However, the potential overlapping effects of stress domains have been overlooked, as well as the role of parenting-related stressors that mothers are widely exposed to in mid-adulthood. Therefore, we assessed the association of overlapping effects of stress domains, including parenting-related stress, with subsequent adiposity in mothers. In 3957 mothers from the population-based Generation *R* Study, life stress was assessed during the first 10 years of child-rearing and measured as a reflective latent variable of stress domains. Structural equation modelling was used to assess the association of life stress and its individual domains with body mass index (BMI) and waist circumference after 14 years of follow-up. Greater life stress over the course of 10 years was associated with a higher BMI (standardized adjusted difference: 0.57 kg/m^2 [95% CI: 0.41–0.72]) and a larger waist circumference (1.15 cm [0.72–1.57]). When examining individual stress domains, we found that life events was independently associated with a higher BMI (0.16 kg/m^2) and contextual stress was independently associated with a higher BMI (0.43 kg/m^2) and larger waist circumference (1.04 cm). Parenting stress and interpersonal stress were not independently associated with adiposity at follow-up. The overlap of multiple domains of stress in mothers is associated with a higher risk of adiposity. This effect was stronger than for individual life stress domains, reiterating the need to consider overlapping effects of different life stress domains.

KEYWORDS
adiposity, BMI, life stress, parenting stress, waist circumference

1 | INTRODUCTION

An increasing body of evidence suggests that prolonged exposure to life stress is a risk factor for adiposity (Block et al., 2009; Cuevas et al., 2019; Hamer & Stamatakis, 2008; Moore & Cunningham, 2012; Wardle et al., 2011). Life stress can be defined as exposure to life stressors, which are any sudden events, chronic demands, or traumas that require an individual to extensively readjust their life (Thoits, 2010). They can be everyday stressors, such as work stress or lack of social support, but also more acute events such as death of a loved one or losing a job. Wardle et al. (2011) showed in a meta-analysis of 14 longitudinal cohort studies that individual psychosocial stressors indeed might be risk factors for adiposity. These stressors often co-occur or lead to each other, and are thought to have overlapping effects (Evans et al., 2013; Thoits, 2010; Wardle et al., 2011). Therefore, a common approach used to model stress effects has been to sum individual stressors into cumulative scores. Studies using this approach have shown that experiencing a greater number of stressors linearly associates with higher body mass index (BMI) at follow-up (Cuevas et al., 2019; Harding et al., 2014; Mehlig et al., 2020; Thoits, 2010). Yet, this purely cumulative approach neglects that the effects of stressors might overlap and that potentially, this shared variance is what drives the association with adiposity (Thoits, 2010). Previous work has successfully used a shared-variance approach to model life stress exposure in children (Cecil et al., 2014; Rijlaarsdam et al., 2016), but such a model has not yet been applied to examine life stress in adulthood and its prospective association with adiposity.

Research into individual domains of life stress has shown that experiencing contextual stressors, such as financial strain, is associated with a higher risk of becoming overweight or developing obesity (Cuevas et al., 2019; Siahpush et al., 2014). Similar associations have been demonstrated for life events, such as adverse events during childhood and death of a loved one, and interpersonal problems, such as divorce and negative relationships (Bentley & Widom, 2009; Hughes et al., 2017; Kershaw et al., 2014) Another important domain of prolonged stress during mid-adulthood are the specific demands associated with child-rearing (Hayes & Watson, 2013; Holly et al., 2019; Kim et al., 2007; Raphael et al., 2010). Its effects on adiposity are unknown: evidence is limited to a small study with short follow-up that found that parenting stress was associated with higher total adiposity (BMI) and central adiposity (waist circumference) (Hruska et al., 2020). Moreover, these studies have investigated these domains of life stress separately. It is yet unclear whether they independently associate with adiposity.

In this population-based study, our main objective is to assess the association of life stress during the first 10 years of child-rearing with subsequent adiposity, that is, BMI and waist circumference, after an average of 14 years of follow-up in a cohort of mothers. To this end, we constructed a latent factor measuring the shared variance of four cumulative stress domains: life events, contextual stress, parenting stress and interpersonal stress. We expected more life stress to be associated with more adiposity after follow-up. Additionally, we assessed the associations between cumulative stress domains and adiposity individually and adjusted for other domains. Based on previous literature, we expected all cumulative stress domains to be positively associated with adiposity.

2 | METHODS

2.1 | Study design and participants

The present study is embedded in the Generation *R* Study, a population-based prospective cohort study from foetal life onwards (Kooijman et al., 2016). Pregnant women within Rotterdam, the Netherlands, with a delivery date between April 2002 and January 2006, were invited to participate. In total, 9778 mothers were enroled in the study and followed up from pregnancy onwards. Parents and their children are invited to fill out questionnaires and participate in research visits every 2–4 years (Kooijman et al., 2016).

In total, 4262 mothers participated in a research visit approximately 14 years after intake. Of those, 180 participants were excluded because their life stress score could not be computed due to high missing value frequencies (more than 50% of stressors missing). Additionally, 229 mothers were excluded as they had no data available on BMI or waist circumference, resulting in a final sample size of 3957 persons.

General design, research aims, and specific measurements of the Generation *R* study have been approved by the Medical Ethical Committee of the Erasmus Medical Centre, in Rotterdam, the Netherlands, in accordance with the Declaration of Helsinki of the World Medical Association. Written informed consent was obtained from all participants.

2.2 | Measurements

2.2.1 | Life stress

The life stress score was developed in line with previous work (Cecil et al., 2014; Rijlaarsdam et al., 2016; Schuurmans et al., 2022). Life stress was defined as a reflective latent factor of four cumulative stress domains: life events (e.g. death of a family member, maltreatment during childhood), contextual stress (e.g. financial difficulties), interpersonal stress (e.g. marital status) and parenting stress (e.g. child wellbeing, family functioning). These cumulative stress domain scores were calculated by adding up the scores of the single stressors (coded yes/no). Stressors were included if they fit the following definition: any sudden event, chronic demand or traumas that requires an individual to extensively readjust their life (Thoits, 2010). Stressors were mainly derived from questionnaires that were completed at several time points from pregnancy (trimester 2 and/or 3) to 10 years after the birth of their child. Additionally, a structured interview was performed after 9 years to

obtain information on previous pregnancies and life events. Detailed information on the stressors can be found in Table S1. Model fit and factor loadings of the latent variable are shown in Figure S1, and model fit was found to be good following the criteria of Iacobucci (2010).

2.2.2 | Adiposity

At baseline, height and weight were measured without shoes or heavy clothing during a research centre visit when women were on average 14.9 weeks pregnant, and were used to calculate baseline BMI (kg/m^2). If women were included in mid- or late pregnancy (gestational age ≥18 weeks) baseline BMI was considered missing. Body mass index measured at enrolment in early pregnancy agreed strongly with self-reported pre-pregnancy BMI (two-way mixed effects intraclass correlation: 0.95).

At follow-up, weight, height and waist circumference were measured without shoes or heavy clothing at the research centre. Waist circumference and weight were measured after an average of 14.1 years of follow-up. Height was measured after 9 years of follow-up, but was assumed to be constant over time. For use in additional analyses, BMI was categorised as underweight (<18.5 kg/m^2), normal weight (18.5–24.9 kg/m^2), overweight (25–29.9 kg/m^2), and obesity (≥30 kg/m^2) (World Health Organization, 2000).

2.2.3 | Other variables

Age, parity, and country of origin of the participants were obtained by self-report at intake during pregnancy. Country of origin was categorised as either European or non-European (Jaddoe et al., 2008). Follow-up time was calculated as the difference between age at intake and age at follow-up. Gestational age at enrolment was determined by foetal ultrasound examination upon enrolment (Kooijman et al., 2016). Physical activity, diet, smoking, alcohol use, depressive symptoms and anxiety were self-reported in questionnaires after approximately 10 years of follow-up (Kooijman et al., 2016). Smoking was categorised as never, former, or current. Diet was measured with the cardiovascular healthy diet score (Lloyd-Jones et al., 2010). Scores ranged from 0 to 5, with higher scores indicating a more unhealthy diet. Physical activity was categorised as never, less than 2.5 h or more than 2.5 h of vigorous and moderate activity per week, following the recommendation of the World Health Organization to undertake at least 150 min of moderate-intensity physical activity per week (World Health Organization, 2020) Alcohol use in the past 3 months was measured on a 6-points Likert scale, with higher scores indicating more alcohol intake. Depressive symptoms and anxiety were measured using the depression and anxiety subscales of the Brief Symptom Inventory (Derogatis & Melisaratos, 1983), with higher scores indicating poorer mental health.

2.3 | Statistical analyses

Median frequency of missing data was 6.8% for stressors (*IQR* = 1.8%–16.4%) and 16.1% for covariates (*IQR* = 5.8%–17.2%). In order to reduce potential bias associated with these missing data, missing values were imputed using the multiple chained equation algorithm in the *mice* package (version 3.13.0 (van Buuren & Groothuis-Oudshoorn, 2011)). Stressors were imputed using the outcomes, covariates, auxiliary variables, other stressors within the same cumulative stress domain, and other cumulative stress domain scores. The following stressors were used as auxiliary variables: immigration (life events domain), education (contextual stress domain), and divorce and family distress (interpersonal stress domain), based on criteria by van Buuren (van Buuren & Groothuis-Oudshoorn, 2011). Missing values in auxiliary variables and covariates were imputed based on other auxiliary variables, covariates and the cumulative stress domains. Cumulative stress domain scores were calculated using passive imputation. We created 30 imputed datasets using 60 iterations.

We assessed the association of the reflective latent variable of life stress with adiposity using path analysis in *Lavaan* (version 0.6–6 (Rosseel, 2012)). All analyses were run separately with BMI and waist circumference as continuous outcomes. Two sets of covariates were used to adjust the associations. Model 1 included potential confounders: age of mother, parity, gestational age at intake, duration of follow-up, and BMI at baseline. Model 2 was additionally adjusted for behavioural and psychological factors that have been hypothesised to be on the causal pathway between life stress and adiposity and were assessed after 10 years of follow-up (Tomiyama, 2019; Wardle et al., 2011). Factors were physical activity, diet, smoking, alcohol use, depressive symptoms and anxiety. Average mean differences, 95% confidence intervals (95% CI) and *p* values for the association between life stress and the adiposity outcome were calculated for each model.

For our second aim, to investigate the link between individual cumulative stress domains and adiposity, we followed an approach previously described by Cecil et al. (2017). In short, we standardized cumulative stress domain scores and performed two sets of multivariate linear models. First, the cumulative stress domains were entered as separate predictors of BMI and waist circumference in individual models. Second, all cumulative stress domains were entered simultaneously as predictors of BMI and waist circumference in one model, meaning effects were mutually adjusted. Both sets of models were adjusted for all covariates.

Three additional analyses were performed to assess the robustness of the main association between life stress and adiposity. First, we assessed the association between life stress and adiposity stratified for baseline BMI category, as previous studies have suggested that the associations between life stress and adiposity depends on baseline adiposity status (Block et al., 2009; Harding et al., 2014; Kivimäki et al., 2006). Second, analyses were stratified for country of origin. Third, we performed a multinomial regression with life stress as the exposure, and categorised BMI at follow-up as the outcome (reference: BMI 18.5–25).

All analyses were performed in *R* (version 3.6.3 (Team, 2020)). As we have 30 imputed datasets, estimates were pooled using Rubin's rules (Rubin, 1987), using the *semTools* package (version 0.5–3 (Jorgensen et al., 2020)). The p-values of the two outcomes (i.e., BMI and weight circumference) were corrected using false-discovery rate correction following the Benjamini-Hochberg method (Benjamini & Hochberg, 1995).

3 | RESULTS

3.1 | Sample characteristics

Population characteristics can be found in Table 1. The mothers in this sample had a mean age of 31.2 (*SD* = 4.9) at baseline. The mean BMI was 24.6 kg/m^2 (*SD* = 4.3) at baseline, and 26.3 kg/m^2 (*SD* = 5.0) after an average of 14.1 (*SD* = 0.53) years of follow-up. Average waist circumference at follow-up was 83.6 cm (*SD* = 11.2). The four cumulative stress domains were all positively correlated (Table S2).

3.2 | Life stress and adiposity

The associations of the latent life stress factor with BMI and waist circumference, estimated with path analysis, are shown in Table 2. Mothers with one *SD* more life stress had a 0.57 kg/m^2 (95% CI: 0.41–0.72) higher BMI than mothers with average life stress. Similarly, one SD more life stress was associated with 1.15 cm (0.72–1.57) more waist circumference at follow-up. Effect sizes were comparable when additionally adjusting for the covariates in model 2. Standardized effect estimates for the association of life stress with BMI and waist circumference (Table S3) were comparable (β = 0.11 vs. β = 0.10).

3.3 | Cumulative stress domains and adiposity

Next, we investigated the role of the specific cumulative stress domains as predictors of BMI and waist circumference at follow-up (Table 3). Life events, contextual stress and interpersonal stress were positively associated with both BMI (adjusted mean differences range 0.26–0.50) and waist circumference (adjusted mean differences range 0.40–1.09), when entered in separate models while adjusting for all covariates. Parenting stress was not associated with BMI and waist circumference. When all four cumulative stress domains were entered simultaneously as predictors, the positive associations of life events with BMI (adjusted mean difference = 0.16, [0.04–0.28]) and contextual stress with BMI (adjusted mean difference = 0.43, [0.29–0.56]) remained. Only contextual stress was independently associated with larger waist circumference (adjusted mean difference = 1.04, [0.67–1.40]).

TABLE 1 Characteristics of the study population (*n* = 3957).

Variable	Mean ± SD or *N* (%)
Age at baseline, years	31.2 ± 4.9
Parity, number, median (IQR)	0 (0–1)
Country of origin	
European	2643 (66.8)
Non-European	1276 (32.2)
Gestational age at intake, weeks, median (IQR)	13.8 (12.4–16.5)
Duration of follow-up, years	14.1 ± 0.5
Physical activity	
Never	896 (22.6)
Less than 2.5 h per week	1241 (31.4)
More than 2.5 h per week	993 (25.1)
Diet score, median (IQR)	3 (2–4)
Smoking	
Never	1804 (45.76
Former	1058 (26.7)
Current	459 (11.6)
Alcohol consumption in the past 3 months	
Never	747 (19.4)
Less than 1 glass per week	807 (20.9)
1–3 glasses per week	697 (18.1)
4–6 glasses per week	515 (19.4)
1 glass per day	262 (20.9)
2 or more glasses per day	180 (18.1)
Depressive symptoms, total score, median (IQR)	0 (0–0.17)
Anxiety, total score, median (IQR)	0.17 (0–0.33)
BMI at baseline, kg/m^2	24.6 ± 4.3
Underweight (<18.5 kg/m^2)	58 (1.5)
Normal (≥18.5 & <25 kg/m^2)	2225 (56.2)
Overweight (≥25 & <30 kg/m^2)	935 (23.6)
Obesity (≥30 kg/m^2)	374 (9.5)
BMI at follow up, kg/m^2 [a]	26.3 ± 5.0
Underweight (<18.5 kg/m^2)	24 (0.5)
Normal (≥18.5 & <25 kg/m^2)	1851 (46.8)
Overweight (≥25 & <30 kg/m^2)	1224 (30.9)
Obesity (≥30 kg/m^2)	811 (20.5)
Waist circumference, cm [b]	83.6 ± 11.2

Note: Data are presented as mean(*SD*) or N(%) unless otherwise indicated, and shown for non-imputed data. % missing data in covariates: median(*IQR*) = 16.1%(5.9%–17.2%).

[a] *n* = 3910.

[b] *n* = 3927.

TABLE 2 The association between life stress and body mass index (BMI) and waist circumference.

	BMI (n = 3910)			Waist circumference (n = 3927)		
Life stress	**Adj. MD**	**95% CI**	***p***	**Adj. MD**	**95% CI**	***p***
Model 1	0.58	0.45; 0.72	<0.001	1.25	0.89; 1.62	<0.001
Model 2	0.57	0.41; 0.72	<0.001	1.15	0.72; 1.57	<0.001

Note: Adj. MD = Adjusted mean difference in the outcome for 1 standard deviation increase in life stress. Life stress was measured as a latent factor of four cumulative stress domains, with higher scores indicating more life stress. Model 1 was adjusted for age, gestational age at intake, duration of follow-up, parity and BMI at baseline; model 2 was additionally adjusted for physical activity, diet, smoking, alcohol use, depressive symptoms and anxiety. *p*-values were corrected for the double outcome using false discovery rate correction.

TABLE 3 Associations between cumulative stress domains and adiposity.

	BMI (n = 3910)						Waist circumference (n = 3927)					
	Separate models			Simultaneous model			Separate models			Simultaneous model		
Stress domain	**Adj. MD**	**95% CI**	***p***	**Adj. MD**	**95% CI**	***p***	**Adj. MD**	**95% CI**	***p***	**Adj. MD**	**95% CI**	***p***
Life events	**0.29**	0.18; 0.40	<0.001	**0.16**	0.04; 0.28	0.022	**0.49**	0.18; 0.80	0.002	0.19	−0.14; 0.52	0.26
Contextual stress	**0.50**	0.38; 0.62	<0.001	**0.43**	0.29; 0.56	<0.001	**1.09**	0.77; 1.41	<0.001	**1.04**	0.67; 1.40	<0.001
Interpersonal stress	**0.26**	0.14; 0.38	<0.001	0.07	−0.06; 0.20	0.65	**0.40**	0.09; 0.71	0.01	−0.07	−0.41; 0.28	0.71
Parenting stress	0.11	−0.01; 0.24	0.07	−0.05	−0.18; 0.08	0.76	0.35	0.02; 0.67	0.07	0.05	−0.30; 0.40	0.76

Note: Standardized cumulative domain sum scores were first entered as predictors of BMI in separate models and then simultaneously. Adj. MD = adjusted mean difference in the outcome for 1 standard deviation increase in the cumulative stress domain. Associations were adjusted for age, gestational age at intake, duration of follow-up, parity, BMI at baseline, physical activity, diet, smoking, alcohol use, depressive symptoms and anxiety. *p*-values were corrected for the double outcome using false discovery rate correction. Significant effect estimates are marked in bold.

3.4 | Additional analyses

In the analysis stratified for baseline BMI (Table S4), we found that in those who had underweight at baseline, one *SD* more life stress was associated with a 1.19 kg/m^2 ([−0.52–2.90]) higher BMI and a 3.71 cm ([−0.58–8.00]) larger waist circumference at follow-up, but these effects were not significant. One *SD* more life stress was associated with a 0.51 kg/m^2 (0.35–0.67) higher BMI in those who had normal weight and 0.66 kg/m^2 higher (0.34; 0.97) in those who were overweight. Similarly, life stress was positively associated with a 1.03 cm (0.50–1.31) larger waist circumference in those with normal weight and 1.19 cm (0.15–1.52) larger in those with overweight. No association of life stress with BMI or waist circumference was observed in those having obesity at baseline.

When the association was estimated for participants with European and with non-European country of origin separately, effect sizes were smaller in both strata compared to our main analysis (Table S5) but still significant. Multinomial regression analysis with BMI as a dependent categorical variable showed that an increase in life stress was not significantly associated with the odds of being underweight (odds ratio (*OR*) = 1.17, [0.69–1.99]). It was associated with higher odds of being overweight (*OR* = 1.34, [1.12–1.49]) and having obesity (*OR* = 1.63, [1.41–1.89]) after 14 years with normal weight as the reference category (Figure 1).

4 | DISCUSSION

In this population-based study of mothers, we demonstrated that more life stress, measured as the shared variance of contextual stress, life events, interpersonal stress and parenting stress, is associated with more adiposity after an average of 14 years of follow-up in mothers, after adjusting for baseline BMI. When assessing the independent effects of the cumulative stress domains, we found evidence of an association of contextual stress and life events with adiposity. We did not find evidence of an association of interpersonal stress and parenting stress with adiposity.

The observed positive association between life stress and adiposity is consistent with multiple previous studies that have shown a positive association of a sum score of multiple psychosocial stressors with adiposity (Cuevas et al., 2019; Harding et al., 2014; Isasi et al., 2015; Mehlig et al., 2020). In line with a previous meta-analysis, we observe that the effect of life stress on central adiposity (i.e., waist circumference) is comparable with that on total adiposity (i.e., BMI), implying that life stress is associated with a higher risk of weight gain in general, but also specifically with the accumulation of fat mass in the abdominal region (Wardle et al., 2011). Our study extends previous findings by applying latent modelling to capture life stress. Compared to previous literature, we used a wide range of stressors that were repeatedly assessed over a 10-year period, including stressors related to parenting (Harding

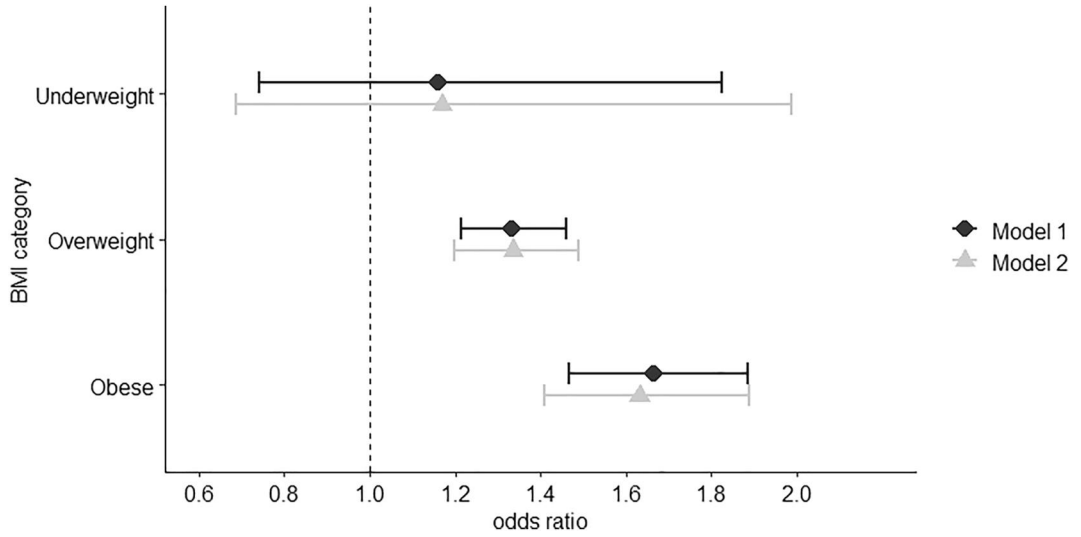

FIGURE 1 Associations of life stress with odds of being underweight, overweight and having obesity. Odds ratios and 95%CI are displayed for 1 SD increase in life stress. Life stress was measured as a latent factor of four cumulative stress domains, with higher scores indicating more life stress. Normal weight was considered as the reference category. Model 1 was adjusted for age, gestational age at intake, parity and body mass index (BMI) at baseline; Model 2 was additionally adjusted for physical activity, diet, smoking, alcohol use, depressive symptoms and anxiety.

et al., 2014). We observe that the association between the shared variance of life stress and adiposity is stronger than that of specific cumulative domains, suggesting that experiencing multiple stressors across different domains is more strongly associated with adiposity, rather than experiencing stress within one particular domain. Our results underscore the importance of shifting focus from individual domains of stress to the overlapping effects of co-occurring life stress domains in research. Moreover, when designing public health interventions overlap between co-occurring stressors should be considered.

When studying the role of individual cumulative stress domains, we did not find evidence for an association between parenting stress and adiposity, even though parenting is an important source of stress during mid-adulthood. This contrasts previous work (Hruska et al., 2020) and suggests that the effect of parenting stress on physical health in the general population might be different from other cumulative stress domains. However, parenting stress did load on to the latent life stress factor similarly to the domain of life events, indicating that even though it is not individually associated with the outcome, parenting stress does contribute to life stress. Furthermore, the significant individual effects of interpersonal stress and life events do not maintain when assessed in the presence of other cumulative stress domains, indicating that previously found effects (Harding et al., 2014; Kershaw et al., 2014; Mehlig et al., 2020) might be partly explained by their overlap with contextual stress. Our observation that contextual stress is most consistently associated with adiposity is in line with Cuevas et al. (2019), who showed that of eight different psychosocial stressors only financial strain was independently associated with higher odds of obesity. A potential explanation is that financial stressors have a more chronic nature and affect health behaviours more directly than other stressors by limiting access to healthy food or leisure activities (Beenackers et al., 2017; Mehlig et al., 2020). However, it should be noted that this relation was observed even when adjusting for multiple lifestyle factors, indicating that other mechanisms might also play a role. As contextual stressors are likely to accumulate in disadvantaged groups (Thoits, 2010) and adiposity is an important determinant of health (World Health Organization, 2000), it is possible that accumulation of contextual life stress might play a role in the persistent social inequalities of health observed in Western countries (Barnett et al., 2012). Addressing the structural inequalities that facilitate accumulation of contextual stress in this group could therefore have preventive effects on weight gain (Thoits, 2010).

Life stress may be associated with adiposity through several pathways including behavioural mechanisms, such as eating behaviour and physical activity as well as physiological mechanisms, such as HPA-axis activation and alterations in the gut microbiome. An extensive discussion of these mechanisms goes beyond the scope of this article, but we refer to Tomiyama et al. (Tomiyama, 2019) for a narrative review. Our finding that the shared variance of stressors is particularly associated with adiposity, rather than specific stressors could be explained by the allostatic load theory (Juster et al., 2016). This theory posits that cumulative exposure to psychosocial stressors can lead to progressive dysregulation of multiple physiological systems. Based on these possible mechanisms, the behavioural and psychological factors that we adjusted for in our second model are hypothesised to be on the causal pathway between life stress and adiposity. In line with previous studies (Block et al., 2009; Harding et al., 2014), we show that adjusting for these factors does not alter the magnitude of our effect sizes. This implies that their role may be smaller than expected, but a formal causal mediation analysis, using more comprehensive measures of physical activity and diet, is

warranted. Identifying potential moderating and mediating factors is of importance, as exposure to life stress can inherently not be fully prevented or avoided. Moderating and mediating factors could therefore serve as valuable targets for intervention or prevention studies (Harding et al., 2014; Isasi et al., 2015).

We also found evidence that the association between life stress and adiposity is modified by baseline BMI status, as life stress was only significantly associated with higher adiposity in those that had a normal or overweight BMI status at baseline. This is in accordance with other studies and possibly reflects a ceiling effect in those with obesity (Block et al., 2009; Harding et al., 2014; Kivimäki et al., 2006). Under the assumption that life stress is a causal risk factor for adiposity, this suggests that prevention strategies potentially are beneficial for a majority of the population, those who have not yet developed obesity. In those who are underweight at baseline, we observe that the effect size of life stress on both BMI and waist circumference was larger than in the other strata. However, interpretation of this finding is limited by the large confidence interval and small population ($n = 58$). If this finding is replicated in future studies, it may suggest that life stress affects adiposity the strongest in those who have the lowest weight, contrasting earlier work suggesting that stress is related to weight loss instead of weight gain in this group (Kivimäki et al., 2006).

In modelling life stress, we chose to take a broad approach and include stressors, such as a low education, that not only may contribute to life stress directly, but potentially also affect life stress through other pathways, for example, by diminishing perceived controllability or by increasing the likelihood of enduring other stressors (Crielaard et al., 2021). It has therefore been debated whether education should be included in cumulative stress measures. In our model, education is part of the contextual stress domain, which indeed is most strongly associated with adiposity as compared to other stress domains. However, in mutually adjusted models controlling for contextual stress, the domain of life stress was still found to independently associate with adiposity. This suggests that the association of cumulative life stress with adiposity is not merely explained by education. Other limitations of our study should be kept in mind. First, the interpretation of the difference between central and total adiposity is limited, as we were unable to adjust for baseline waist circumference. Anthropometry measures of adiposity do not measure fat mass directly, warranting more advanced measures of body to gain more precise estimates (Bridger Staatz et al., 2021). As enrolment in our study was during pregnancy, we could only measure pre-pregnancy BMI via self-report, which is prone to bias (Stommel & Schoenborn, 2009). However, baseline BMI, which was measured in early pregnancy, agreed strongly with self-reported pre-pregnancy BMI (two-way mixed effects intraclass correlation: 0.95). Furthermore, we could not examine associations in men, as we did not have sufficient data available for the fathers in our study. Previous studies have presented mixed results on potential effect modification by sex in the relationship between psychosocial stressors and adiposity (Isasi et al., 2015; Newton et al., 2017; Wardle et al., 2011; Witkam et al., 2021). Next, previous work has shown that associations might be reversed in non-Western countries (Tan & Leung, 2021), limiting external validation of our results. Lastly, from our model we cannot draw inference on the effects of individual stressors on the outcome and timing and duration of exposure to the stressors is not taken into account. Other cohorts with consistent repeated measures may be better employed to study timing effects of stressors. Of note, however, we were able to estimate prospective associations between life stress and adiposity using longitudinal data and to adjust for baseline adiposity, limiting the possibility of reverse causation.

5 | CONCLUSIONS

The present study showed that exposure to life stress in mid-adulthood, measured as the shared variance of four cumulative stress domains, is associated with higher levels of total and central adiposity in mothers after an average of 14 years of follow-up. This association was strongest for contextual stress and in those who did not have obesity at baseline. Our findings reiterate the need to consider the overlapping effects of different life stress domains when studying exposure to cumulative life stress. Identification of potential moderating and mediating factors of the effect of life stress on adiposity and targeting systems which facilitate accumulation of contextual stressors could potentially limit adiposity in mothers.

ACKNOWLEDGEMENTS

We gratefully acknowledge the contribution of general practitioners, hospitals, midwives, and pharmacies in Rotterdam. The general design of Generation *R* Study is funded by the Erasmus Medical Center, Rotterdam, ZonMw, the Netherlands Organization for Scientific Research (NWO), and the Ministry of Health, Welfare and Sport, and is conducted by the Erasmus Medical Center in close collaboration with the Faculty of Social Sciences of the Erasmus University Rotterdam, and the Stichting Trombosedienst & Artsenlaboratorium Rijnmond (STAR-MDC), Rotterdam. The work of CC is supported from the European Union's Horizon 2020 Research and Innovation Programme (EarlyCause; grant agreement No 848158). The financial supporters did not influence the results of this article. The funders had no role in the study design, data collection, analysis, interpretation of the data, or writing of the report.

CONFLICT OF INTEREST STATEMENT

The authors declared no conflict of interest.

DATA AVAILABILITY STATEMENT

Because of restrictions based on privacy regulations and informed consent of the participants, data cannot be made freely available in a public repository. However, data can be obtained upon request (datamanagementgenr@erasmusmc.nl).

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How to cite this article: Hoepel, S. J. W., Schuurmans, I. K., Cecil, C. A. M., Jaddoe, V. W. V., & Luik, A. I. (2023). Life stress and adiposity in mothers: A 14-year follow-up in the general population. *Stress and Health*, 1–9. https://doi.org/10.1002/smi.3290