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RESEARCH ARTICLE

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Associations between maternal stressful life events and child health outcomes in indigenous and non-indigenous groups in New Zealand

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ABSTRACT

Exposure to stressful life events (SLE) around the time of pregnancy is associated with adverse health outcomes for mothers and children. Previous New Zealand research found Indigenous Māori women are more likely to be exposed to SLE than non-Māori, and are exposed to a higher number of SLE. The consequences of this for ethnic inequities in child health outcomes are unknown. This paper examines the relationship between patterns of maternal SLE exposure with child health and development outcomes at age 3 years, for Indigenous and non-Indigenous children. We found most children had a stressful early life environment at least sometimes, but more than a quarter of Māori children had a mother experiencing multiple SLE on all occasions measured. We found a clear association between maternal experiences of SLE and disordered child sleep and development concerns. While not able to fully assess the contribution of maternal SLE to ethnic inequities in child health outcomes, we did clearly demonstrate that more Māori children have mothers exposed to multiple SLE, and that these maternal SLE are associated with poorer child outcomes. The impacts of chronic SLE exposure need to be better understood, especially given the large ethnic disparity in chronic SLE exposure.

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Indigenous; maternal stress; stressful life events; child health; women's health; inequities

Introduction

Exposure to stressful life events (SLE), such as job loss, death of a close family member or imprisonment, around the time of pregnancy is associated with a range of adverse health outcomes for mothers and their infants. Previous New Zealand (NZ) research (Paine et al. 2022), from the *Moe Kura* longitudinal study, found that Indigenous Māori

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women are more likely to be exposed to SLE than non-Māori women, before, during and after pregnancy, and are exposed to a higher number of SLE. Younger maternal age and greater socioeconomic deprivation explained some but not all of the inequities observed, with older age reducing the odds of experiencing SLE mostly for non-Māori women. Given the inequitable burden of SLE experienced by Māori women, this paper seeks to examine how this might impact on Indigenous child health inequities.

Exposure to SLE has been associated with higher perceived stress in individuals, although not everyone experiences the same level of perceived stress in response to SLE (van Eck et al. 1998). However, there is accumulating evidence that maternal stress during or after pregnancy can have a negative impact on child health and development. Numerous epidemiological and case–control studies have found maternal stress to be associated with an increased risk of emotional, behavioural and cognitive problems in the offspring, including anxiety and depression, ADHD, conduct disorders and autism spectrum disorders (Lautarescu et al. 2020; Van den Bergh et al. 2020). Both maternal exposure to stressful events and perceptions of distress have been associated with adverse child mental health outcomes, in distinct patterns, and sometimes additively (Rudd et al. 2022). There is also evidence that maternal stress during pregnancy is associated with: reduced telomere length suggestive of a shortened lifespan (Entringer et al. 2018); an increased risk of preterm delivery (Wadhwa et al. 2001); altered immune function (Hahn et al. 2019); an increased risk of asthma and atopic disorders (Cookson et al. 2009; Andersson et al. 2016); an altered child microbiome (Hu et al. 2019); and an altered sex ratio at birth (Walsh et al. 2019). These effects may be influenced by other factors including the type of stress, the timing of the stressful experience and the sex of the foetus (Glover and Hill 2012). Evidence also indicates that these effects can be transmitted across multiple generations through complex endocrine, socio-behavioural and epigenetic pathways (Matthews 2020). This includes evidence of the intergenerational transmission of trauma and stress for Indigenous (Lewis et al. 2021) and other racially marginalised groups (Geronimus et al. 2006), resulting in an accumulation of health disadvantage across the life-course and across generations.

Potential mechanisms for maternal stress impacting on child outcomes include the biological embedding of stress primarily during the prenatal and early post-natal periods of development, through processes such as foetal programming (Langley-Evans 2006; Wadhwa et al. 2009) and epigenetics (Bianco-Miotto et al. 2017). Research on adverse childhood experiences (ACEs) (Hughes et al. 2017), suggests that direct exposure to SLE/living conditions or maternal experiences of SLE influences parenting behaviour, maternal/child interactions and attachment. Some adverse child health outcomes may also arise as a consequence of prematurity or low birth weight, which have been associated with maternal SLE. New Zealand longitudinal research has found children exposed to adverse psychosocial experiences (including socioeconomic deprivation, maltreatment and social isolation) are at elevated risk of depression, high inflammation levels and metabolic risk markers in adulthood (Danese et al. 2009). In young children stress can be either: positive (brief and mild to moderate in magnitude), tolerable (a more serious threat but associated with at least one protective relationship) or toxic (strong, frequent, or prolonged stress without the buffering of a supportive relationship) (Shonkoff et al. 2021). When the biological disruptions caused by toxic stress persist during sensitive periods of development, they can result in enduring structural and

physiological changes that impair learning, behaviour, and both physical and mental health (Shonkoff et al. 2021).

Aspects of these frameworks for thinking about stress align with Indigenous scholarship on intergenerational and historical trauma and the transmission of harms from one generation to the other. However, any relationship between maternal SLE and child ACEs must also acknowledge how colonialism, and its associated processes of patriarchy and racism, create and maintain the conditions in which maternal SLE can thrive (Paine et al. 2022). For example, up to two-thirds of Māori women reported financial stressors, and more than 50% of Māori women reported multiple stressors at every time point assessed (Paine et al. 2022). Inequities in the distribution of stressors associated with socioeconomic deprivation are considered to be markers of the unfair structuring of power, resources and opportunities (Reid et al. 2000), and a reflection of how the ongoing presence of colonialism and structural racism in NZ operate to limit Indigenous lives and livelihoods (Reid et al. 2019). Evidence from the Growing Up in New Zealand cohort study has found a strong association between Māori women's experiences of racial discrimination and adverse maternal mental health (Bécares and Atatoa-Carr 2016), and has also found that children who experienced maternal pre- or post-natal stress are more likely to have higher body mass index (BMI) at 54-months of age (Farewell et al. 2018).

As the Indigenous people of NZ, Māori have a life expectancy of 7.5 years less (Stats NZ 2021) than non-Māori and experience higher rates of unmet health needs and disease-specific mortality rates. Māori infants are almost twice as likely to die as non-Māori non-Pacific infants and ambulatory sensitive hospitalisation (ASH) rates for Māori 0–4 year-olds are over one and a half times higher than non-Māori non-Pacific ASH rates (Simpson et al. 2017). Health equity, particularly for Māori, is an objective within key NZ health policy documents (New Zealand Public Health and Disability Act 2000 2000; Ministry of Health 2016, 2020) and NZ has endorsed the UN Declaration on the Rights of Indigenous Peoples and the UN Convention on the Rights of the Child (UN General Assembly 20 November 1989). The Treaty of Waitangi provides an additional constitutional and legal obligation for the government to ensure equity for Māori.

There is insufficient evidence on how maternal stress influences Indigenous child health inequities, including the role this may play in the accumulation of health disadvantage over the life-course and the intergenerational transmission of health inequities. Therefore, the aim of this study was to examine the relationship between various patterns of maternal SLE exposure, and child health and development outcomes at age 3 years, using a large cohort of Indigenous and non-Indigenous children.

Methods

We undertook a secondary analysis of data from 418 self-identified Māori and 768 non-Māori women from the *Moe Kura* cohort study, which was designed to investigate the associations between sleep changes across the perinatal period and maternal mood. *Moe Kura* was informed by a Kaupapa Māori scientific positioning, which is an Indigenous approach to health research that upholds Māori rights to health, including the right to participate in, and benefit from health research, and critiques the unequal power structures that contribute to Māori health inequities (Paine et al. 2020). The Kaupapa Māori

principles applied in this study were: (1) Māori leadership, participation and control at all levels of the research, including via a Māori co-Principal Investigator and involvement of Māori advisors, emerging researchers and research assistants; (2) equal explanatory and analytical power, which informed the sampling approach, recruitment, and retention strategies and collection of high- quality ethnicity data; and, (3) a commitment to undertake research that considers the structural causes of Māori/non-Māori inequities, and rejection of deficit-framed interpretations of the data (Paine et al. 2013). This secondary analysis has been conducted in line with the same positioning and kaupapa Māori methodological groundings of the *Moe Kura* study.

Methodological details of the *Moe Kura* study have been reported elsewhere (Paine et al. 2013; Signal et al. 2022) but pregnant women, recruited nationally through maternity care providers supplemented by promotion through Māori-specific media and events, were enrolled between October 2009 and November 2011, with comprehensive questionnaires about their sleep, mood and other selected measures of health and wellbeing completed in late pregnancy (T1, 35–37 weeks gestation), 12 weeks post-partum (T2) and when the child was 3–4 years old (T3). At T3, maternal and child-proxy questionnaires expanded to include a broader range of questions about sleep, mood and wellbeing. Child health outcomes included the Children's Sleep Habits Questionnaire (CSHQ) (Owens et al. 2000), Strengths and Difficulties Questionnaire (SDQ) (Muris et al. 2003), along with questions about general health, height and weight, nutrition, and whether the child had been diagnosed with specific health conditions. Questionnaires were mailed out and completed at home, with the option to complete over the phone with either a Māori or non-Māori researcher. Retention rates (T1 to T3) were 69.5% for Māori and 85.4% for non-Māori women. Participants provided written informed consent, with ethics approval granted by the Central Health and Disability Ethics Committee of NZ (CEN/09/09/070/AM02).

Key variables

Variable selection was based on prior knowledge of determinants of Māori maternal health inequities and availability at each time point. Maternal Stressful Life Events were measured using the Life Events Checklist from PRAMS (Burns et al. 2015) which asks respondents to indicate whether they had experienced any of 13 life events in the previous 12 months. For this analysis any women who reported experiencing ≥2 SLE items were considered to be exposed to 'high stress' whereas <2 SLE was 'low stress' (Signal et al. 2016). To assess the impact of patterns of maternal stress over the three time points measured, we adapted the approach taken by Farewell et al. (2018) and created six trajectories of maternal SLE:

- stably low (<2 SLE at T1, T2 and T3);
- fluctuating low (with 'low-high-low' SLE over the three time points);
- fluctuating high (with 'high-low-high' SLE);
- decreasing (≥2SLE in T1 or T1 & T2 only);
- increasing (≥2SLE only in T3, or T2 & T3) and;
- stably high (≥2SLE at T1, T2 and T3).

Child health outcomes selected for this analysis were the CSHQ score, SDQ total difficulties score and presence of a diagnosis of specific health conditions (any diagnosis of asthma, eczema or allergy). The CSHQ is a 33-item sleep-screening questionnaire completed by a parent that examines 8 sleep domains. A higher score is indicative of more disturbed sleep, and a total score higher than 41 identifies 80% of children with a clinically diagnosed sleep disorder (Owens et al. 2000). The SDQ consists of 25 questions, reflecting 5 domains of prosocial or problematic behaviours or emotions. A total 'total difficulties score' is generated by adding scores from all the domains except the prosocial scale (Emotional Symptoms, Conduct Problems, Hyperactivity, Peer Problems) and a higher total score (ranging from 0–40) denotes greater problems (Goodman et al. 1998). Diagnoses of asthma, eczema and allergy were selected because of prior evidence of inequities for Māori (Mills et al. 2012; Jones et al. 2013; Craig et al. 2014), and these variables offered the best data quality/coverage in our survey results.

Self-identified ethnicity was measured for both mother and child using the NZ population census question, with a child's ethnicity reported by the mother. *Moe Kura* uses a Māori/non-Māori analytical framework as a reflection of the relationship that exists between Māori and the British Crown under Te Tiriti o Waitangi (the founding document of New Zealand), and as a method for monitoring Crown action or inaction with respect to Māori rights to health (Reid and Robson 2007). Therefore, any person who identified as Māori, either alone or as one of multiple ethnic groups were classified as Māori, with all other participants considered non-Māori.

Other covariates included maternal age-group at T1 (16–24, 25–30 and 30–34 and 35 years and older) and socioeconomic deprivation measured at T1 using the NZDep2006 score (Salmond et al. 2007), an area-level measure of material deprivation that can be assigned to individuals based on their residential address, categorised as quintiles from Q1 = the least deprived to Q5 = the most deprived areas.

Statistical analysis

Descriptive summaries (% and 95% CI) were produced for Māori and non-Māori children separately, with differences in proportions examined using chi-square tests. Māori/non-Māori risk ratios (RR and 95% CI) were used to identify relative inequities in health outcomes. Multiple linear regression models were used to identify the association between child CSHQ and SDQ scores (separately), with maternal SLE exposure, adjusted for maternal ethnicity (Māori, non-Māori), maternal age (16–24, 25–29, 30–34, 35+ years) and maternal NZDep quintile at T1 (Q1–Q5). Least square means method was used to estimate differences between SLE groups. Analysis was performed using SAS 9.4 (SAS Institute Inc., Cary, NC, USA).

Results

Data were collected from a total of 340 Māori children and 570 non-Māori children. The demographic characteristics of the children are described in Table 1. Compared to non-Māori children in the study, Māori children were less likely to have mothers >30 years of age and were overrepresented in the more deprived socioeconomic quintiles at T1 and T3. Table 2 summarises the key health outcome measures, and patterns of maternal

Table 1. Demographic characteristics of Māori and non-Māori children in the study.

	Māori ($n = 340$) Number (%)	non-Māori ($n = 570$) Number (%)	***p*-value**
Sex			0.8664
Female	166 (48.8)	287 (50.4)	
Male	171 (50.3)	277 (48.6)	
Age			
Mean age (years)	3.1 (SD = 0.3)	3.2 (SD = 0.3)	
Deprivation score at T3 (quintile)			<0.0001
1 (least deprived)	48 (14.3)	189 (34.7)	
2	51 (15.2)	144 (26.4)	
3	63 (18.8)	102 (18.7)	
4	62 (18.5)	61 (11.2)	
5	111 (33.1)	49 (9.0)	
Maternal ethnicity			<0.0001
Māori	268 (82.0)	8 (1.4)	
non-Māori	59 (18.0)	552 (98.6)	
Maternal age group (years)			<0.0001
16–24	92 (28.1)	44 (7.9)	
25–29	76 (23.2)	115 (20.6)	
30–34	99 (30.3)	224 (40.0)	
35+	60 (18.4)	177 (31.6)	
Maternal deprivation at T1 (quintile)			<0.0001
1 (least deprived)	39 (12.0)	169 (30.3)	
2	48 (14.7)	130 (23.3)	
3	68 (20.8)	123 (22.0)	
4	81 (24.8)	75 (13.4)	
5	91 (27.8)	61 (10.9)	

Note: Missing observations were noted for the following variables: gender ($n = 9$).

Table 2. Prevalence of child health outcomes and maternal SLE exposure, at age 3 years (T3), by ethnicity.

	Māori ($n = 340$) % (95% CI)	Non-Māori ($n = 570$) % (95% CI)
Medical condition		
Any of: asthma/eczema/allergy	36.76 (31.61, 41.92)	27.72 (24.03,31.41)
Strengths and Difficulties total score (mean [SD])	8.72 [4.84]	7.60 [4.33]
Child Sleep Habits total score (mean [SD])	45.03 [7.40]	42.65 [5.98]
Maternal Stressful Life Events exposure		
Stably low	21.22 (16.65, 25.79)	48.25 (44.03,52.47)
Fluctuating Low*	9.97 (6.62, 13.32)	6.26 (4.22, 8.31)
Fluctuating High*	6.43 (3.69, 9.17)	3.68 (2.09, 5.27)
Decreasing	14.47 (10.54, 18.40)	11.42 (8.73, 14.10)
Increasing	22.19 (17.54, 26.83)	19.15 (15.83, 22.47)
Stably high	25.72 (20.84, 30.61)	11.23 (8.57, 13.90)

Note: For the CSHQ, a higher score indicates a higher number of altered sleep habits. For the SDQ, a higher score indicates a higher number of reported difficulties.

*Fluctuating Low refers to low SLE at T1 & T3, but high SLE at T2. Fluctuating High refers to high SLE at T1 & T3, with low SLE at T2.

SLE exposure, for Māori and non-Māori children. Māori children had a higher mean CSHQ score, indicating more disordered sleep than non-Māori children. Māori children were also significantly more likely than non-Māori to have received a diagnosis of asthma/eczema/allergy (36.8% vs 27.7%). Māori children also had a higher mean SDQ score compared to non-Māori children, suggesting more developmental concerns. Māori children were more likely than non-Māori to have mothers exposed to SLE, of any pattern. Most Māori children (78.8%, compared to 51.8% of non-Māori) in our study had a mother experiencing multiple SLE during at least one period in their first 3 years of life. The ethnic difference was greatest in the 'stably high' group, with 25.7% (95% CI 20.8–30.6) of Māori children having mothers exposed to ≥2 SLE at all time periods, compared to 11.2% (95% CI 8.6–13.9) of non-Māori children.

Table 3 presents the results of multiple regression analyses of child CSHQ and SDQ scores adjusted for maternal ethnicity, maternal age, NZDep quintiles and maternal SLE trajectory. For both CSHQ and SDQ, there was a relationship between maternal exposure to multiple SLE and more concerning sleep and development scores. For CHSQ, the association was strongest for the 'fluctuating high' and the 'stably high' groups, and was also strong in the 'increasing' and 'fluctuating low' groups. The 'stably high' and 'increasing' maternal SLE groups were independently associated with more concerning SDQ scores, whereas 'fluctuating' and 'decreasing' groups were not. There was no statistically significant relationship between either child's NZDep score and CSHQ or SDQ. Older maternal age at T1 was associated with lower (better) SDQ

Table 3. Multiple regression analysis of CSHQ and SDQ scores, by maternal demographic variables & SLE exposure.

	Model 1 – CSHQ total score		Model 2 – SDQ total score	
Variable	Estimate (Standard error)	*p*-value	Estimate (Standard error)	*p*-value
Maternal Stressful Life Event exposure				
Stably low	0 (ref)		0 (ref)	
Fluctuating Low*	***2.127* (*0.917*)**	***0.0207***	−0.196 (0.611)	0.748
Fluctuating High*	***2.953* (*1.183*)**	***0.0128***	0.436 (0.753)	0.563
Decreasing	1.326 (0.770)	0.0853	0.736 (0.502)	0.143
Increasing	***2.366* (*0.639*)**	***0.0002***	***1.020* (*0.422*)**	***0.0159***
Stably high	***2.78* (*0.729*)**	***0.0001***	***1.798* (*0.482*)**	***0.0002***
Maternal neighbourhood deprivation				
Quintile 1 (least deprived)	0 (ref)		0 (ref)	
Quintile 2	0.771 (0.692)	0.266	0.116 (0.458)	0.801
Quintile 3	0.412 (0.675)	0.542	0.131 (0.453)	0.772
Quintile 4	0.501 (0.753)	0.507	0.697 (0.495)	0.159
Quintile 5 (most deprived)	0.887 (0.794)	0.265	0.965 (0.525)	0.0665
Maternal ethnicity				
Māori	***2.47* (*0.552*)**	***<0.0001***	0.682 (0.364)	0.0616
Non-Māori	0 (ref)		0 (ref)	
Maternal age group				
16–24	0 (ref)		0 (ref)	
25–29	−0.326 (0.817)	0.69	−1.02 (0.532)	0.0559
30–34	−0.459 (0.774)	0.554	***−1.714* (*0.507*)**	***0.0008***
35+	0.107 (0.819)	0.896	***−1.95* (*0.537*)**	***0.0003***

Notes: For the CSHQ, a higher score indicates a higher number of altered sleep habits. For the SDQ, a higher score indicates a higher number of reported difficulties. Statistically significant values in bold.

* Fluctuating Low refers to low SLE at T1 & T3, but high SLE at T2. Fluctuating High refers to high SLE at T1 & T3, with low SLE at T2.

Table 4. Estimated mean CSHQ and SDQ score by maternal SLE trajectory group.

Maternal SLE group	Mean CSHQ	95% CI		Mean SDQ	95% CI	
Fluctuating low SLE	44.90	43.28	46.53	7.63	6.54	8.71
Fluctuating high SLE	45.73	43.55	47.91	8.26	6.88	9.63
Decreasing SLE	44.10	42.79	45.42	8.56	7.71	9.40
Increasing SLE	45.14	44.12	46.17	8.84	8.17	9.52
Stable high SLE	45.56	44.43	46.68	9.62	8.87	10.36
Stable low SLE	42.78	41.93	43.63	7.82	7.26	8.38

Notes: For the CSHQ, a higher score indicates a higher number of altered sleep habits. For the SDQ, a higher score indicates a higher number of reported difficulties.

scores. Māori ethnicity was independently significantly associated with more concerning CSHQ scores, even after controlling for SLE exposure, and deprivation.

Table 4 shows the estimated CSHQ and SDQ scores (and 95% CI) for each of the different SLE exposure groups which were calculated using the Least Squares Means obtained from our models. For example, the difference in mean CSHQ scores is significantly higher between the 'fluctuating high' (45.73) and 'stably low' group (42.78), followed by 'stably high' (45.56) vs. 'stably low', then 'increasing' (45.14) vs. 'stably low' and finally 'fluctuating low' (44.90) vs. 'stably low'. For SDQ, the difference in mean scores is significantly highest between the 'stably high' (9.62) vs. 'stably low' (7.82) and 'increasing' (8.84) vs. 'stably low' groups.

Table 5 shows a multiple regression analysis of the chance of children reporting a diagnosis of asthma, eczema or allergy at age 3 years, by demographic variables and SLE exposure. Children in the 'fluctuating high' group were 2.22 times more likely (95% CI 1.11–4.41) to have a diagnosis of these conditions compared with the 'stably low' group, but the relationship for other SLE exposure groups was not significant. We found no statistically significant relationship between NZDep, ethnicity, or maternal age with child diagnoses of asthma, eczema or allergy.

Table 5. Regression analysis of chance of reporting a diagnosis of asthma, eczema or allergy, by demographic variables and SLE exposure.

	Reporting asthma/eczema/allergy OR (95% CI)
Deprivation	
NZ Dep Quintile 2	1.23 (0.78, 1.93)
NZ Dep Quintile 3	1.17 (0.75, 1.83)
NZ Dep Quintile 4	1.20 (0.74, 1.94)
NZ Dep Quintile 5	1.13 (0.68, 1.88)
Ethnicity	
Māori vs Non-Māori	1.18 (0.84, 1.67)
Age	
16–24	1.30 (0.78, 2.17)
25–29	0.88 (0.56, 1.38)
30–34	1.04 (0.71, 1.51)
Maternal Stressful Life Event exposure	
Fluctuating Low	1.18 (0.65, 2.14)
Fluctuating High	**2.22 (1.11, 4.41)**
Decreasing	1.19 (0.73, 1.94)
Increasing	1.35 (0.90, 2.04)
Stably high	1.22 (0.77, 1.95)

Notes: Statistically significant values in bold. Fluctuating Low refers to low SLE at T1 & T3, but high SLE at T2. Fluctuating High refers to high SLE at T1 & T3, with low SLE at T2.

Discussion

Using data from a large longitudinal cohort of Māori and non-Māori, women and their children, this study examined the association between longitudinal trajectories of maternal stressful life events and child health and development. To our knowledge, we present the first information to suggest that having a mother experiencing multiple SLE during a child's critical early years is very common in New Zealand – but what is especially concerning is that it appears as though this is almost the norm for Māori children with more than a quarter of Māori children having a mother who is experiencing multiple stressful life events from late pregnancy through to when their child is 3–4 years of age. The impacts of multiple stressful events versus more isolated episodes need to be better understood, especially given this large ethnic disparity in the nature of exposure.

This paper highlights evidence of significant systemic failures which impact Māori mothers and children. Upholding Māori women's rights to live in safe and secure environments needs greater policy intervention, and is required to achieve the New Zealand Government's objective for New Zealand to be the best place in the world to be a child (Department of the Prime Minister and Cabinet 2019), and its ambition to create peaceful homes where children, families and whānau thrive (New Zealand Government 2021). This includes more support/services for Māori parents, as well as addressing the socioeconomic and structural drivers contributing to stressful life events in the first place, as recognised in Te Aorerekura: the national strategy to eliminate family violence and sexual violence (New Zealand Government 2021).

Given how common multiple maternal SLE are during children's early years, it is even more important and urgent to understand the impacts this stress has on child health and development outcomes. We were able to demonstrate a clear association between maternal experiences of stressful life events and child health outcomes – at least for disordered sleep and concerns about development. While we did find some evidence of association between maternal SLE and selected child health diagnoses, the strength of our associations was likely limited by fact that diagnoses in this young age group are relatively uncommon which impacted on statistical power. We were also not so able to fully assess the contribution of maternal experiences of SLE to ethnic inequities in child health outcomes, although we did clearly demonstrate that more Māori children have mothers exposed to multiple SLE, and that these maternal SLE are associated with poorer child sleep and development outcomes. Future research drawing on a larger sample size of Māori and non-Māori children is required to further assess the impact of this disproportionate exposure to maternal SLE on Māori children, and what contribution this makes to ethnic inequities in child health and wellbeing. It is also important to note that not all children who are exposed to maternal stressful life events go on to have poor development or health outcomes. Further research is needed to better understand potential protective factors for Māori children whose mothers experienced stressful life events but who had good health outcomes.

Our analysis suggests that there may be some differences in impact depending on when the exposure to SLE occurs. For sleep, maternal SLE when the child is 3 years of age is associated with poor child sleep at that age, regardless of whether or not the mother has experienced SLE at earlier times. The impact of 'historical' maternal SLE

exposure on child sleep disturbances was less clear: maternal SLE starting high in pregnancy +/− around the time of birth, and then recovering (decreasing group) did not seem to be associated with poorer child sleep patterns at age 3 years, although the fluctuating low group who were exposed to stress around the time of birth then recovered did experience more disordered child sleep at age 3 years. For child development, our findings also suggested that timing of exposure matters. In contrast, previous NZ research found that timing of stress exposure did not seem to make a difference for child BMI outcomes (Farewell et al. 2018). When considering SDQ scores, the groups with high SLE at 3 years of age were more likely to be associated with more concerning child development scores at 3 years of age. However, exposure to multiple maternal SLE earlier in pregnancy or infancy was not associated with concerning development scores at 3 years of age.

These findings raise the suggestion that the mechanism through which SLE impacts child sleep and development may be more likely to be 'environmental' rather than 'biological', because if SLE exposure in pregnancy was biologically embedded we would expect to still see a higher rate of problematic sleep and development in the 'decreasing' group. These findings also suggest that there may be an immediacy of the relationship between SLE exposure and adverse child outcomes co-occurring – it makes sense that if children are exposed to stressful environments, there might be an impact on socioemotional wellbeing and behaviours at the same time, although what is not yet understood is whether this impact on children reverses/recovers once the stressful environment is resolved. The mother and young child are a unit, and SLE experienced by the mother are also experienced in some way directly by the child in a shared environment (whether, for example, during pregnancy through factors in the maternal bloodstream, or in early childhood in the shared home environment). Our study is not able to disentangle the relative impact on the child of SLE that the mother may experience alone, or SLE that are also experienced more directly by the child. Rather we consider that our findings reinforce the importance of looking after the wellbeing of mothers well beyond the pregnancy/puerperium, by demonstrating that maternal SLE exposure when her child is aged 3 years is associated with adverse child sleep and development measures, even if there were no SLE during pregnancy and the puerperium.

A strength of this study is that it was Indigenous-led research, applying critical Kaupapa Māori research principles not only to the research process, but to the framing of questions, analysis and interpretation of data. Other strengths include the large proportion of the sample who were Māori (almost 40%), and the longitudinal cohort methodology which provided repeated measures with the same participants at each time period. Although *Moe Kura* does not claim to be a nationally-representative sample, the demography of Māori and non-Māori participants closely mirrors that expected for the pregnant population of New Zealand at the time of recruitment (Paine et al. 2022), thus results are broadly generalisable.

Moe Kura was predominantly designed as a longitudinal study of maternal wellbeing, and so collected relatively little child-centred health information which limited the extent to which we could investigate the relationship between maternal SLE and child health outcomes. It is also important to note that this study contained no measure of perceived maternal stress, so we are not able to determine how stressful life events related to levels of perceived stress for the mothers. Other limitations include the reliance on self-

reported measures, and the low prevalence of health diagnoses in the child cohort at age 3 years. Whilst the CSHQ and SDQ are internationally validated tools, there is uncertainty about the validity of tools in different ethnic groups, which may introduce bias into the study. For example, previous research has identified that the SDQ tool is not culturally equivalent across different ethnic groups in New Zealand (Kersten et al. 2016). It is also important that the results of this analysis are not interpreted from a deficit view which seeks to scrutinise Māori women and blame individual Māori mothers for the inequities experienced by Māori children. The findings of this study must be interpreted within the context and frame of ongoing systemic inequities and institutional disadvantage for Māori in New Zealand society.

Conclusions

In summary, this analysis demonstrates a clear association between maternal experiences of SLE and disordered child sleep and development concerns at age 3 years. While not able to fully assess the contribution of maternal SLE to ethnic inequities in child health outcomes, we did clearly demonstrate that more Māori children have mothers exposed to multiple SLE, and that these maternal SLE are associated with poorer child outcomes. Most children in our cohort had a stressful early life environment at least sometimes, but concerningly more than a quarter of Māori children had a mother experiencing multiple SLE on all occasions measured. The impacts of pervasive exposure to stressful events versus more isolated episodes need to be better understood, especially given this large ethnic disparity in the nature of SLE exposure. This research also suggests that maternal SLE cause harm for children even when they occur beyond the pregnancy and puerperal period, and this reinforces the importance of optimising wellbeing for all mothers of young children.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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Data availability statement

The data that support the findings of this study may be available from the Primary Investigators of the *Moe Kura* study (SJP and TLS), upon reasonable request.

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