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RESEARCH ARTICLE

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# Associations between specific and cumulative adverse childhood experiences, childhood obesity, and obesogenic behaviours

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## ABSTRACT

**Background**: Individuals impacted by adverse childhood experiences (ACEs) are at greater risk of developing obesity, however, few studies have prospectively measured ACEs and obesity during childhood. Associations with the adoption of obesogenic behaviours during childhood, which directly contribute to obesity are also understudied.

**Objective**: To examine associations between individual and cumulative ACEs, obesity, and obesogenic behaviours during childhood.

**Methods**: Data came from *Growing Up in New Zealand*. The study sample was restricted to those who provided obesity data at age 8 and one child per mother, resulting in an analytic sample of 4895 children. A newly developed ACEs index consisted of nine individual ACEs and cumulative ACEs scores (0, 1, 2, 3, 4+ ACEs), two obesity measures (BMI and waist circumference/height ratio), and eight obesogenic behaviours including unhealthy dietary behaviours, inadequate sleep duration, excessive screen time, and physical inactivity were included in the analyses.

**Results**: ACEs were prevalent among this cohort of NZ children. By age eight, 87.1% of children experienced at least one ACE and 16% experienced at least 4 ACEs. Six individuals assessed ACEs showed significant associations with childhood obesity (AORs ranging from 1.22 to 1.44). A significant dose–response effect was observed where the experience of a higher number of ACEs was associated with greater risk for obesity (AORs increased from 1.78 for one ACE to 2.84 for 4+ ACEs). Further, a significant dose–response relationship was found between experiencing two or more ACEs and higher odds of adopting obesogenic behaviours (AORs ranging from 1.29 for physical inactivity to 3.16 for no regular breakfast consumption).

**Conclusions:** ACEs exposure contributes to population-level burden of childhood obesity. Our findings highlight the importance of a holistic understanding of the determinants of obesity, reinforcing calls for ACEs prevention and necessitating incorporation of ACEs-informed services into obesity reduction initiatives.

## Asociaciones entre experiencias adversas en la infancia específicas y acumulativas, obesidad infantil y conductas obesogénicas

**Antecedentes**: Las personas afectadas por experiencias adversas en la infancia (ACEs, por sus siglas en inglés) tienen un mayor riesgo de desarrollar obesidad. Sin embargo, pocos estudios han medido prospectivamente las ACEs y la obesidad durante la infancia. Además, las asociaciones con la adopción de conductas obesogénicas en la infancia, que contribuyen directamente a la obesidad, están insuficientemente estudiadas.

**Objetivo**: Examinar las asociaciones entre ACEs individuales y acumulativas, la obesidad y las conductas obesogénicas durante la infancia.

**Métodos**: Los datos provienen del estudio *Growing Up in New Zealand*. La muestra del estudio se restringió a niños con datos de obesidad a los 8 años y a un solo niño por madre, resultando en una muestra analítica de 4.895 niños. Un índice de ACEs desarrollado recientemente incluyó nueve ACEs individuales y puntuaciones acumulativas de ACEs (0, 1, 2, 3, 4+ ACEs). Se analizaron dos medidas de obesidad (IMC y relación circunferencia de cintura/altura) y ocho conductas obesogénicas, incluidas conductas alimentarias poco saludables, duración inadecuada del sueño, tiempo excesivo frente a pantallas e inactividad física.

**Resultados**: Las ACEs fueron prevalentes en esta cohorte de niños neozelandeses. A los 8 años, el 87.1% de los niños había experimentado al menos una ACE y el 16% había experimentado al menos cuatro ACEs. Seis ACEs individuales evaluadas mostraron asociaciones significativas con

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Adverse childhood experiences; childhood obesity; obesogenic behaviours; obesity reduction; New Zealand

## PALABRAS CLAVE

Experiencias adversas en la infancia; obesidad infantil; conductas obesogénicas; reducción de la obesidad; Nueva Zelanda

## HIGHLIGHTS

- Adverse Childhood Experiences (ACEs) were prevalent among children in New Zealand.
- Six of nine assessed ACEs were associated with higher risk of adopting obesogenic behaviours and developing childhood obesity.
- Experience of a higher number of ACEs was associated with a higher risk of adopting obesogenic behaviours and developing childhood obesity.

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la obesidad infantil (razones de probabilidades ajustadas [AOR] entre 1.22 y 1.44). Se observó un efecto dosis–respuesta significativo, donde un mayor número de ACEs se asoció con un mayor riesgo de obesidad (AOR que aumentaron de 1.78 para una ACE a 2.84 para 4+ ACEs). Además, se encontró una relación dosis–respuesta significativa entre la experiencia de dos o más ACEs y mayores probabilidades de adoptar conductas obesogénicas (AOR de 1.29 para inactividad física a 3.16 para la falta de consumo regular de desayuno).
**Conclusiones**: La exposición a ACEs contribuye a la carga poblacional de obesidad infantil. Nuestros hallazgos destacan la importancia de una comprensión holística de los determinantes de la obesidad, reforzando los llamados a la prevención de ACEs y la necesidad de incorporar servicios informados en ACEs en las iniciativas de reducción de obesidad.

## 1. Introduction

Childhood obesity is a serious public health issue due to short- and long-term effects on physical and mental health (Di Cesare et al., 2019; Simmonds et al., 2015). New Zealand (NZ) has the third-highest childhood obesity rates globally (UNICEF 2020), with approximately one in eight children aged 2–14 years (12.7%) classified as obese in 2022, an increase from 9.5% in 2019/2020 (Health NZMo, 2014). Early prevention efforts that address structural, environmental, and inequitable contributors are critical at the population level (Pandita et al., 2016), as is a deeper understanding of early childhood risk factors (Zhou et al., 2020). Obesity has proven resistant to conventional and individualised treatments, partly due to biological adaptations such as reduced metabolic rate and increased appetite-increasing hormones (Greenway, 2015). There has been calls to examine a broader range of social, emotional, and environmental contexts of childhood development to gain a comprehensive understanding of obesity risk factors (Baranowski et al., 2019; Davison & Birch, 2001; Schroeder et al., 2021). Childhood trauma or severely stressful life events have gained traction as significant factors that increase the likelihood of developing obesity (Schroeder et al., 2021). Adverse childhood experiences (ACEs) are defined as potentially traumatic events or environmental factors during childhood that can undermine a child's sense of safety, stability, and bonding (CDC, 2021). Since the seminal ACEs Study, these experiences have become a key focus in research on child development (Felitti et al., 1998). ACEs include indicators of child abuse and maltreatment (e.g. child physical, psychological, and sexual abuse), as well as family dysfunctions such as household mental illness, substance abuse, incarceration, parental separation/divorce or death, and intimate partner violence against female caregiver, all occurring before the age of 18 (CDC, 2021; Felitti et al., 1998). In a recent retrospective investigation undertaken in New Zealand, 55% of the sample population had experienced one or more ACEs (Fanslow et al., 2021), which have recently garnered increasing recognition as significant determinants of health in NZ (Hashemi et al., 2021).

Previous international research has established a link between ACEs and obesity in adulthood, suggesting a positive dose–response relationship, where the odds of obesity increase with a higher number of ACEs (Simmonds et al., 2015). Recent studies have also found that experiencing an adverse event before the age of nine increases the risk of obesity later in life (Gardner et al., 2019). However, few studies have specifically examined the link between these two public health issues earlier in life, with most focusing on adolescents (aged 10 and 18) (Ahn et al., 2020; Clark et al., 2010; Shenk et al., 2016; Veldwijk et al., 2012) and only a limited number addressing early to middle childhood (McKelvey et al., 2019; Mehari et al., 2020). This leaves a critical gap in understanding the developmental periods during which individuals are most vulnerable to the impact of ACEs on obesity. Moreover, existing research on this topic is limited by several factors. For example, while the cumulative impact of multiple ACEs on obesity risk is recognised, more research is needed to determine whether different types of ACEs have varying impacts on obesity risk (Danese & Tan, 2014). Additionally, some studies have relied on retrospective data, which involve adults recalling their childhood experiences and weight status (Baranowski et al., 2019), a method prone to recall biases and measurement error that may lead to over- or under-estimation of the prevalence or consequences of ACEs. Some studies have narrowly focused on maltreatment-related ACEs (physical, sexual, and/or emotional abuse), either examining each type individually or collectively, without considering other types of ACEs (Hawton et al., 2018; Keeshin et al., 2013; Schneiderman et al., 2015). Moreover, findings across studies are inconsistent, with some identifying associations only for specific ACEs (Boynton-Jarrett et al., 2010; Keeshin et al., 2013; Morris et al., 2016), in particular age groups (Morris et al., 2016), or within one gender (either boys or girls) (Boynton-Jarrett et al., 2010; Kidman et al., 2020; Schneiderman et al., 2015). Variations in research methods, such as reliance on parent- or

child-reported measurements of weight and height instead of objective measurements or using only a single measure of unhealthy weight outcomes (e.g. BMI versus body fat percentage) may have contributed to these consistencies and further complicated the results (Boynton-Jarrett et al., 2010; Deng & Lacey, 2022; Isohookana et al., 2016; Kidman et al., 2020). More research is required to verify the ACEs-obesity link found in some studies. Furthermore, the differential impact of ACEs on obesity across various ethnic, gender, and socio-economic groups is also not well understood and warrants further investigation.

Another significant research gap lies in the original list of preventable childhood adversities proposed in the seminal U.S. and subsequently reused in many other studies. This list omits several key domains that many developmental researchers consider important predictors of immediate health and well-being outcomes (Finkelhor et al., 2013), including exposure to violence outside the family such as peer victimisation. International ACE scales may also overlook contextual or cultural specificities; for example, wholesale adoption of international ACE scales may overlook experiences related to discrimination and racism in countries like NZ with histories of colonisation (Joy & Beddoe, 2019). Further research is needed to explore the extent to which these adverse events, both individually and in combination with other ACEs, are associated with childhood obesity.

There have been speculations about potential mechanisms linking ACEs to poor weight outcomes. The suggested mechanisms for adults encompass (but are not limited to) mental and emotional perturbations (e.g. depression), social disruption, maladaptive coping responses (e.g. binge eating), chronic stress responses, inflammation and metabolic disturbances (Wiss & Brewerton, 2020; Zeller et al., 2015). Research on adult populations has also shown that these pathways may also contribute to the adoption of high-risk 'obesogenic' unhealthy behaviours (e.g. excessive screen time, high consumption of low-nutrient food, physical inactivity, and sleep difficulties) by those who experience ACEs (Maurya & Maurya, 2023). However, to date, few population-based epidemiologic studies have investigated the joint associations between ACEs, a constellation of obesogenic behaviours, and obesity risk in populations of children (Harada et al., 2021; Santos et al., 2023). By integrating these interconnected factors into a unified investigation, a more holistic understanding of the pathways linking early life experiences to obesity risk can be attained.

Much of the research on the ACEs-obesity link has been concentrated in the US and Europe. To better understand its global relevance, more studies are needed in other regions and countries (Schroeder et al., 2021). Despite the high prevalence of ACEs in NZ (Fanslow et al., 2021; Hashemi et al., 2021), their connections with childhood obesity and obesogenic behaviours have not yet been verified. This research aimed to: (a) describe the prevalence of exposure to a comprehensive index of ACEs among a cohort of NZ school-aged children and by their sociodemographic characterises, (b) investigate associations between individual and cumulative ACEs and childhood obesity in NZ context, (c) investigate whether association between ACEs and childhood obesity vary across different gender, ethnic, and socio-economic groups, (d) examine associations between ACEs and a constellation of childhood obesogenic behaviours that are well-known contributors to childhood obesity.

## 2. Methods

### 2.1. Study sample

Participants were members of Growing Up in NZ (GUiNZ), a contemporary child cohort study, which enrolled 6822 pregnant women and 4401 of their partners, and then the 6853 children born to these women who survived to age 6 weeks (Morton et al., 2015). Cohort profile and recruitment have been described in depth elsewhere (Morton et al., 2012; Morton et al., 2014). Briefly, pregnant women were eligible if they had an estimated delivery date between 25 April 2009 and 25 March 2010, and resided in a defined geographical region of New Zealand selected for its ethnic, socioeconomic and geographic diversity (Auckland, Counties-Manukau and Waikato). The original child cohort included 11% of the national birth cohort during the recruitment period and their characteristics were broadly generalisable to the contemporary New Zealand birth cohort (2007–2010) (Morton et al., 2015). Data collection waves (DCWs) involved both face-to-face and phone interviews to gather comprehensive information on various aspects of the children's development and family environment. Face-to-face interviews, conducted during all major DCWs except 6-week DCW, were primarily used to collect detailed and sensitive information, such as health assessments and anthropometric measurements, ensuring accuracy and reliability. Phone interviews were utilised to gather follow-up data that required less in-depth interaction, reducing the burden on participants and maintaining high response rates.

Follow-up procedures included regular data collection points from pregnancy through various stages of the children's development. Major DCWs occurred antenatally, at birth, at 6 weeks, at 9 months, at 2 years, at 4.5 years, and at 8 years, up to the time of this study.

### 2.2. Current study sample

The current study was restricted to one child per participating mother to prevent data clustering effects,

(i.e. where mothers gave birth to twins or triplets), and those who responded to the 8-year DCW and had obesity data, which reduced the sample to 4895 children. The reduction in sample size from 6-week to 8-years DCW was due to several exclusion criteria: loss to follow-up (1015), incomplete or missing data on obesity variables (647), withdrawal of consent by parents (282), and child deaths (14). Ethics approval was granted by with the NZ Ministry of Health Northern Y Regional Ethics Committee, and all enrolled parents provided informed consent. Reporting of this analysis follows the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines (von Elm et al., 2007).

### 2.3. Measures

All variable definitions are provided in Table 1. Nine ACEs were measured using indicators and instruments of childhood adversities (child report, parent report, and standard questionnaires) in 8 waves of GUiNZ (from birth up to age 8): ethnic discrimination, parental divorce, emotional abuse, parental incarceration, exposure to intimate partner violence (IPV), physical abuse, parental substance abuse, household mental illness, and peer bullying. An ACEs score (0, 1, 2, 3, 4+ ACEs) was generated. No data on children's direct experience of ethnic discrimination was collected as part of the GUiNZ study up to and including the 8-year data collection wave. Therefore, the mother's exposure to ethnic discrimination was used as a proxy for the child's exposure to this ACE, which is considered appropriate for the NZ context (Joy & Beddoe, 2019). The concept of 'linked lives' supports this approach, showing how a parent's experiences, such as discrimination, can impact a child's development due to the intergenerational transmission of stress and the significant influence maternal experiences can have on a child's development (Kelly et al., 2013). Research also suggests that vicarious experiences of discrimination, especially during 'sensitive periods', can lead to feelings of helplessness and have lasting effects on a child's mental and physical health (Heard-Garris et al., 2018).

As recommended by prior research (Schroeder et al., 2021), two measures of obesity were used, BMI and waist circumference/height ratio or WHtR (a measure of central/abdominal adiposity). Body mass was measured by interviewers collecting child's weight, height, and waist circumference measurements at age 8. BMI-for-age z scores were derived using World Health Organization 2006 child growth chart standards and binarised into presence of obesity defined as BMI ≥95th percentile vs absence of obesity. WHtR was calculated by dividing the waist circumference values (cm) by the height values (cm), with a cutoff point of ≥0.5 considered as high (Yoo, 2016).

Eight mother-reported obesogenic behaviours were explored at age 8 (Daniels & Hassink, 2015), including unhealthy dietary behaviours (4 indicators), inadequate sleep duration, excessive screen time, and physical inactivity (2 indicators). The selection of these behaviours was based on their established associations with an increased risk of childhood obesity, as documented in the literature and confirmed by the current study (see Table 5). These behaviours include frequent fast food and soft drink consumption (Braithwaite et al., 2014), inadequate fruit and vegetable consumption (Bourke et al., 2014), skipping or no regular breakfast, age-specific inadequate sleep duration (Malihi et al., 2021) age-specific excessive screen time (Malihi et al., 2021) and low physical activity (Agbaje et al., 2023; Anderson et al., 2017) (see Table 5). NZ Ministry of Health and WHO guidelines were followed in creating derived variables (Ministry of Health, 2012; Ministry of Health, 2020; WHO, 2020) (see Table 1 for variable definitions).

Sociodemographic variables were used to explore the prevalence rates of ACEs and obesity measures among sub-populations and to account for potential confounders in multivariable analyses. Child's sex and ethnicity were included as demographic characteristics, and food insecurity and area deprivation level (Atkinson et al., 2014) were included as socioeconomic measures. Sensitivity analyses demonstrated that the child's birth weight, maternal age at pregnancy, and maternal educational level did not significantly alter the observed associations between ACEs and weight outcomes. Therefore, they were not included in the multivariable analyses reported in this paper.

### 2.4. Statistical analyses

Statistical analyses were performed using Stata 15 (Stata Statistical Software 2017: Stata Statistical Software: Release 15, 2017). Prevalence rates for all variables were computed for the whole sample and by sociodemographic characteristics, and chi-square tests were used to evaluate bivariate associations between ACEs, obesity, and sociodemographic variables (Tables 2 and 3). Associations between individual ACEs, ACE scores and each obesity outcome were assessed using logistic regression analyses, presenting odds ratios unadjusted and adjusted for sociodemographic characteristics (child's sex, child's ethnicity, and food security) (Table 4). We did not adjust for area-level deprivation to avoid multicollinearity with other sociodemographic variables. Individual ACEs were analysed separately to explore whether certain ACEs were driving associations when using the ACE score or whether patterns differed across types of ACEs. To explore sociodemographic differences in the associations between

**Table 1.** Variable definitions of adverse childhood experiences (ACEs) and obesogenic behaviours used in analysis.

| Derived variable | Question |
|---|---|
| Emotional abuse | Child was classified as being emotionally abused if mothers and/or partner reported the following in DCW2, DCW54M, or DCW8:<br>– **Always or almost always** have lost their temper with child, raised their voice, yelled or shouted at child (mother reported, DCW8),<br>– **Very often or always** yelled or shouted when child misbehaved or exploded with anger (mother reported, DCW54M),<br>– **Very often** shouted when child was naughty or **very often, extremely often, or all the time** got angry at the child, criticise their child's ideas, shouted at the child because they were upset with their child (mother and/or partner reported, DCW2). |
| Physical abuse | Child was coded as having this ACE if:<br>– Mothers responded '**often', 'always or almost always'** to using physical punishment such as smacking as a way of interacting with their child (DCW8)<br>– Mothers used physical punishment as a way of disciplining their child '**half the time', 'very often', or 'always'** (DCW54M).<br>– Mother reported smacking the child '**often' 'very often'** when he/she was naughty (DCW2). |
| Parental substance abuse (including alcohol abuse) | Child was coded as exposed to this ACE if the mother or partner reported any of the followings:<br>– Mother reported that the child has experienced this ACE (drug taking/alcoholism in the immediate family, DCW8)<br>– Mother reported heavy episode drinking (drinking 5 or more standard drinks on a typical day when drinking, DCW54M, DCW8, or drinking 6 or more standard drinks on one occasion at least monthly, DCW54M and DCW8).<br>– Mother reported drinking 10 or more drinks per week (DCW54M)<br>– Mother ever sought help in relation to alcohol use (DCW54M)<br>– Mother or partner reported using amphetamines, cocaine, ecstasy, opiates, hallucinogens or party pills since the birth of the child (DCW9M) |
| Parental mental illness | Child was assigned to this ACE if any of the following was the case:<br>– Mother reported that the child has been exposed to mental illness in the immediate family (DCW8)<br>– Mother or partner was classified as moderately or severely depressed (a score of 15 or higher on the Patient Health Questionnaire Depression Screener) or as probably depressed (a score of 12 or higher on Edinburgh Postnatal Depression Scale) (DCW8, DCW54M, DCW1, or DCW0) |
| Parental incarceration | Child was assigned to this ACE if any of the following was the case:<br>– Mother reported that the child has experienced this ACE (parent in prison, DCW8)<br>– Mother reported that she has ever been convicted of a crime which resulted in a jail sentence (DCW54M)<br>– Partner reported that he has ever been convicted of a crime which resulted in a jail sentence (DCW1) |
| Parental separation/ divorce | Child was coded as having this ACE if the mother reported any of the following:<br>– Child has experienced divorce/separation of parents (DCW8)<br>– Mother was not in a relationship with this same partner when her child was two years old<br>– Mother did not have a current partner during 54 months, 2 years, or 9 months interviews. |
| IPV against mother | Child was coded as having this ACE if mother reported:<br>– Child was present when she had a physical conflict with her partner.<br>– Or, mother reported any of the following during past 4 weeks prior to the data collection in any frequency other than never:<br>– Their partner slapped them or threw things at them that could have hurt them; pushed or shoved them or pulled their hair; hit them with a fist or something else that could have hurt them (DCW54M, DCW8) or<br>– Mother and partner pushed and shoved each other while arguing (DCW1) |
| Mother discrimination | Child was coded as having this ACE if mother reported that:<br>– She has ever been treated unfairly in New Zealand because of her ethnicity (DCW2) Or mother has ever felt any of the following within the past 12 months or more than 12 months ago (DCW0):<br>– Being a victim of an ethnically motivated attack – that is verbal, or physical abuse to the person or property in New Zealand<br>– Being treated unfairly (e.g. treated differently, kept waiting) by a health professional (e.g. a doctor, nurse, dentist) because of your ethnicity in New Zealand<br>– Being treated unfairly at work or been refused a job because of your ethnicity in New Zealand<br>– Being treated unfairly when renting or buying housing because of your ethnicity in New Zealand<br>– Being treated unfairly by the police, the justice system (courts), or the corrections department (prison, community service, periodic detention, parole, probation) because of your ethnicity in New Zealand<br>– Being treated unfairly when asking for loans, a mortgage, hire purchase or credit cards because of your ethnicity in New Zealand<br>– Being treated unfairly when attending a place of learning because of your ethnicity in New Zealand |
| Recurrent peer-bullying/peer victimisation | Child was coded as having this ACE if he/she reported frequently (at least once or twice a month) experiencing of one of the following (DCW8):<br>– Do other children put you down, call you names, or tease you in a mean way?<br>– Do other children leave you out in a mean way?<br>– Do other students hit, push, or hurt you in a mean way?<br>– Do other children tell lies about you in a mean way?<br>– Do other children threaten you in a mean way, or force you to do things?<br>– Do other children take or break your stuff in a mean way (e.g. money or pens)?<br>– Do other children say mean things about your culture or family?<br>– Are other children mean to you because you learn in a different way to them?<br>– Do other children use cell phones (like texting) or the Internet (like Facebook) to be mean to?<br>– At school, are you bullied by other students? |
| Inadequate fruit (<2) & vegetable (<3) consumption | On average, how many servings of fruit does [child] eat per day? On average, how many servings of vegetables does [child] eat per day? Responses were categorised as eating less than 5 servings of vegetables or fruits per day versus more than 5 servings. |
| Frequent soft drink consumption | In the past 7 days, how many times did [child] have a fizzy or soft drink, such as cola or lemonade? Responses were categorised as two or more fizzy drink versus none. |

(*Continued*)

**Table 1.** Continued.

| Derived variable | Question |
|---|---|
| Frequent fast food consumption | In the past 7 days, how many times did [child] eat any food from a fast-food place or takeaway shop, such as fish and chips, burgers, fried chicken or pizza? Includes snacks & mealtimes. Responses were categorised as two or more times versus none. |
| No regular/daily breakfast consumption | Over a usual week, how many days does [child] eat breakfast? Responses were coded as 7 days a week versus <7. |
| Inadequate sleep duration | On average, how much time does [child] spend asleep at night in total? Following ministry of health guideline responses were coded as less than 9 h (inadequate sleep duration) versus 9 or more hours. |
| Excessive screen time | – On a normal **weekday**, spending time watching television programming including free to-air, online, and pay TV or DVDs either on TV or other screen-based devices? – On a normal **weekend** day, spending time watching television programming including free to-air, online, and pay TV or DVDs either on TV or other screen-based devices? Following NZ Ministry of Health and WHO guidelines for screen viewing limitations at age 5 to 17 years, total screen time per day was created as a dichotomous variable representing children who limited screen-viewing time to less than two hours per day (met the guideline) vs. those who viewed one hour or more per day (exceeded the guideline). |
| Leisure-time physical inactivity | What does [child] usually do when they have a choice about how to spend free time? A binary variable was created for 'usually doing inactive things: yes/no'. |
| Leisure time sedentary behaviour | Some kids would rather play outdoors in their spare time BUT Other kids would rather watch TV (Would prefer TV sort of/really true for me versus would prefer playing outdoors sort of/really true) |
| Food security | At DCW8, Mothers were asked: 'We can afford to eat properly. How often has this been true for your household over the past year?' Responses were categorised as 'Always', labelled as food secure, versus 'Sometimes'/'Never', labelled as food insecure. |
| Area deprivation level | Area deprivation level at DCW8 was derived by GUINZ team according to NZDep2013 Index of Deprivation (Atkinson et al., 2014). |
| Child's prioritised ethnicity | Child ethnicity was externally prioritised based on total response ethnicity collected following the NZ Ministry of Health protocol: Māori, Pacific, Asian, MELAA [Middle East, Latin American, African], European/New Zealander/Other. European and New Zealanders categories were combined, as prior research has shown that the majority of those who identify as New Zealanders are of European descent (Cormack & Robson, 2010). Due to small sample size, 'Other' ($n = 2$) was also combined with European/New Zealander. For simplicity, we refer to this group as European throughout this paper. |

ACEs scores and childhood obesity, interaction terms were added to multivariable logistic regressions. No interaction terms were significant (except one which may have been due to chance given the large number of tests), therefore no stratified analyses are reported. Associations between ACEs scores and obesogenic behaviours were assessed using logistic regression analyses and odds ratios, adjusting for sociodemographic characteristics (Table 5). The association between obesogenic behaviours and obesity (BMI) was also explored to ensure that the obesity-related behaviours included in the analyses were indeed associated with an increased risk of obesity, confirming findings from the literature (Bourke et al., 2014; Braithwaite et al., 2014; Malihi et al., 2021). Statistical significance was set at $p<.05$.

## 3. Results

### 3.1. Characteristics of the study sample

Just over half of the study population were boys (51.5%), and half were European (49.8%). Māori children comprised 22.8% of the sample, followed by Asian (14.4%), and Pacific children (10.9%), 19.1% of the sample were identified as food insecure (Table 3). Compared to children recruited at baseline, children lost to attrition by age 8 were more likely to be in lower socioeconomic groups, identified with an ethnicity other than European, have younger mothers, and recorded as having obesity at age 4.5 years (Supplementary eTable 1).

### 3.2. Prevalence of obesity outcomes, individual, and cumulative ACEs among the study sample and by sociodemographic characteristics

Obesity and high WHtR were equally prevalent in the sample, with almost 15% of children identified with obesity or high WHtR status. Boys, children identified as Pacific and Māori, those living in a food insecure household or in the most deprived areas had significantly higher obesity and WHtR rates (Table 3). Of the individual ACEs, experience of bullying was the most prevalent ACE and reported by more than half of the sample (58.5%), followed by parental experience of discrimination (33.0%). The least prevalent exposure category was having an incarcerated parent (2.1%). Of cumulative ACEs scores, 87.1% of the study sample reported at least one ACE by age 8 and exposure to multiple ACEs was widespread with 27% reported 2 ACEs and one in three (33%) reported at least 3 ACEs. Higher ACEs scores and experience of each specific ACEs were significantly more prevalent among those identified as Māori or Pacific, those living in food insecure households or in the most deprived areas (Tables 2 and 3).

### 3.3. Prevalence of obesity among those exposed to individual and cumulative ACEs

Children who experienced physical abuse had the highest rate of obesity (24.8%), followed by those whose mothers experienced IPV (22.9%) and those who experienced parental separation (22.5%). The prevalence of obesity among those who reported no experience of

**Table 2.** Prevalence of individual ACEs for the whole analytic sample and by sociodemographic characteristics.

| | Emotional abuse | Physical abuse | Parental substance abuse | Parental mental illness | Parental incarceration *n* (%) | Parental separation or divorce | IPV against mother | Parent experience of discrimination | Bullying |
|---|---|---|---|---|---|---|---|---|---|
| Total sample *n* (%) | 1019 (20.6) | 870 (17.6) | 1314 (26.6) | 1054 (21.3) | 104 (2.1) | 763 (15.4) | 548 (11.5) | 1625 (33.0) | 2849 (58.5) |
| Missingness | <10[a] (0.0) | <10 (0.1) | <10 (0.0) | <10 (0.0) | 20 (0.4) | <10 (0.0) | 184 (3.7) | 20 (0.4) | 72 (1.5) |
| Child's gender | | | | | | | | | |
| Boy | 543 (21.3) | 507 (19.9) | 648 (25.4) | 552 (21.6) | 52 (2.1) | 394 (15.5) | 287 (11.7) | 848 (33.4) | 1527 (61.1) |
| Girl | 476 (19.9) | 363 (15.2) | 666 (27.8) | 502 (20.9) | 52 (2.2) | 369 (15.4) | 261 (11.3) | 777 (32.5) | 1322 (55.7) |
| $\chi^2$ (*P*-value) | 1.52 (0.3) | **19.96 (0.001)** | 3.63 (0.06) | 0.36 (0.55) | 0.10 (0.75) | 0.002 (0.96) | 0.17 (0.68) | 0.40 (0.53) | **14.85 (0.001)** |
| Child's prioritised ethnicity | | | | | | | | | |
| Māori | 269 (24.2) | 234 (21.1) | 497 (44.7) | 288 (25.9) | 60 (5.4) | 291 (26.2) | 146 (14.2) | 491 (44.4) | 666 (61.0) |
| Pacific | 189 (35.2) | 214 (39.9) | 143 (26.6) | 165 (30.7) | 11 (2.1) | 127 (23.7) | 124 (25.0) | 205 (38.4) | 350 (66.4) |
| Asian | 174 (24.7) | 145 (20.6) | 42 (6.0) | 172 (24.4) | <10 (0.7) | 64 (9.1) | 162 (23.4) | 309 (44.2) | 387 (56.1) |
| MELAA | 18 (17.1) | 15 (14.3) | 14 (13.3) | 17 (16.2) | <10 (0.0) | 14 (13.3) | <10 (8.9) | 39 (37.50) | 67 (63.8) |
| European/NZer | 360 (14.9) | 258 (10.7) | 608 (25.1) | 386 (16.0) | 24 (0.99) | 243 (10.1) | 101 (4.2) | 557 (23.1) | 1339 (56.0) |
| $\chi^2$ (*P*-value) | **134.6 (0.001)** | **275.64 (0.001)** | **351.52 (0.001)** | **90.11 (0.001)** | **83.96 (0.001)** | **204.91 (0.001)** | **315.22 (0.001)** | **220.04 (0.001)** | **25.62 (0.001)** |
| Area level deprivation (NZ DEP 2013) | | | | | | | | | |
| Least deprived (1–3) | 253 (14.4) | 175 (10.0) | 377 (21.4) | 289 (16.4) | 14 (0.8) | 161 (9.2) | 115 (6.7) | 492 (28.0) | 940 (54.3) |
| Moderately deprived (4–7) | 373 (20.1) | 290 (15.6) | 480 (25.9) | 371 (20.0) | 30 (1.6) | 253 (13.6) | 185 (10.3) | 609 (32.9) | 1072 (58.3) |
| Most deprived (8–10) | 388 (29.8) | 399 (30.7) | 451 (34.6) | 389 (29.8) | 60 (4.6) | 345 (26.5) | 244 (20.2) | 514 (39.8) | 824 (64.5) |
| $\chi^2$ (*P*-value) | **108.31 (0.001)** | **229.09 (0.001)** | **67.29 (0.001)** | **83.21 (0.001)** | **56.45 (0.001)** | **179.07 (0.001)** | **132.74 (0.001)** | **46.53 (0.001)** | **31.27 (0.001)** |
| Experienced food insecurity | | | | | | | | | |
| Yes | 262 (29.6) | 249 (28.2) | 329 (37.1) | 307 (34.7) | 48 (5.5) | 262 (29.6) | 182 (22.6) | 371 (42.1) | 551 (63.6) |
| No | 664 (17.9) | 532 (14.3) | 930 (25.0) | 657 (17.7) | 49 (1.3) | 440 (11.8) | 319 (8.8) | 1119 (30.2) | 2093 (57.0) |
| $\chi^2$ (*P*-value) | **61.11 (0.001)** | **97.31 (0.001)** | **52.62 (0.001)** | **124.22 (0.001)** | **58.73 (0.001)** | **173.77 (0.001)** | **126.56 (0.001)** | **45.74 (0.001)** | **12.33 (0.001)** |

[a]Cells have fewer than 10 counts. Exact numbers not reported to protect the anonymity of participants.

**Table 3.** Prevalence of ACEs scores and obesity outcomes by sociodemographic characteristics in the analytic sample of GUiNZ participants.

| | Total sample *n*(%) | 0 ACE | 1 ACE | 2 ACEs *n* (%) | 3 ACEs | 4+ ACEs | Any ACEs | Obesity (BMI) | WHtR |
|---|---|---|---|---|---|---|---|---|---|
| Full sample | 4946 (100) | 640 (12.9) | 1355 (27.4) | 1334 (27.0) | 828 (16.7) | 789 (16.0) | 4306 (87.1) | 709 (14.48) | 752 (15.59) |
| Child's gender | | | | | | | | | |
| Boy | 2522 (51.5) | 303 (11.9) | 689 (27.1) | 693 (27.2) | 432 (16.9) | 433 (17.0) | 2247 (88.1) | 410 (16.26) | 376 (15.16) |
| Girl | 2373 (48.5) | 337 (14.1) | 666 (27.8) | 641 (26.8) | 396 (16.5) | 356 (14.9) | 2059 (85.9) | 299 (12.60) | 376 (16.04) |
| $\chi^2$ (*P*-value) | | 8.52 (0.07) | | | | | **5.22 (0.02)** | **13.20 (0.001)** | 0.72 (0.397) |
| Child's prioritised ethnicity | | | | | | | | | |
| Māori | 1099 (22.8) | 85 (7.7) | 199 (17.9) | 298 (26.8) | 216 (19.5) | 313 (28.2) | 1026 (92.4) | 235 (21.38) | 224 (20.65) |
| Pacific | 524 (10.9) | 27 (5.0) | 82 (15.3) | 126 (23.5) | 131 (24.4) | 171 (31.8) | 510 (94.97) | 224 (42.75) | 201 (38.80) |
| Asian | 695 (14.4) | 75 (10.7) | 175 (24.9) | 219 (31.1) | 130 (18.5) | 105 (14.9) | 629 (89.4) | 70 (10.07) | 90 (13.06) |
| MELAA | 105 (2.2) | <10 (8.6) | 35 (33.3) | 35 (33.3) | 18 (17.1) | <10 (7.6) | 96 (91.4) | <10 (8.57) | 15 (14.42) |
| European/NZer | 2406 (49.8) | 431 (17.8) | 853 (35.3) | 638 (26.4) | 318 (13.2) | 179 (7.4) | 1988 (82.2) | 160 (6.65) | 209 (8.84) |
| $\chi^2$ (*P*-value) | | **579.85 (0.001)** | | | | | **114.14 (0.001)** | **514.16 (0.001)** | **319.52 (0.001)** |
| Area level deprivation | | | | | | | | | |
| Least deprived (1–3) | 1745 (35.8) | 308 (17.5) | 610 (34.7) | 491 (27.9) | 221 (12.6) | 128 (7.3) | 1450 (82.5) | 147 (8.42) | 160 (9.30) |
| Moderately deprived (4–7) | 1841 (37.8) | 242 (13.1) | 521 (28.1) | 517 (27.9) | 320 (17.3) | 256 (13.8) | 1614 (87.0) | 202 (10.97) | 235 (12.92) |
| Most deprived (8–10) | 1283 (26.3) | 86 (6.6) | 218 (16.7) | 317 (24.3) | 280 (21.5) | 403 (30.9) | 1218 (93.4) | 354 (27.59) | 353 (28.02) |
| $\chi^2$ (*P*-value) | | **468.64 (0.001)** | | | | | **79.39 (0.001)** | **248.68 (0.001)** | **209.56 (0.001)** |
| Experienced food insecurity | | | | | | | | | |
| Yes | 871 (19.1) | 62 (7.0) | 141 (15.9) | 189 (21.3) | 193 (21.8) | 301 (33.9) | 824 (93.0) | 232 (26.64) | 235 (27.6) |
| No | 3689 (80.9) | 543 (14.62) | 1134 (30.5) | 1053 (28.5) | 566 (15.2) | 418 (11.3) | 3171 (85.4) | 403 (10.92) | 444 (12.2) |
| $\chi^2$ (*P*-value) | | **354.59 (0.001)** | | | | | **36.39 (0.001)** | **145.12 (0.001)** | **127.13 (0.001)** |

[a]Cells have fewer than 10 counts. Exact numbers not reported to protect the anonymity of participants.

**Table 4.** Prevalence and risk of obesity by individual ACEs and ACEs scores by age 8 in analytic sample from Growing Up in New Zealand study.

| | Obesity (BMI) ($n = 709$) | | | WHtR ($n = 752$) | | |
|---|---|---|---|---|---|---|
| Individual ACEs | **No. (%)** | **OR (95%CI)** | **AOR (95%CI)** | **No. (%)** | **OR (95%CI)** | **AOR (95%CI)** |
| Emotional abuse ($n$= 1019) | 203 (20.1) | **1.68 (1.41–2.02)** | **1.22 (1.01–1.50)** | 206 (21.0) | **1.58 (1.32–1.89)** | **1.21 (1.01–1.48)** |
| Physical abuse ($n = 870$) | 212 (24.8) | **2.36 (1.97–2.83)** | **1.44 (1.16–1.78)** | 209 (24.7) | **2.08 (1.74–2.49)** | **1.42 (1.16–1.75)** |
| Parental substance abuse ($n = 1314$) | 229 (17.7) | **1.39 (1.17–1.65)** | 1.16 (0.95–1.41) | 241 (18.9) | **1.39 (1.17–1.64)** | **1.23 (1.02–1.49)** |
| Parental mental illness ($n = 1054$) | 210 (20.2) | **1.70 (1.46–2.04)** | **1.26 (1.02–1.55)** | 209 (20.5) | **1.56 (1.30–1.86)** | 1.18 (0.97–1.47) |
| Parental incarceration ($n = 104$) | 19 (18.6) | 1.36 (0.82–2.26) | 0.83 (0.47–1.45) | 21 (20.8) | 1.43 (0.88–2.33) | 0.88 (0.51–1.52) |
| Parental separation/Divorce ($n = 763$) | 168 (22.5) | **1.93 (1.59–2.34)** | **1.27 (1.01–1.59)** | 186 (25.3) | **2.10 (1.74–2.54)** | **1.59 (1.28–1.96)** |
| IPV against mother ($n$= 548) | 123 (22.9) | **2.03 (1.63–2.54)** | 1.18 (0.91–1.54) | 117 (22.2) | **1.73 (1.38–2.16)** | 1.14 (0.86–1.48) |
| Parent experience of discrimination ($n$= 1625) | 289 (18.1) | **1.52 (1.29–1.79)** | **1.32 (1.09–1.59)** | 283 (18.0) | **1.31 (1.11–1.54)** | 1.12 (0.94–1.34) |
| Bullying ($n = 2849$) | 459 (16.22) | **1.49 (1.26–1.76)** | **1.35 (1.12–1.63)** | 492 (17.6) | **1.50 (1.27–1.77)** | **1.41 (1.18–1.69)** |
| ACEs Score | | | | | | |
| Ref. 0 ACEs ($n = 640$) | 41 (6.5) | Ref. | Ref. | 46 (7.3) | | |
| 1 ACE ($n = 1355$) | 137 (10.2) | **1.65 (1.15–2.36)** | **1.78 (1.19–2.67)** | 157 (11.9) | **1.70 (1.21–2.40)** | **1.82 (1.25–2.64)** |
| 2 ACEs ($n = 1334$) | 180 (13.6) | **2.28 (1.60–3.24)** | **1.93 (1.29–2.88)** | 193 (14.7) | **2.18 (1.56–3.06)** | **1.84 (1.27–2.67)** |
| 3 ACEs ($n = 828$) | 143 (17.5) | **3.07 (2.13–4.42)** | **2.08 (1.37–3.16)** | 151 (18.7) | **2.91 (2.05–4.12)** | **2.17 (1.48–3.20)** |
| 4+ ACEs ($n = 789$) | 208 (26.9) | **5.32 (3.74–7.59)** | **2.84 (1.88–4.28)** | 205 (27.0) | **4.67 (3.32–6.56)** | **2.87 (1.95–4.21)** |
| Any ACEs (One or more) ($n = 4306$) | 668 (15.7) | **2.69 (1.94–3.74)** | **2.06 (1.42–2.98)** | 706 (16.8) | **2.55 (1.87–3.48)** | **2.05 (1.46–2.90)** |

OR = Unadjusted odds ratios; AOR = Odd ratios adjusted for child's gender, food insecurity, child's prioritised ethnicity' CI = confidence interval.

ACEs (6.5%) was almost half of that reported for the whole sample (14.5%) and that reported for those with at least one ACE (15.7%). A dose–response effect was observed, as those with higher ACEs scores had higher rates of obesity (10.2%, 13.6%, 17.5%, 26.9% for those who respectively experienced one, two, three, or at least four ACEs) (Table 4).

### 3.4. Associations between individual and cumulative ACEs and obesity outcomes

After adjustment for potential confounders, six out of nine assessed ACEs (emotional and physical abuse, parental mental illness, parental separation/divorce, exposure to discrimination, and peer-bullying) showed significant associations with childhood obesity (AORs ranging from 1.22 to 1.44). Physical abuse showed stronger associations with obesity than other ACEs. At the cumulative level, exposure to any number of ACEs (even one) was associated with increased risk for obesity. A significant dose–response effect was also observed where experience of a higher number of ACEs was associated with greater risk for obesity (AORs increased from 1.78 for one ACE to 2.84 for 4+ ACEs). A similar pattern was observed using WHtR (Table 4).

### 3.5. Associations between individual and cumulative ACEs and obesogenic behaviours

Experience of two ACEs was significantly associated with higher odds of adopting dietary obesogenic behaviours, such as higher odds of consuming inadequate servings of fruits and vegetables, higher odds of consuming fast food and soft drinks, and lower odds of regularly consuming breakfast, after adjustment for socio-demographic factors (AORs ranging between 1.33 and 1.85). The risk increased as the numbers of ACEs increased, as those with 4+ ACEs had 2 to 3-fold increased risk of reporting obesogenic dietary behaviours (Table 5). A similar dose–response pattern was observed for non-dietary obesogenic behaviours (inadequate sleep duration, excessive screen time, and sedentary behaviours) but associations reached significance for increased likelihood of reporting the non-dietary obesogenic behaviours where children experienced at least three ACEs (1.51 to 1.80 times for excessive screen time and inadequate sleep duration, respectively). The odds were higher for those who experienced 4+ ACEs (AORs ranging between 1.59 and 2.43) (Table 5).

## 4. Discussion

ACEs were common among this prospective cohort of children in NZ. By age eight, 87.1% of children experienced at least one ACE and 16% reported four or more. Notably, observable inequities were observed; children from financially disadvantaged households and those identified as Māori or Pacific had the highest prevalence of almost all types of ACEs and higher ACE scores. The prevalence rates observed in this study were substantially higher than those reported in retrospective cross-sectional ACEs studies utilising standardised ACEs questionnaires (e.g. CDC study) (CDC, 2021; Hashemi et al., 2021). However, they align with rates from other prospective ACE studies using data collected at multiple time points (Houtepen et al., 2020). This consistency suggests that prospective studies, like ours, may provide a more accurate representation of ACE prevalence by capturing experiences in real-time rather than relying on adult recollections.

The lack of standardised measures for assessing ACEs complicates comparisons across studies, yet our findings suggest that studies relying on a single retrospective questionnaire may underestimate the prevalence of ACEs. Our comprehensive list of ACEs, including those with the highest prevalence (such as exposure to ethnic discrimination and peer

**Table 5.** Adjusted multivariable logistic regression for associations between obesogenic behaviours and ACEs score, and obesogenic behaviours and childhood obesity.

| | | ACEs | | Obesity (BMI) | Obesity (WHtR) |
|---|---|---|---|---|---|
| Obesogenic behaviour | **ACEs Score** | **No. (%)** | **AOR (95% CI)[a]** | | |
| Inadequate fruit and vegetable consumption | 1 | 245 (34.8) | 1.31 (0.98.1.75) | **1.51 (1.16–1.97)** | **1.86 (1.44–2.40)** |
| (<2 fruits, <3veg) | 2 | 283 (40.8) | **1.39 (1.03–1.86)** | | |
| | 3 | 200 (49.0) | **1.62 (1.17–2.23)** | | |
| | 4+ | 211 (58.0) | **2.00 (1.42–2.81)** | | |
| Frequent soft drink consumption | 1 | 285 (23.7) | 1.19 (0.93–1.52) | **1.99 (1.62–2.43)** | **1.73 (1.43–2.10)** |
| (2+ fizzy/soft drink) | 2 | 332 (28.9) | **1.43 (1.12–1.82)** | | |
| | 3 | 257 (39.0) | **1.97 (1.51–2.56)** | | |
| | 4+ | 280 (48.0) | **2.23 (1.70–2.93)** | | |
| Frequent fast food consumption | 1 | 306 (25.0) | 1.07 (0.85–1.35) | **1.51 (1.24–1.84)** | **1.64 (1.36–1.98)** |
| (2+ times) | 2 | 383 (32.3) | **1.33 (1.05–1.67)** | | |
| | 3 | 305 (43.5) | **1.90 (1.48–2.44)** | | |
| | 4+ | 330 (52.5) | **2.26 (1.74–2.93)** | | |
| No regular/daily breakfast consumption | 1 | 84 (6.7) | 1.37 (0.89–2.10) | **1.86 (1.47–2.35)** | **1.82 (1.45–2.30)** |
| | 2 | 129 (10.5) | **1.85 (1.22–2.80)** | | |
| | 3 | 121 (16.3) | **2.55 (1.67–3.89)** | | |
| | 4+ | 179 (25.6) | **3.16 (2.08–4.81)** | | |
| Inadequate sleep duration | 1 | 118 (9.8) | 1.36 (0.94–1.98) | **1.59 (1.24–2.04)** | **1.69 (1.33–2.14)** |
| (<9 h) | 2 | 119 (10.3) | 1.25 (0.87–1.82) | | |
| | 3 | 117 (17.1) | **1.80 (1.23–2.63)** | | |
| | 4+ | 147 (24.9) | **2.43 (1.66–3.56)** | | |
| Excessive screen time | 1 | 456 (44.0) | 1.05 (0.85–1.30) | **1.65 (1.31–2.08)** | **1.65 (1.32–2.05)** |
| ( = >2 h) | 2 | 465 (47.3) | 1.15 (0.93–1.42) | | |
| | 3 | 280 (56.0) | **1.51 (1.17–1.94)** | | |
| | 4+ | 245 (61.4) | **1.78 (1.35–2.36)** | | |
| Leisure time physical inactivity | 1 | 493 (40.4) | 1.20 (0.98–1.47) | **1.85 (1.53–2.24)** | **1.73 (1.45–2.08)** |
| (reported by mother) | 2 | 507 (43.1) | **1.29 (1.05–1.58)** | | |
| | 3 | 301 (42.8) | 1.24 (0.99–1.56) | | |
| | 4+ | 352 (54.1) | **1.87 (1.47–2.37)** | | |
| Leisure time sedentary behaviour | 1 | 443 (33.1) | 1.07 (0.86–1.32) | **1.41 (1.17–1.70)** | **1.53 (1.28–1.82)** |
| (reported by child) | 2 | 481 (36.6) | 1.23 (0.99–1.53) | | |
| | 3 | 348 (42.4) | **1.56 (1.23–1.97)** | | |
| | 4+ | 338 (43.4) | **1.59 (1.25–2.03)** | | |

AOR = Odd ratios adjusted for child's gender, food insecurity, child's prioritised ethnicity; CI = 95% confidence interval.

bullying), likely contributed to the high overall prevalence observed in this study.

Compared to those with zero ACEs, children who experienced any number of ACEs were more likely to develop obesity and a high Waist-to-Height Ratio (WHtR) in middle childhood, with the risk increasing as the number of ACEs rose. This dose–response relationship is consistent with previous research, which has found that the accumulation of ACEs is associated with a higher risk of obesity and related outcomes (McKelvey et al., 2019; Mehari et al., 2020; Schroeder et al., 2021). Interestingly, while associations by individual ACEs followed similar patterns to ACEs scores, certain ACEs (e.g. physical abuse) related more strongly to obesity than others. These findings underscore the importance of assessing associations by individual ACEs, which is often overlooked in ACE research (Schroeder et al., 2021). Associations between ACEs (both individual and cumulative) and obesity outcomes attenuated after adjustment for ethnicity and socioeconomic factors but remained significant. This suggests that while socioeconomic factors considerably confound the association between ACEs and obesity, these patterns cannot be fully explained by socioeconomic settings in which they occur. Similarly, in line with previous research (Björkenstam et al., 2013; Copeland et al., 2018; Halfon et al., 2017; Houtepen et al., 2020), we found no evidence to suggest that associations between ACEs and obesity were weaker among children from families of higher socioeconomic status, as interaction terms between ACEs and socioeconomic factors were non-significant and associations were of similar magnitude in families regardless of their experience of material deprivation. This indicates that the impact of ACEs on obesity is pervasive across socioeconomic strata, reinforcing the need for universal ACE prevention and support interventions rather than targeting such initiatives exclusively to populations of lower socioeconomic status.

Nevertheless, higher prevalence of ACEs among ethnic minority groups, particularly Māori and Pacific peoples, and those with lower socioeconomic status likely exacerbates inequities in obesity and other health outcomes across the life course. Trauma-informed and culturally-appropriate approaches, along with adequate resourcing of such services, are especially important for communities that experience structural inequities and discrimination in access to healthcare, including obesity treatment (Pihama et al., 2017). The need for these approaches is supported by global research highlighting the intergenerational and historical impacts of trauma on Indigenous communities (Pihama et al., 2017).

Our findings demonstrated that an increased count of ACEs is associated with elevated risk of adopting

obesogenic behaviours in a dose–response manner. This relationship aligns with previous studies that have examined the impact of ACEs on weight-related behaviours, although our study extends this research by examining these behaviours in combination rather than in isolation (Al-shoaibi et al., 2024; Ashour et al., 2024; Jackson et al., 2021). The number of ACEs required to reach significance varied across the different obesogenic behaviours; with the odds of engaging in unhealthy dietary behaviours becoming significant at a lower ACEs level than those for non-dietary behaviours.

Although our study did not explicitly test for mediation effects, the observed associations between ACEs, obesogenic behaviours, and childhood obesity suggest a plausible pathway in which the increased likelihood of adopting obesogenic behaviours may mediate the relationship between ACEs and the development of childhood obesity. This supports the hypothesis put forward by previous research, which has proposed that the accumulation of early stressors in children's family, school and social environments can lead to psychological distress, influencing appetite regulation and preference for highly palatable 'comfort foods', thereby increasing the risk of obesity (Tate et al., 2015).

Importantly, obesogenic dietary behaviours are also driven by structural factors, such as poverty-related distress and geographic and cost inaccessibility of healthful foods for those experiencing material deprivation (Drewnowski, 2012; Pearce et al., 2007). In combination with the current toxicity of external environments where fast foods and soft drinks are easily available, cheap, and extensively marketed (Vandevijvere et al., 2016), unresolved stress and trauma in a large segment of the child population may limit the success of siloed child obesity prevention plans.

These findings suggest that addressing the associations identified in this study requires considering children's social environments, experiences, and ACE-related psychopathology in obesity-reduction efforts (Fröhlich et al., 2011). This challenges the approach of traditional weight-loss interventions that exclusively focus on behavioural change for obesity management.

The high prevalence of ACEs found in this study and their associations with childhood obesity and key obesogenic behaviours suggests that children presenting to relevant services with obesity should be carefully evaluated for the presence of ACEs to provide appropriate responses. Where ACEs are identified, clear pathways for healing and trauma-informed responses must be readily available to all affected children. This is crucial for mitigating the negative impact of ACEs on health outcomes and preventing re-traumatisation. It is important that individual-level care is embedded within trauma-informed environments (e.g. at healthcare and educational settings) and consider structural and social violence, including racism and discrimination (Blitz & Lee, 2015; Tabone et al., 2020). This requires sufficient training and support for those working with children to offer trauma-sensitive and effective care.

### 4.1. Strengths

This study utilised a comprehensive index of ACEs that extended beyond the traditional list to capture a broader range of childhood experiences. Our prospective design, with data collected over multiple time points, mitigated the common limitations of retrospective studies, such as recall bias and measurement error, by tracking children's experiences in real-time rather than relying on adult recollections. Our use of two different objective measurements for assessing obesity (BMI and WHtR) also ensures robustness of our outcome measures and findings. Additionally, by exploring two key aspects of the ACEs-obesity link in our study (obesogenic behaviours and the socioeconomic differences), we offered a holistic perspective on this relationship. This holistic approach facilitates the identification of pathways through which ACEs might influence obesity and variations in impact across different groups, offering valuable insights for targeted interventions. This research contributes valuable data from NZ, broadening the global understanding of ACEs and obesity, supporting the development of ACE-informed policies and interventions.

### 4.2. Limitations

The study is limited by loss to follow-up particularly among groups at higher risk for obesity and ACEs, such as Māori and Pacific children and those with lower socioeconomic status. The loss has likely resulted in underestimations of the magnitude of associations between ACEs and obesity outcomes, as well as the prevalence of ACEs in the wider population. This limitation not only affects the generalisability of our findings but also introduces potential selection bias. We cannot determine causation between ACEs and obesity or obesogenic behaviours due to the observational nature of the study and insufficient time between measurement of some ACEs exposure and outcome variables; research has previously shown that it may take at least two years for ACEs to manifest as unhealthy weight (Schroeder et al., 2021). However, this concern is alleviated by the consistency of findings assessing ACEs and weight outcomes, the dose–response relationship observed, and the articulation of causal pathways between

ACEs and health that have been identified through other studies (Wiss & Brewerton, 2020).

Due to ethical considerations, data on sexual abuse (an ACE strongly associated with obesity) (Schroeder et al., 2021) has not been collected in GUiNZ as of age eight. Additionally, this study mainly used mother-reported data, as only a subgroup of the cohort had available or complete partner data. Mothers' reports may have presented social desirability biases for questions that may invoke feelings of guilt or shame, such as those related to parenting behaviours, parental relationships, mental health, drug abuse, and health behaviours. Child-reported data (including on sexual abuse) and health behaviours should continue to be explored as these data become available into adulthood.

The higher prevalence of child-reported ACEs (e.g. peer bullying) compared to proxy-reported ACEs (e.g. parental exposure to discrimination and parental mental illness) likely reflects both true variations in adversities and differences in reporting sources. However, disentangling these factors was beyond the scope of this study and these different reporting methods could influence the observed prevalence rates and, consequently, the study's findings.

Where data was stratified by subgroups (such as ethnicity) in addition to ACEs and obesity, sample size limitations may have resulted in false-negative findings. Future research must explore ethnic-specific needs and experiences to develop understanding of culturally appropriate and effective prevention and intervention programmes and policies to interrupt pathways between adversity and obesity outcomes.

A bidirectional association between peer bullying and obesity may have been present (Puhl & King, 2013; van Geel et al., 2014), which may also be compounded by ethnicity and gender (Gillon, 2020). The potential mediating roles of other factors, such as mental health and coping strategies, merit further investigation. Further research is warranted to obtain a nuanced understanding of the timing and chronicity of ACE occurrences on the likelihood of developing obesity.

We did not explicitly test the mediation effects of obesogenic behaviours in our study. Future research should investigate this potential mediation effect to better understand the pathways connecting ACEs and obesity outcomes.

## 5. Conclusion

This study contributes to the growing evidence that ACEs are associated with children's weight outcomes. We found that six potentially preventable ACEs were significantly associated with the development of obesity among NZ children, and cumulative ACEs exposure presented a dose–response relationship with the likelihood of obesity development. Although our study did not explicitly test for mediation effects, our findings suggest that ACEs may influence obesity outcomes through their associations with unhealthy weight-related obesogenic behaviours. Our results indicate that childhood obesity reduction efforts may benefit from considering the role of ACEs. Understanding and addressing the social determinants of obesity, such as family and social environments, may be important in the context of traditional behavioural change interventions targeting nutrition, sleep, screen time, and physical activity. However, our findings and conclusions should be interpreted with caution due to the observational nature of this study.

## Disclosure statement

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

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## Data availability statement

The data utilised in the submitted manuscript was collected by the Growing Up in New Zealand study team. Researchers seeking access to this data may apply by submitting a data access application to the Data Access Committee at (dataaccess@growingup.co.nz).

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