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RESEARCH ARTICLE

Biocultural and social determinants of ill health and early mortality in a New Mexican paediatric autopsy sample

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Abstract

Illness and mortality have social origins, and infants and children are especially susceptible to the impacts of adverse social experiences. Early-life stress (ELS) – physiological disruptions suffered by a developing organism – is incorporated into human biology through embodiment. This paper examines whether children who lived and died in New Mexico (2011–2019) embodied social determinants of health. Data were collected from 780 postmortem computed tomography scans in conjunction with data from field notes and autopsy reports for individuals aged 0.5–20.99 years from New Mexico. Variables included in linear/logistic regressions are the per cent of families in poverty by ZIP code and year, housing type (trailer/mobile home, apartment, house), rural/urban residence areas, and race/ethnicity. Health outcome variables are age at death, respiratory conditions, growth stunting and arrest, and porous cranial lesions. Intersections of poverty, housing disparities, and race/ethnicity are examined to understand whether children from New Mexico incorporated ELS into their biology. **Results**. Hispanic children have higher odds of growth stunting than non-Hispanic White children. Native American children die younger and have higher odds of respiratory diseases and porous lesions than Hispanic and non-Hispanic Whites. Rural/urban location does not significantly impact age at death, but housing type does. Individuals who lived in trailers/mobile homes had earlier ages at death. When intersections between housing type and housing location are considered, children who were poor and from impoverished areas lived longer than those who were poor from relatively well-off areas. **Conclusions.** Children's health is shaped by factors outside their control. The children included in this study embodied experiences of social and ELS and did not survive to adulthood. They provide the most sobering example of the harm that social factors (structural racism/discrimination, socioeconomic, and political structures) can inflict.

Keywords: morbidity and mortality; social determinants of health; postmortem computed tomography; inequity; early-life stress; housing type; rural/urban

Research highlights

Hispanic children have higher odds of growth stunting. Native American children from New Mexico have younger ages at death than non-Hispanic White and Hispanic children. They also have higher odds of having respiratory conditions. Results indicate that children embody social determinants of health.

Introduction

The society humans live in acts to shape their health and well-being (Krieger, 1999, 2005, 2014; Waitzkin, 1981). Infants, children, and adolescents are especially affected by social factors (Garg *et al.*, 2015). Their health and well-being are shaped not just by parenting and parents' socioeconomic status (SES) but by personal circumstances and other factors (Coll-Seck *et al.*, 2019; Marmot, 2005; Viner *et al.*, 2012).

Humans are biocultural beings, incorporating all aspects of the lived experience into biology through the process of embodiment (Gravlee, 2009; Krieger, 2005). Embodiment begins at the level of the oocyte when stress experienced by the mother (exposures to infections, psychosocial stress, and environmental toxins) is incorporated into its biology (Barker, 1995; Gluckman *et al.*, 2007). Embodiment is observable in population-level patterns of health, disease, and overall well-being (Krieger, 1999, p. 296). Social determinants of health (SDOH), including inadequate housing, lack of access to necessary resources, and poverty, may have lasting impacts across the life course (from conception to death). Infants are born carrying evidence of their mother's stress, but they also incorporate their own experiences into their body as they grow and develop. Early-life stress [ELS] (*in-utero,* infancy, childhood), imposed by societal pressures external to the child, also shapes health outcomes later in life (Barker *et al.*, 1993; Barker *et al.*, 1989). While every child who experiences adverse physical and social conditions is at risk, some have increased burdens.

Society can buffer people from poor health (by buffering them from stress), or it can introduce stressors that create ill health (Schell, 1997). Health-damaging experiences can result from inequitable and poor political, economic, and social policies (Marmot *et al.*, 2008, p. 1661). Health-damaging conditions – poverty, childhood abuse, racism, lack of access to healthcare, and poor housing conditions (Gottlieb *et al.*, 2014; Morone, 2017; Trent *et al.*, 2019) – do not exist in a vacuum; they are interrelated, and their effects are cumulative (Beck *et al.*, 2014). For example, impoverished families experience other issues, including food insecurity, housing instability, and lack of access to transportation and health care (Morone, 2017). The probability of poorer health outcomes increases with risk exposures (Gottlieb *et al.*, 2014; Morone, 2017).

This study examines whether children who lived and died in New Mexico incorporated social stressors and inequity into their bodies [embodiment] and how those stressors shaped their ultimate health outcomes. While their lives were short, their skeletons and bodies may act as a record of how they experienced SDOH. Because SDOH are embodied (Krieger, 2014; Krieger and Davey Smith, 2004), the effects of SDOH can be detected on the skeleton (Harrod *et al.*, 2017; Klaus *et al.*, 2017; O'Donnell and Edgar, 2020). SDOH can lead to elevated burdens of chronic and acute illnesses and traumatic injury (Cassidy *et al.*, 2013; Foote *et al.*, 2015; Gottlieb *et al.*, 2014).

Child health in New Mexico

Compared to other states, New Mexico ranks poorly (50th in the nation) for childhood well-being (NMVoices, 2020). Here, well-being is ranked using multiple metrics, including 1) health (environmental health, substance use, weight, mortality rates, others), 2) family and community (teen birth rates, adult education, and others), 3) education (enrolment and attendance, reading/math proficiency, graduation from high school), and 4) socioeconomic status (housing costs, food insecurity, family income, poverty, parental employment). These low ranks may be driven by characters unique to the state, including population diversity, rural geography, low population density, and relatively low socioeconomic status (herein, SES) (Malik *et al.*, 2014; New Mexico Department of Health, 2012).

In New Mexico, 62% of children are Hispanic, 22% are non-Hispanic White, and 11% are Native American (NMVoices, 2023). Twenty-five per cent of New Mexican children were living in poverty (familial income at or below $21,330) between 2018 and 2019 (NMVoices, 2020). The median household income in New Mexico is $54,020, lower than the United States median of

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$69,021 (NMVoices, 2023). Finally, child and teen death rates are high in New Mexico, 43 per 100,000 (compared to the U.S. average of 30 per 100,000). When these are split into children (0–14 years) and teens (15–19 years), the death rates per 100,000 are 49.8 and 90.5, respectively (NMVoices, 2023). While this information comes from more recent NMVoices reports, the trends reported have remained consistent across recent years (NMVoices, 2010).

All New Mexican children experience increased risks for poor health (NMVoices, 2020), but children who reside in tribal areas (on and off reservation trust lands) fare worse. Child poverty rates, death rates, restriction and lack of resource access, and other issues are heightened in tribal areas (NMVoices, 2020, p. 66). Native Americans who live away from tribal lands have poorer outcomes than individuals in other racial/ethnic groups.

Race, a social construct with biological consequences

Because race/ethnicity is a prominent feature of this paper, some definitions and discussion are necessary. 'Race' is a social construct composed of physical and cultural features (Blakey, 1999; Jablonski, 2012; Krieger, 2010; Marks, 1996). As such, concepts and definitions of 'race' and 'racial' identity are fluid, changing across time and space and between nations (Alba, 1990; Harris and Sim, 2002; Lieberson and Waters, 1993). Despite this, race shapes social experiences, health/well-being, and biology (Gravlee, 2009). Race's complex relationship with biology and health is enabled, in part, by other social and political constructs: racism, discrimination, and structural violence. Through embodiment, SDOH can be expressed biologically (de la Cova, 2012; Gravlee, 2009; Krieger, 2014).

Indigenous people experience high rates of infant and child mortality, low life expectancy at birth, malnutrition and growth retardation, high infectious disease burdens, and health issues, including diabetes and cardiovascular disease (Gracey and King, 2009). These problems may stem partly from the cascade effects of colonization: poverty, poor living conditions, lack of access to healthcare, governmental indifference, and neglect, among others (Gracey and King, 2009).

Just as the impacts of colonization are passed down from mother to child, to grandchild to great-grandchild, etc., embodied ELS experiences may be passed intergenerationally. Social and environmental conditions experienced by oocytes and embryos, infants, children, and adolescents, acting in concert with the experiences of their mothers and grandmothers, can lead to worse health outcomes for adults, culminating in premature mortality (Barker, 1995; Braveman and Gottlieb, 2014; Gluckman and Hanson, 2006; Thayer and Kuzawa, 2011).

Paper objectives and hypotheses

Epidemiologists and osteologists have long recognized that social and ELS experiences are written in our bones (Beatrice and Soler, 2016; Geber, 2014; Krieger and Davey Smith, 2004). Skeletal evidence for ELS included here includes porous cranial lesions (PCLs) and growth stunting/arrest. PCLs of the orbit (cribra orbitalia) and vault bones (porotic hyperostosis) typically form in children between six months and eight years of age (O'Donnell *et al.*, 2023; Watts, 2013), indicating health disturbances suffered by a child (Stuart-Macadam, 1985).

This paper examines the evidence for embodiment of social experiences through investigation of the relationships between SES, rural-urban residence areas, and race/ethnicity to respiratory disease, age at death, and skeletal manifestations of stress (growth stunting, growth arrest lines, PCLs).

Due to intersecting issues confronted by socioeconomically disadvantaged individuals and those from rural areas (Del Rio *et al.*, 2017; James *et al.*, 2017), the expectations are overlapping. Expectations are that individuals from lower SES/rural areas will die earlier (1) and have higher odds of skeletal changes associated with stress (PCLs of the orbit/vault, Harris lines

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(growth arrest), and growth stunting) (2). Some issues experienced by lower SES/rural residents are amplified in individuals from historically underrepresented groups.

Subjects and methods

Data include information from 780 individuals whose resident state was New Mexico; all individuals died in 2011–2019 between 0.5 and 20.9 years of age. All underwent postmortem computed tomography (PMCT) at the New Mexico Office of the Medical Investigator (OMI). This research includes decedent data and is not under the purview of IRB. Research requests for access to PMCT and associated reports were submitted and approved by the research/ethics board at OMI in 2019, 2020, and 2023. For greater detail, see O'Donnell *et al.* (2023); O'Donnell *et al.* (2020); O'Donnell *et al.* (2022). Table 1 provides variable descriptions.

Data on health conditions, race/ethnicity, and cadaver length were recorded from autopsy reports and associated paperwork. Also included are the percentage of families in poverty by ZIP Code Tabulation Area; the Rural-Urban Commuting Area Codes (RUCA) were used to determine the rurality of a place.

Limitations

A potential limitation relates to the study of the dead. Skeletal samples are not entirely representative of the living population (Wood *et al.*, 1992). At any time, there are more people at risk of becoming ill and dying than who actually died (Wood *et al.*, 1992). So, researchers who study the dead [as in this study] likely only see those individuals at greatest risk of illness or death (Vaupel *et al.*, 1979). Further, while sample bias may exist in *who* arrives at OMI, this sample is demographically representative of New Mexican children who die (O'Donnell and colleagues 2020; 2022).

Analytical methods

All analyses were done using Stata15. Multi-level mixed-effects regression (logistic and linear) was used to test for a random effect by ZIP code. Age at death was the dependent variable in linear regression, examining whether SDOH (e.g., housing type) and stress indicators (e.g., Harris lines) are associated with premature death. To further interrogate the relationships between skeletal indicators of stress and SDOH, logistic regressions were done using each indicator of stress as a dependent variable. Interaction terms for the per cent families in poverty variable [levels 0 (0–20% families in poverty) and 1 (>20% families in poverty)] and home type (trailer/mobile home, house, apartment) are included, alongside age and sex.

Predictive margins (probabilities) were estimated, and plots were used to examine interactions. Plots and tables for predicted margins provide all levels, including the base level (comparison category). Bases are as follows: race/ethnicity: Native American; rural/urban: metropolitan; housing type: house; SES: 0–20% poverty]. Sample sizes for some respiratory conditions are not large enough to be explored independently. Pneumonia, bronchitis, and asthma were examined separately. Following those analyses, respiratory conditions were pooled together. See Table 2 for descriptive statistics.

Results

Results of linear and logistic regression are provided in forest plots (Fig. 2). For full models, see Table 3; predictive margins are in Table 3 and Figs. 3–6. No random effects by ZIP code were noted (the likelihood ratio test was insignificant for all models, indicating no difference between

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Table 1. Descriptions of Each Variable used in Analysis

Variable Name	Description/Definition	How data was collected or determined
Sex	Sex categories include male (1) or female (0); this should not be taken to indicate social constructions of gender.	Autopsy report.
Age at Death	Age at death in months/years.	Autopsy report.
Race/Ethnicity	Race/ethnicity categories include Native American, Hispanic (which can include White Hispanic individuals), and non-Hispanic White	Race/ethnicity was reported by the field investigator.
Housing Type	Housing type categories include whether an individual lived in a house, trailer/mobile home, or apartment.	Autopsy reports and field investigator.
Per cent Families in Poverty	0–20% or >20%	The per cent of families in poverty by Zip Code Tabulation Area (Census Bureau, n.d.) was determined using estimated data from the U.S. Census Bureau's American Community Survey (ACS) for the years 2011 through 2019. These were five-year estimates with the last year of the period being matched with each individual's year of death.
Rural/Urban	Levels are metropolitan, micropolitan, small town, and rural.	Rural-Urban Commuting Area Codes (RUCA) were used to establish whether the ZIP code for each individual was a metropolitan, micropolitan, small town, or rural area (Cromartie, 2020). Developed through the U.S. Department of Agriculture's Economic Research Service (ERS), these codes were based on data from the 2010 Decennial Census and the 2006–2010 ACS. RUCA classifications are held constant throughout the decade.
Respiratory Disease	ICD-10 codes were used to classify respiratory conditions (asthma, COPD, chronic lung disease, or upper/lower respiratory tract infections (recurrent or at time of death).	Respiratory disease was recorded by one author from autopsy reports and field investigator notes.
Growth Stunting	Stunting is diagnosed when a child is more than two standard deviations below the mean (or less than the 3rd percentile) height for sex and age (Nwosu & Lee, 2008).	Growth stunting was determined using cadaver length, taken at autopsy, as an approximation for stature. Stunting was classified using the World Health Organization (WHO) (W.H.O, 2007, 2021) standards for individuals under two and the Centers for Disease Control's (CDC) criteria for individuals over two years.
Harris Lines (growth arrest)	Harris lines are transverse lines in the metaphyses of long bones visible in radiographs (Harris, 1931) (Fig. 1). They indicate incidents of temporary growth arrest during development (Harris, 1931; Primeau *et al.*, 2016).	Harris lines (tibia) were recorded from PMCT and radiography. Scoring followed Clark and Mack (1988), a Harris line was recorded as present if it expanded across at least 30% of the tibial shaft.
Cribra Orbitalia & Porotic Hyperostosis	These lesions give bone a sieve-like appearance (Brickley, 2018). (Fig. 1). The causes of PCLs are complex and poorly understood, but their presence indicates increased mortality risk (O'Donnell, 2019).	CO and PH were recorded by two authors from PMCT. PCL data were collected from PMCT, using *Horos,* a freeware DICOM viewer (Horos, 2019); PCLs were collected following methods developed by O'Donnell *et al.* (2023, see Fig. 1a–1d for additional images); O'Donnell *et al.* (2020). Orbit and vault lesions are scored as 'present' or 'absent' based on a range of criteria including whether there is diploic expansion coupled with pitting and/or porosity, or pitting/porosity in the absence of diploic expansion (Fig. 1a–1c).

Table 2. Descriptive Statistics for the Sample.

	Number	%
Sex		
Male	461	59.1
Female	319	40.9
Race/Ethnicity		
Native American	134	18.1
Hispanic	298	40.2
Non-Hispanic White	309	41.7
Housing type		
House	438	65.8
Trailer/Mobile Home	138	20.9
Apartment	87	13.2
Per cent families in poverty		
0–20%	520	66.7
>20%	260	33.3
Rural/Urban		
Metropolitan	461	63.3
Micropolitan	169	23.2
Small Town	38	5.2
Rural	60	8.2
Respiratory diseases (ICD-10)	147/610	24.1
Asthma	33/603	5.5
Pneumonia	80/603	13.3
Bronchitis	17/601	2.83
Bronchiolitis	6/601	1
Other	35/610	5.74
Growth stunting	53/767	6.91
Harris lines	111/746	14.9
Cribra orbitalia	217/751	28.9
Porotic hyperostosis	187/753	24.8

Notes: Health outcome variables have the number of individuals scorable for each condition, including the number of individuals with each condition.

ordinary logistic regression and mixed-effect logistic regression). So, all results presented are for ordinary logistic/linear regression.

Age at death

Rural/urban location does not significantly impact age at death. Trailers/mobile homes or apartments in higher-poverty areas are associated with older ages at death than in lower-poverty

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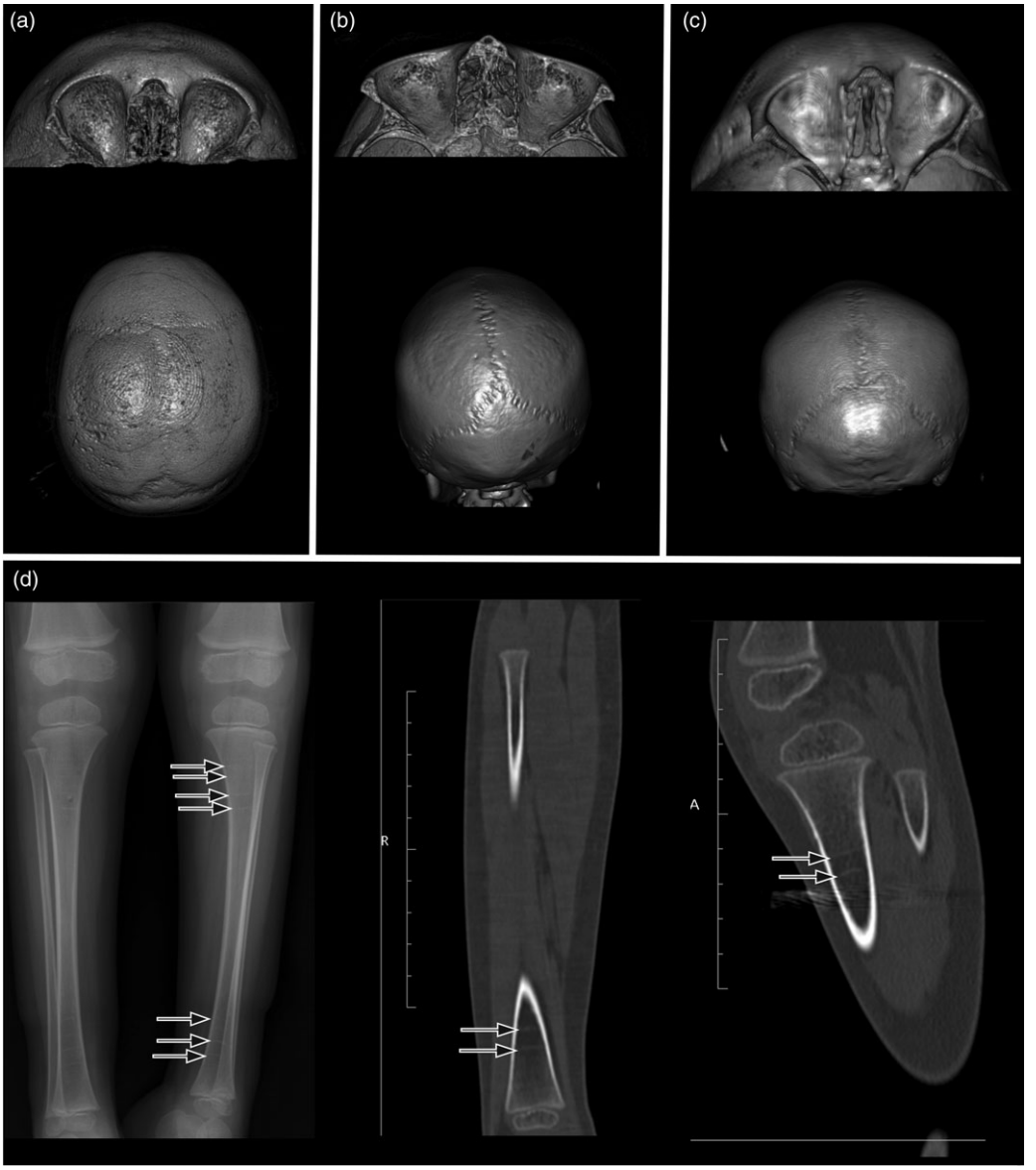

Figure 1. PMCT images of porous cranial lesions of the orbit (cribra orbitalia) and vault (porotic hyperostosis) and Harris lines. (a) Orbital and vault lesions present with marrow expansion; (b) Orbital and vault lesions present without expansion; (c) absence of PCLs; (d) Harris lines (marked with arrows) as observed in a single individual. The far-left image is a radiograph, and the others are from PMCT. Harris lines are observable in both radiographs and PMCT.

areas (Fig. 3a, Table 3, Model 1). On average, Native Americans live three fewer years than Hispanic or non-Hispanic White children (Figs. 2a and 3b; Table 3, Model 1).

Growth stunting and arrest (Harris lines)

Growth stunting is associated with higher odds of Harris lines. Hispanic individuals were more likely to be growth-stunted (Fig. 2b, Table 3, Model 2). Hispanic individuals who lived in trailers/mobile homes have a higher probability of growth stunting versus all other groups (Fig. 4b,

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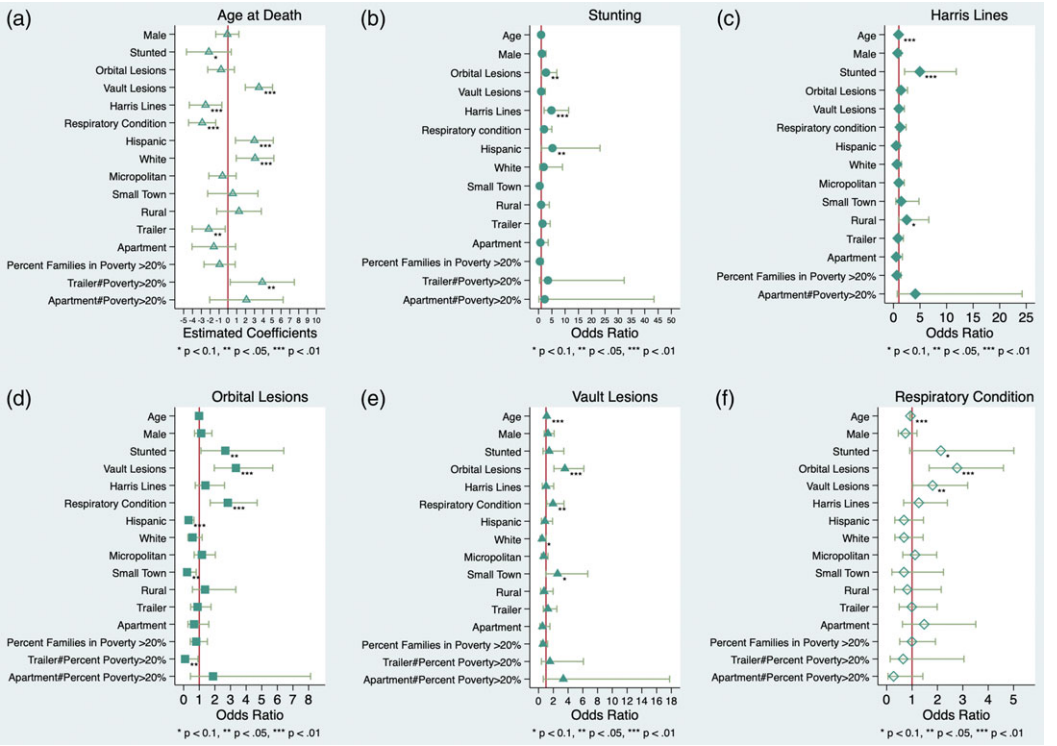

Figure 2. Forest plots of linear regression results (a) and logistic regression results (b–f) for each variable. Included are coefficient estimates (a) and odds ratios (b–f) and 95% confidence intervals. The vertical line is at 0 (a) and 1 (b–d).

Table 4). For other groups, housing type, rural-urban location, and percentage of families in poverty do not affect growth stunting (Fig, 4a and 4b, Table 3).

Native American, Hispanic, and White individuals have similar probabilities of having growth arrest (Fig. 2c). Housing type, rural-urban location, and per cent of families in poverty do not significantly affect the presence of Harris lines (Fig. 4c and 4d, Table 3).

Orbital and vault lesions

Native Americans have a higher probability (0.40) of having orbital lesions than Hispanic individuals (who have lower odds of orbital lesions, Fig. 2d), regardless of housing type (Fig. 5b, Table 4, Model 4). Housing type and per cent of families in poverty in the ZIP code alone do not explain the presence of PCLs (Tables 2 and 3, Models 4, 5). When interaction terms are included, individuals who live in trailers in areas with lower poverty have higher probabilities of having orbital lesions (Fig. 5a). These differences are not as pronounced for vault lesions (Fig. 5c and 5d).

Respiratory conditions

PCLs are associated with higher odds of respiratory conditions (Figs. 2d, 2e and 5a–5d, Table 3). Individuals who live in trailers/mobile homes and apartments in areas with relatively low poverty have increased probabilities of having respiratory conditions (Fig. 6a, Table 4, Model 6). Native Americans have a higher probability of having respiratory conditions, regardless of housing (Fig. 6b, Table 4), than Hispanic or White individuals.

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Table 3. Regression Results for Models 1–6.

Variables	Model 1 Age at death	Model 2 Stunted	Model 3 Harris lines	Model 4 Orbital Lesions	Model 5 Vault Lesions	Model 6 Respiratory
Age at death		0.955	**0.947*****	0.985	**1.101*****	**0.938*****
		(0.028)	**(0.02)**	(0.017)	**(0.024)**	**(0.017)**
Sex	−0.065	1.24	0.798	1.123	1.269	0.738
	(0.664)	(0.511)	(0.235)	(0.273)	(0.327)	(0.181)
Features of the skeleton						
Orbital lesions	−0.776	**2.79****	1.401		**3.571*****	**2.773*****
	(0.765)	**(1.28)**	(0.451)		**(0.987)**	**(0.716)**
Vault lesions	**3.506*****	0.994	1.01	**3.345*****		**1.809****
	(0.783)	(0.472)	(0.361)	**(0.912)**		**(0.524)**
Stunted	−2.152*		**4.959*****	**2.667****	**1.434**	2.133*
	(1.285)		**(2.196)**	**(1.194)**	**(0.637)**	(0.93)
Harris lines	**−2.523*****	**4.841*****		1.394	1.013	1.264
	(0.935)	**(2.099)**		(0.448)	(0.363)	(0.411)
Respiratory illness						
Respiratory condition	**−2.908*****	2.072	1.234	**2.828*****	**1.946****	
	(0.78)	(0.928)	(0.409)	**(0.735)**	**(0.567)**	
Race/ethnicity, base Native American						
Hispanic	**2.991*****	**5.232****	0.5	**0.312*****	0.858	0.68
	(1.088)	**(3.968)**	(0.23)	**(0.12)**	(0.345)	(0.262)
Non-Hispanic White	**3.066*****	1.943	0.632	0.571	0.464*	0.682
	(1.081)	(1.521)	(0.277)	(0.212)	(0.188)	(0.259)
Rural/urban, base metropolitan						
Micropolitan	−0.611	0.406	1.001	1.169	0.683	1.118
	(0.784)	(0.234)	(0.357)	(0.329)	(0.216)	(0.323)
Small town	0.551		1.458	**0.219****	2.571*	0.679
	(1.445)		(0.886)	**(0.145)**	(1.251)	(0.413)
Rural	1.258	0.974	2.489*	1.371	0.75	0.814
	(1.28)	(0.704)	(1.247)	(0.622)	(0.369)	(0.403)
Home type, base house						
Trailer	**−2.167****	1.501	0.825	0.889	1.256	0.989
	(0.949)	(0.814)	(0.34)	(0.307)	(0.435)	(0.352)
Apartment	−1.59	0.691	0.511	0.669	0.527	1.476
	(1.252)	(0.583)	(0.309)	(0.299)	(0.288)	(0.652)
Per cent families in poverty, base <20%						
Poverty >20%	−0.938	0.482	0.616	0.804	0.596	0.994

(*Continued*)

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Table 3. (*Continued*)

Variables	Model 1 Age at death	Model 2 Stunted	Model 3 Harris lines	Model 4 Orbital Lesions	Model 5 Vault Lesions	Model 6 Respiratory
	(0.896)	(0.313)	(0.259)	(0.261)	(0.218)	(0.335)
Per cent families in poverty##home type						
Trailer## poverty>20%	**3.886****	3.522		**0.091****	1.536	0.647
	(1.843)	(3.979)		**(0.107)**	(1.078)	(0.511)
Apartment## poverty>20%	2.092	2.294	4.111	1.88	3.357	0.273
	(2.11)	(3.443)	(3.72)	(1.404)	(2.854)	(0.23)
Intercept	10.853***	0.017***	0.466	0.549	0.064***	0.644
	(1.251)	(0.016)	(0.25)	(0.252)	(0.036)	(0.3)
Observations	441	416	417	441	441	441
R^2 or Pseudo R^2	0.159	0.198	0.115	0.164	0.13	0.132

Notes: Standard errors are in parentheses.
Model 1 is a Linear Regression with Age at Death as the Dependent Variable; Models 2–6 are Logistic Regressions with Odds Ratios Presented. Significant Values Are in Bold, Standard Errors are in Parentheses.
***$P < .01$,
**$P < .05$,
*$P < .1$

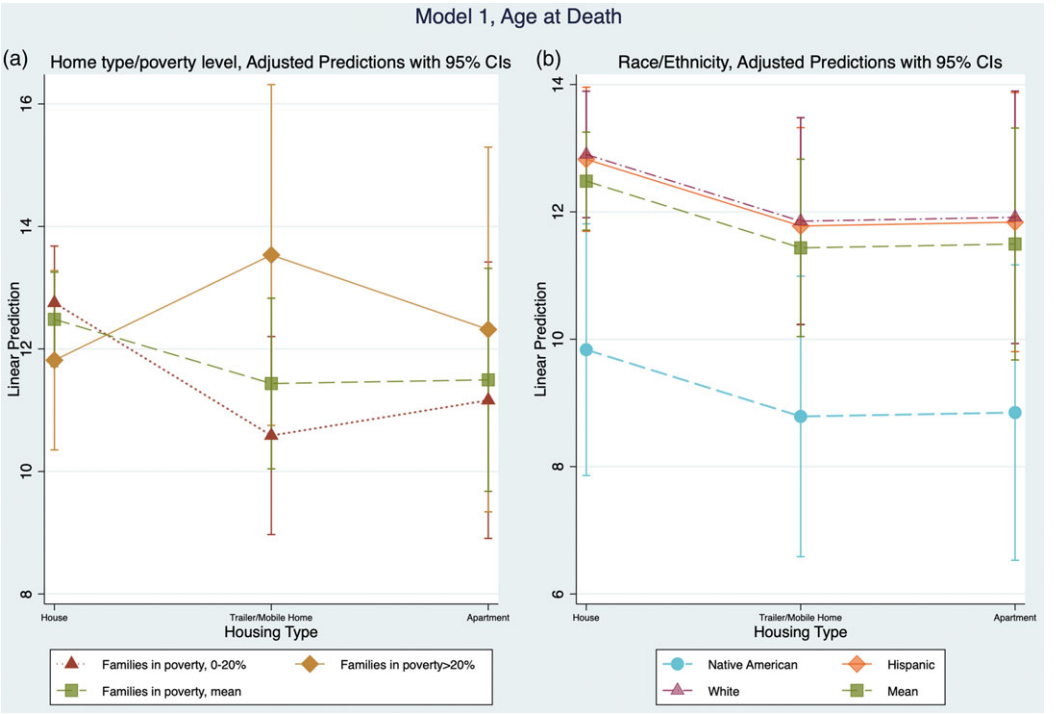

Figure 3. Margins plots for interaction terms from Table 4 (for age at death). The left plot shows predictive margins for housing type and poverty, and the right shows predictive margins for housing type and race/ethnicity.

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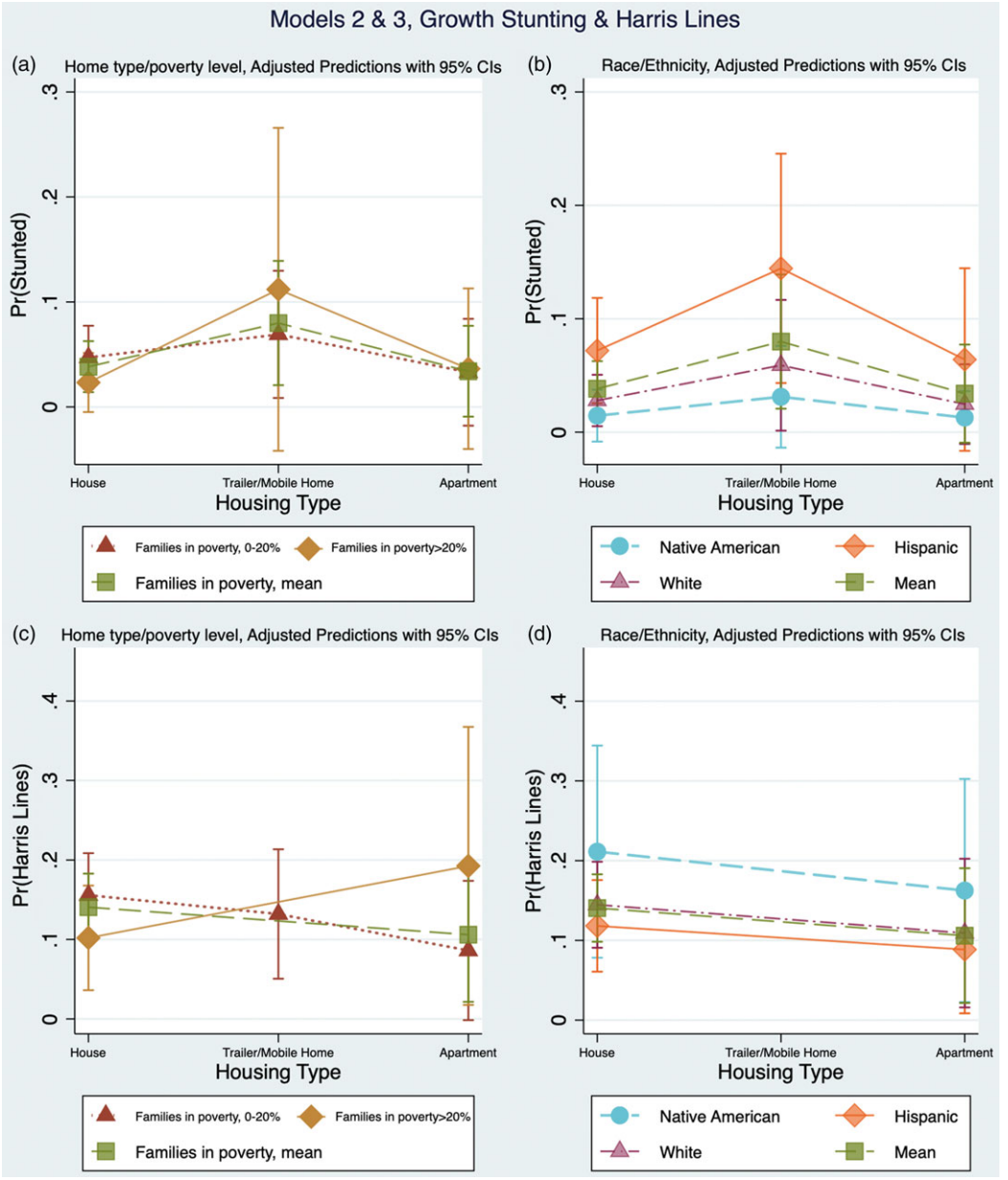

Figure 4. Margins plots for interaction terms from Table 4 for stunting [Model 2] (a, b) and Harris Lines [Model 3] (c, d). The left plots (a, c) show predictive margins for housing type and poverty, and the right plots (b, d) show predictive margins for housing type and race/ethnicity.

Discussion

The culture constructed by adults can and does impact the lives, health, and well-being of infants, children, and adolescents. Ill health is driven by complex interactions of genetic, epigenetic, behavioural, social, environmental, economic, and political forces (Schell, 1997; Waitzkin, 1981). Children's social and physical well-being is shaped by forces outside their control. The idea that social factors impact health is not new. Engels (1844) noted that inequity has consequences over the life course, 'Common observation shows how the sufferings of childhood are indelibly

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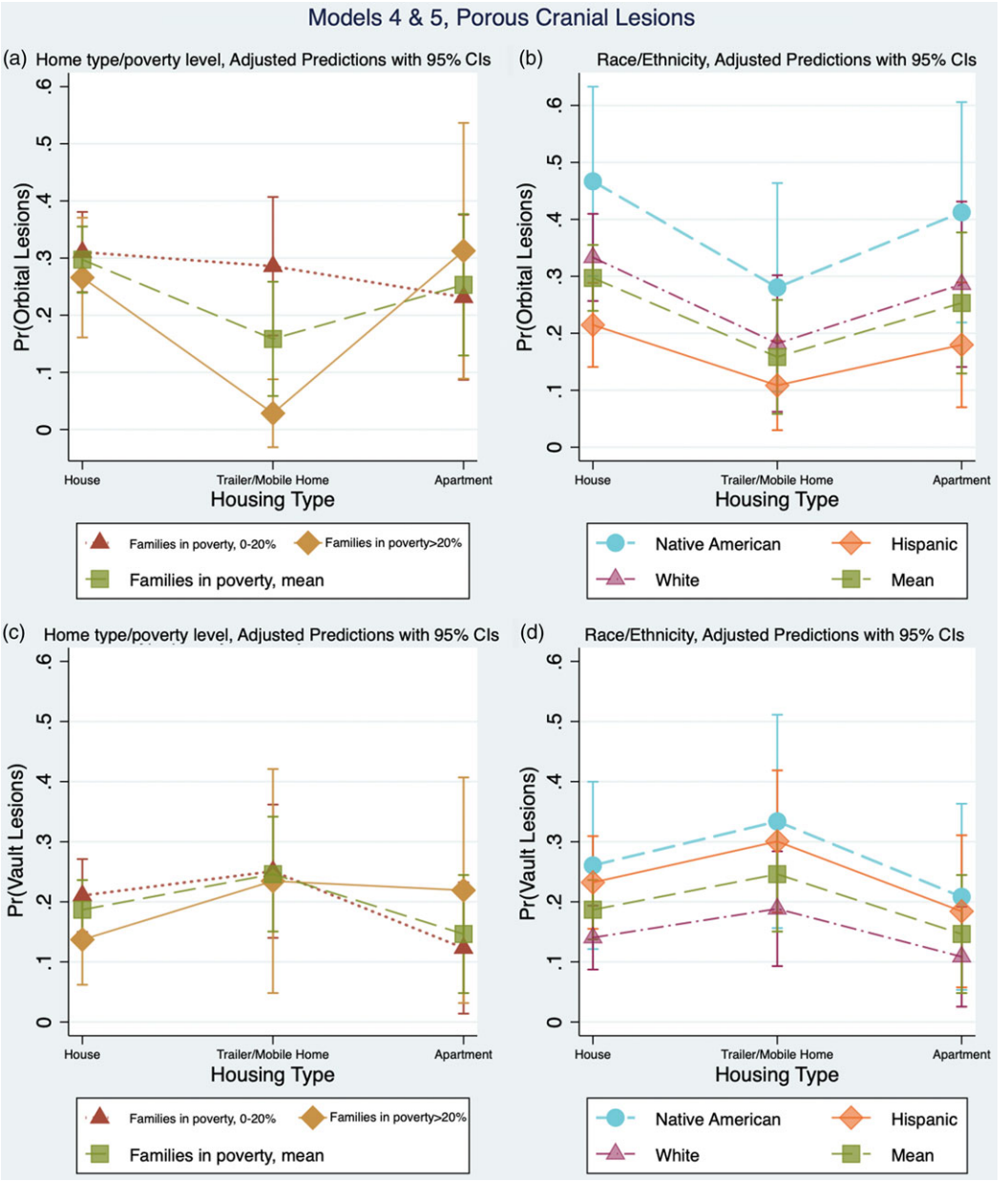

Figure 5. Margins plots for interaction terms from Table 4 for orbital lesions [Model 4] (a, b) and vault lesions [Model 5] (c, d). The left plots (a, c) show predictive margins for housing type and poverty, and the right plots (b, d) show predictive margins for housing type and race/ethnicity.

stamped on the adults' (Krieger and Davey Smith, 2004, p. 94). However, Engels assumed that children who suffer survive to adulthood, which is not always true. The sufferings of childhood are stamped onto children who die as well.

When we control for SES and rural/urban residence, disparities between Native American, Hispanic, and non-Hispanic White children persist (see also Braveman, 2012), meaning that differences between the groups cannot be explained by SES or rural life alone. This study finds that Hispanic children have higher odds of growth stunting. In contrast, Native American children

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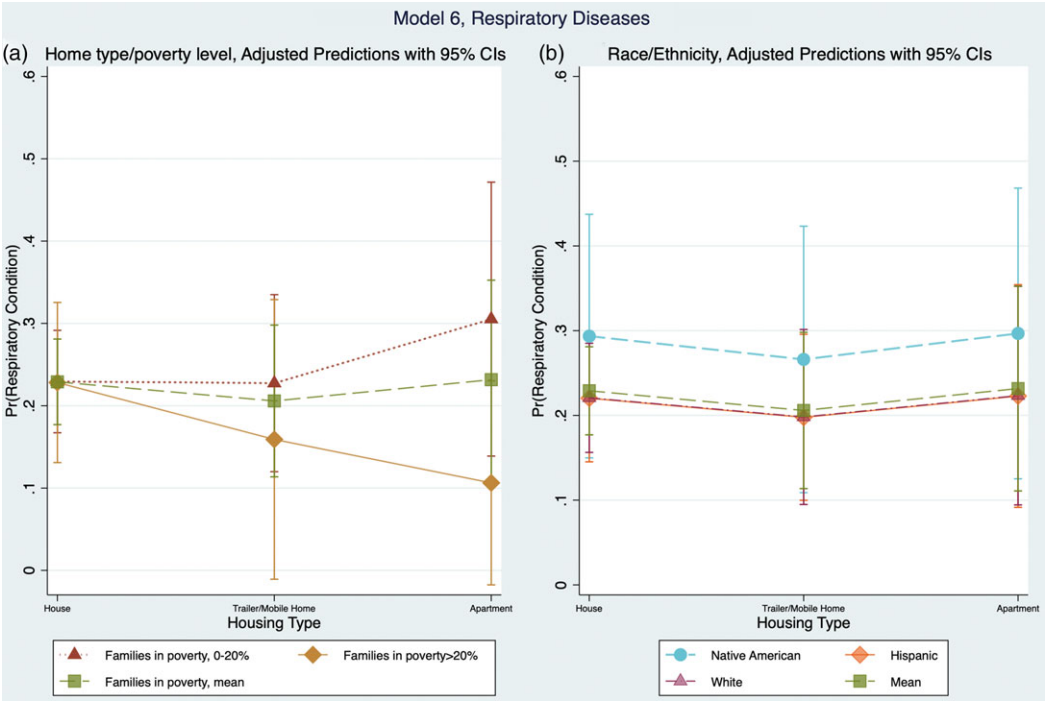

Figure 6. Margins plots for interaction terms from Table 4 for respiratory conditions [Model 6]. Left plot (a) shows predictive margins for housing type and poverty, and right (b) shows predictive margins for housing type and race/ethnicity.

have earlier ages at death, higher odds of respiratory diseases, and higher odds of PCLs than non-Hispanic White children. These are all indications of how marginalization, discrimination, and structural forces are embodied, even in lives that were cut short. Inequity literally 'gets under the skin'.

Age at death

Early death is the ultimate outcome of poor health (O'Donnell and Edgar, 2020), and people who die as children are among the least 'healthy'. They likely experienced increased risks for illness and early death than other individuals from their age and social cohorts (e.g., increased frailty) (Vaupel *et al.*, 1979; Wood *et al.*, 1992). The longevity of children in this study may be partly determined by the inheritance of frailty from their parents (e.g., Vaupel, 1988), but also by interactive factors, including intrauterine and ELS experiences, such as illness, poor quality environment, or low socioeconomic status. This study finds that Native American children die earlier than non-Hispanic White and Hispanic children. Other research has similar findings, but in adults, on average Native Americans die earlier [13 years (women) and 12 years (men)] than White individuals (Gorzig *et al.*, 2022).

Indigenous people experience high rates of infant and child mortality, low life expectancy at birth, malnutrition and growth retardation, high infectious disease burdens, and other health issues (Gracey and King, 2009). These problems may stem partly from the cascade effects of colonization: poverty, poor living conditions, lack of access to healthcare, governmental indifference, and neglect, among others (Gracey and King, 2009). Results of the present study demonstrate that SDOH have deleterious effects not just on adult health outcomes but also on childhood health outcomes.

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Table 4. Estimated Margins at Means for Interactions by Regression Model. Delta-Method Standard Errors Are in Italics, *P*-Values are Underlined, Significant Results Are in Bold. Base Levels: Race/ethnicity – Native American; Manner of Death – Natural; Rural-Urban – Metropolitan; Per cent Families in Poverty 0–20%; home Type – House. Age at Death Is the Mean Age at Death, All Others Are Probabilities. Age at Death (Model 1), Stunting (Model 2), Harris Lines (Model 3), Cribra Orbitalia (Model 4), Porotic Hyperostosis (Model 5), and Respiratory Conditions (Model 6).

	Model 1) Age at death	Model 2) Stunting	Model 3) Harris Lines*	Model 4) Orbital lesions	Model 5) Vault lesions	Model 6) Respiratory illness
Housing Type						
House	**12.48**	**0.04**	**0.13**	**0.29**	**0.19**	**0.23**
	0.39	***0.01***	***0.02***	***0.03***	***0.03***	***0.03***
	<.001	**0.002**	**<.001**	**<.001**	**<.001**	**<.001**
Trailer/Mobile Home	**11.44**	**0.08**	**0.1**	**0.16**	**0.25**	**0.21**
	0.71	***0.03***	***0.03***	***0.05***	***0.05***	***0.05***
	<.001	**0.01**	**<.001**	**<.001**	**<.001**	**<.001**
Apartment	**11.5**	0.03	**0.14**	**0.25**	**0.15**	**0.23**
	0.93	*0.02*	***0.04***	***0.06***	***0.05***	***0.06***
	<.001	0.12	**<.001**	**<.001**	**0.003**	**<.001**
Per cent Families in Poverty						
0–20%	**12.1**	**0.05**	**0.14**	**0.29**	**0.21**	**0.24**
	0.38	***0.01***	***0.02***	***0.03***	***0.03***	***0.03***
	<.001	**<.001**	**<.001**	**<.001**	**<.001**	**<.001**
>20%	**12.2**	**0.03**	**0.1**	**0.18**	**0.16**	**0.19**
	0.62	***0.02***	***0.03***	***0.05***	***0.04***	***0.04***
	<0.001	**0.03**	**<0.001**	**<0.001**	**<0.001**	**<0.001**
Interaction, Home type, and Per cent Families in Poverty						
House/0–20%	**12.8**	**0.05**	**0.15**	**0.31**	**0.21**	**0.23**
	0.47	***0.02***	***0.02***	***0.04***	***0.03***	***0.03***
	<.001	**0.002**	**<.001**	**<.001**	**<.001**	**<.001**
House/>20%	**11.8**	0.02	**0.1**	**0.27**	**0.14**	**0.23**
	0.75	*0.01*	***0.03***	***0.05***	***0.04***	***0.05***
	<.001	0.11	**<.001**	**<.001**	**<.001**	**<.001**
Trailer/Mobile Home/0–20%	**10.6**	**0.07**	**0.12**	**0.29**	**0.25**	**0.23**
	0.82	***0.03***	***0.04***	***0.06***	***0.06***	***0.05***
	<0.001	**0.04**	**<0.001**	**<0.001**	**<0.001**	**<0.001**
Trailer/Mobile Home/>20%	**13.5**	0.1	0.06	0.03	**0.23**	0.16
	1.42	*0.08*	*0.05*	*0.03*	***0.09***	*0.09*
	<.001	0.15	0.21	**<.001**	**0.01**	0.06
Apartment/0–20%	**11.2**	0.03	**0.11**	**0.23**	**0.12**	**0.31**
	1.42	*0.03*	***0.05***	***0.07***	***0.06***	***0.08***
	<0.001	0.2	**0.01**	**0.002**	**0.03**	**<0.001**

(*Continued*)

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Table 4. (*Continued*)

	Model 1) Age at death	Model 2) Stunting	Model 3) Harris Lines*	Model 4) Orbital lesions	Model 5) Vault lesions	Model 6) Respiratory illness
Apartment/>20%	**12.3**	0.04	**0.23**	**0.31**	**0.22**	0.11
	1.52	*0.04*	***0.09***	***0.11***	***0.09***	*0.06*
	<.001	0.35	**0.02**	**0.01**	**0.02**	0.09
Race/ethnicity						
Native American	**9.49**	0.02	**0.14**	**0.42**	**0.27**	**0.29**
	0.95	*0.01*	***0.05***	***0.08***	***0.07***	***0.07***
	<0.001	0.18	**<0.001**	**<0.001**	**<0.001**	**<0.001**
Hispanic	**12.5**	**0.08**	**0.1**	**0.18**	**0.24**	**0.22**
	0.52	***0.02***	***0.02***	***0.03***	***0.04***	***0.03***
	<0.001	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**<0.001**
White (non-Hispanic)	**12.6**	**0.03**	**0.16**	**0.3**	**0.14**	**0.22**
	0.48	***0.01***	***0.03***	***0.04***	***0.03***	***0.03***
	<0.001	**0.01**	**<0.001**	**<0.001**	**<0.001**	**<0.001**
Interaction, Race/ethnicity, and Housing type						
House-Native American	**9.84**	0.01	**0.11**	**0.47**	**0.26**	**0.29**
	1.01	*0.01*	***0.04***	***0.08***	***0.1***	***0.07***
	<0.001	0.21	**0.01**	**<0.001**	**<0.001**	**<0.001**
Trailer/Mobile-Native American	**8.79**	0.03	**0.12**	**0.28**	**0.33**	**0.27**
	1.12	*0.02*	***0.04***	***0.09***	***0.09***	***0.08***
	<0.001	0.2	**0.01**	**0.003**	**<0.001**	**0.001**
Apartment-Native American	**8.85**	0.01	**0.15**	**0.41**	**0.21**	**0.29**
	1.18	*0.01*	***0.06***	***0.09***	***0.08***	***0.09***
	<0.001	0.28	**0.01**	**<0.001**	**0.01**	**0.001**
House-Hispanic	**12.8**	**0.07**	**0.1**	**0.21**	**0.23**	**0.22**
	0.58	***0.02***	***0.02***	***0.04***	***0.04***	***0.04***
	<0.001	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**<0.001**
Trailer/Mobile-Hispanic	**12.9**	**0.14**	**0.08**	**0.11**	**0.3**	**0.19**
	0.85	***0.05***	***0.03***	***0.04***	***0.06***	***0.05***
	<0.001	**0.01**	**<0.001**	**0.01**	**<0.001**	**<0.001**
Apartment-Hispanic	**11.8**	0.06	**0.11**	**0.18**	**0.18**	**0.22**
	1.04	*0.04*	***0.04***	***0.06***	***0.06***	***0.07***
	<0.001	0.12	**0.01**	**0.001**	**0.004**	**0.001**
House-White	**12.9**	**0.03**	**0.16**	**0.33**	**0.14**	**0.22**
	0.51	***0.01***	***0.06***	***0.04***	***0.03***	***0.03***
	<0.001	**0.02**	**<0.001**	**<0.001**	**<0.001**	**<0.001**

(*Continued*)

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Table 4. (*Continued*)

	Model 1) Age at death	Model 2) Stunting	Model 3) Harris Lines*	Model 4) Orbital lesions	Model 5) Vault lesions	Model 6) Respiratory illness
Trailer/Mobile-White	**11.8**	**0.06**	**0.12**	**0.18**	**0.19**	**0.19**
	0.79	***0.03***	***0.04***	***0.06***	***0.05***	***0.05***
	<0.001	**0.05**	**0.01**	**0.003**	**<0.001**	**<0.001**
Apartment-White	**11.8**	0.02	**0.16**	**0.29**	**0.11**	**0.22**
	1.04	*0.02*	***0.05***	***0.07***	***0.04***	***0.07***
	<0.001	0.17	**<0.001**	**<0.001**	**0.01**	**0.001**
Figure	2a, 3	2b, 4	2c, 5	2d, 6a	2e, 6b	2f, 7

**Note:* Harris Lines margins were estimated for a regression that did not include respiratory illness, as small sample sizes made certain margins inestimable.

Respiratory illness and porous cranial lesions

While other studies indicate that living in trailer/mobile homes is associated with an increased likelihood of having respiratory conditions and increased morbidity (Gan *et al.*, 2017; Salo *et al.*, 2018), no impacts of housing type on respiratory illness were noted in the present analyses. Instead, results show that Native Americans have higher probabilities of having respiratory conditions, regardless of housing type.

PCLs are markers of ELS that manifest on the skeleton. The causes of PCLs are complex and poorly understood (Brickley, 2018), but recently, PCLs have been associated with respiratory infections (O'Donnell *et al.*, 2020). Not only do Native American children in this study have younger ages at death and higher odds of respiratory illness but they also have higher odds of PCLs. So, PCLs may mark the presence of infection in children and higher odds of infection for Native American children in this study.

The development of respiratory illness is driven by the interactions of multiple social and environmental factors, which are often compounded for individuals of lower SES (Thakur *et al.*, 2014). Poorer people and children have a lower ability to reduce their exposure to conditions that increase the likelihood of developing respiratory disease (Isaacs and Schroeder, 2004), partly because they have little control over altering their environments.

Native American children in this study may have experienced more exposures that could have impacted their risk of developing respiratory disease than Hispanic and non-Hispanic White children. Southwest Native Americans have higher morbidity and mortality from infectious respiratory diseases compared to other groups (Sutcliffe *et al.*, 2019). (Groom *et al.*, 2014). While disparities in respiratory illness rates between Native Americans and the general population have decreased over time, they still exist (Burki, 2021; Foote *et al.*, 2015; Groom *et al.*, 2014). Proposed reasons for the observed disparities include social and environmental factors: exposure to indoor smoke or mould, crowding in the household, poverty, and lack of household water security (Ali *et al.*, 2022; Deitz and Meehan, 2019, p. 2; Findling *et al.*, 2020; Foote *et al.*, 2015; Liu *et al.*, 2022; Seltenrich, 2012; Smit, 2022). These factors may also impact PCL formation processes.

The above results may be driven partly by environmental exposures in the household. First, housing shortages and poverty may lead to household overcrowding, a risk factor for infections and underlying health conditions (Kamis *et al.*, 2021; Krieger *et al.*, 2020; Simpson *et al.*, 1995).

Second, while many households on tribal lands use gas/electricity for heating and cooking, solid fuels (e.g., coal, wood) are still used for cooking or heating (Bunnell *et al.*, 2010; Hadeed *et al.*, 2021; Morris *et al.*, 1990). Rates of solid fuel use are highest in the Southwest United States, including New Mexico (Hadeed *et al.*, 2021). Burning solid fuels increases levels of particulate

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matter (e.g., $PM_{2.5}$, PM_{10}) (Hadeed *et al.*, 2021; Seltenrich, 2012). Exposure to PM is complex; for example, the quality of the wood stove impacts the amount of $PM_{2.5}$ pollution in a home (Walker *et al.*, 2021). A study of indoor PM_{10} concentrations and respiratory infections in children from the Navajo Nation found that homes heated with solid fuel (e.g., wood, coal) had higher PM_{10} than those heated through other means (Robin *et al.*, 1996). This result is repeated in other studies (Bunnell *et al.*, 2010; Hadeed *et al.*, 2021). In homes cooking with wood, children had increased risks of acute lower respiratory tract infections (including pneumonia and bronchitis) (Robin *et al.*, 1996). Other work implicates woodsmoke in increased paediatric risk of lower respiratory tract infections (Honicky *et al.*, 1985; Morris *et al.*, 1990).

Finally, many tract homes on tribal lands were built with funding from The Department of Housing and Urban Development (HUD) and the Bureau of Indian Affairs (BIA) between the 1950s and 1970s (Seltenrich, 2012). These homes were built according to federal rather than local or state codes, which may render them more prone to developing indoor mould (Seltenrich, 2012). Mould or dampness in the home is associated with childhood pneumonia (Dales *et al.*, 1991; Lu *et al.*, 2022).

Growth stunting & arrest

A population's average stature reflects interactions between nutrition, the physical environment, and exposure to disease and illness (Carson, 2011; Steckel, 1995). Growth stunting stems from varied causes, including inadequate nutrition (Iriart *et al.*, 2013; Iriart *et al.*, 2011), frequent infections in early life (Frongillo Jr., 1999), and intrauterine growth retardation (Dewey and Begum, 2011). Likewise, growth arrest lines (Harris lines), which indicate temporary halting of growth, are associated with nutrient deficiency (Geber, 2014; Harris, 1931), infectious diseases (Hewitt *et al.*, 1955), trauma (Kennedy *et al.*, 2014), and shorter life spans (Nowak and Piontek, 2002).

In this study, Hispanic children are more likely to be growth-stunted compared to non-Hispanic White children. Achieved stature is indicative of individual health and is determined not just by genetic predisposition but by environmental and social factors (Steckel, 1995, p. 1903). In developed countries, children who live in impoverished conditions are more likely to be stunted (Jansen and Hazebroek-Kampschreur, 1997). Short stature in adults is associated with poor health outcomes, including heart disease (Forsen *et al.*, 2000).

Hispanic children are reported to have a higher prevalence of stunting than non-Hispanic Whites in other works (Iriart *et al.*, 2013; Iriart *et al.*, 2011). Hispanic children likely suffer from malnutrition and obesity (the 'nutritional double burden'): they have high-calorie diets, which are low in necessary nutrients, which may, in turn, lead to growth stunting and obesity (Iriart *et al.*, 2013). In New Mexico (2017–2018), 33% of Hispanic children were obese, compared to 26% of Native American children and 24% of non-Hispanic White children (NMVoices, 2020). The results of the present study may reflect interacting factors including poverty and food insecurity. However, when confounding effects (SES, housing type, some health indicators) are included in analyses, they do not entirely 'erase' the impact of race/ethnicity on stunting. Following the conclusions made by Iriart and colleagues, we interpret this as evidence for inequities suffered by Hispanic children, especially when compared with non-Hispanic White children.

Housing type, per cent families in poverty

Housing is an indirect correlate of health and well-being, and housing type can be used as a proxy for SES (Juhn *et al.*, 2011; Shaw, 2004). Unlike permanent structures (houses), the value of trailers/mobile homes depreciates over time, and they have no equity value (Latimer and Woldoff, 2010; Twiss and Mueller, 2004). Overall, the results of this study show that individuals who lived in trailers/mobile homes had earlier ages at death than those who lived in other types of housing.

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Living in trailers/mobile homes is associated with increased morbidity and lack of access to necessary resources (e.g., reliable water services) (Gan *et al.*, 2017; Pierce and Jimenez, 2015; Salo *et al.*, 2018).

When we consider intersections between housing type and poverty levels, a different pattern emerges. While the results of this study indicate that individuals who live in trailers/mobile homes and apartments in relatively higher SES areas live fewer years than their counterparts who live in houses, this result does not hold for those who live in relatively poorer areas. People who live in poorer areas may have access to resources due to governmental outreach that does not exist in areas with lower poverty. In those areas, poor people may be further marginalized and experience the greatest restrictions to resource access. Another possibility is that people benefit from living near and around individuals of similar circumstances (Kirby, 2008, p. 344). If this is the case, the community may act as a buffer from negative external inputs.

Significance

This paper demonstrates how the complex interplay between social, cultural, environmental, and biological forces can impact children's health outcomes. By understanding how political, environmental, and social forces work in concert to create inequity and induce poor health, we can perhaps intervene before a premature death. Children who live in similar circumstances to those in this sample are alive now. They will either die or live into adulthood and suffer the effects of their adverse childhood experiences. The society within which we operate can buffer children from sickness and death, or it can induce ill health and poor outcomes (Braveman, 2012; Gravlee, 2009; Krieger, 2014). In the children in this sample, we see evidence for society's buffering capabilities and the harmful effects of systems not created for the individuals operating within them.

Conclusions

Evidence for inequity and the effects of the SDOH exists in deceased children from New Mexico. We find that Hispanic children are more likely to be growth-stunted than non-Hispanic White children. This may be related to inadequate nutrition stemming from inequities in resource access suffered by Hispanic children. Native American children die earlier and are more likely to have skeletal indicators of stress (orbital lesions) and respiratory infections than non-Hispanic White and Hispanic children. These results speak to the increased risks of morbidity and mortality faced by Native Americans in the United States. They also demonstrate that children *embody* ELS, a process which is likely induced by the interplay between various SDOH.

Children's health is shaped by factors that they cannot control. The children included in this study did not survive to adulthood, and they provide the most sobering example of the harm that social constructs (race/ethnicity, SES, and place) can inflict. While race is a social construct, it has real, tangible, and sometimes devastating biological consequences. Social constructs become biology (Gravlee, 2009; O'Donnell and Edgar, 2020), and illness and early mortality have social origins (Waitzkin, 1981). We can prevent early mortality for children and adults by improving health through the improvement of our social systems. One way to do this is to make systems equitable and resources more accessible. In the paraphrased words of Rudolf Virchow (1821–1902), to remedy physical ills, our social structures must be remodelled. It is time that we remedy our social structures.

Data availability statement. Because cases for some individuals included in this study are still open and active, data used in each analysis are available to *Bona fide* researchers only upon email request to the corresponding author. Some data will be presented in abstracted format only to ensure the anonymity of each individual in the sample.

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Competing interests. The authors have no conflicts of interest to report.

Ethical standard. We submitted proposals requesting to collect and analyse the data included here to the New Mexico Office of the Medical Investigator review board. All research requests were approved. We protect individual anonymity through the abstraction of some data.

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