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Article

The scars of the past? Childhood health and health differentials in later life

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ABSTRACT

This study estimates multilevel mixed effects models of three retrospective measures of childhood health – self-rated childhood health, exposure to parental smoking growing up, and missing school for 30 or more consecutive days due to a health event – on levels and changes in physical functioning at age 50 and beyond. Using data from 15 waves of the Household, Income and Labour Dynamics in Australia survey, the results show that variation in the level of later-life physical functioning is associated with childhood health. Poor childhood health however is not associated with the rate of physical functioning decline. Respondents who reported poor childhood health and were migrants to Australia from a non-English speaking country reported better physical functioning in later life, compared with non-Indigenous Australian-born respondents who reported poor childhood health. In contrast, women who reported poor self-rated childhood health reported worse physical functioning compared with men who reported poor self-rated childhood health. These findings are robust to the inclusion of a range of measures of childhood and adult characteristics and circumstances. These results suggest that Australia, with arguably a strong and supportive health care system as compared with the U.S., may mitigate the accumulation of disadvantages to those who reported poor childhood health. We note that though functional health differences due to childhood health are not exacerbated in later life, neither are they eliminated.

Introduction

A rich body of literature is emerging that reports associations between early childhood health and later-life health (Haas, 2006; Haas & Oi, 2018; Haas, Glymour & Berkman, 2011; Palloni, 2006). These studies address scientific questions about the distribution and determinants of health and disease, providing knowledge about whether poor childhood health is associated with health in later life, or with different health trajectories (Haas, 2008). Studies have also examined variations across sub-groups of the population providing a view into whether the effects of poor childhood health may differ by gender, socio-economic status, race and ethnicity (Haas & Rohlfsen, 2010; Haas, Krueger & Rohlfsen, 2012). Understanding these associations not only provides a better understanding of variations across adults in health outcomes, but also potentially highlights areas where early interventions may improve adult outcomes.

In this paper, we contribute to this literature in two ways: First we examine the links between childhood health and later-life physical functioning in Australia, including whether these associations vary across sub-groups. The majority of studies have focused on the United States (Haas, 2008), the United Kingdom (Jackson, 2015), or Europe (Haas & Oi, 2018) while the implications of poor childhood health on later-life physical functions might vary across contexts. Observed differences may be due to potential differences in the healthcare system, or through variations in other social institutions, such as schooling, and employment, which might differ in the way they respond to individuals based on their health status across the life course. Variations by sub-groups may also exist if poor childhood health intersects with other statuses, such as gender, ethnic, or migrant status, to shape health and physical functioning in later life (Haas & Rohlfsen, 2010; Haas, Krueger & Rohlfsen, 2012). Second, we include further measures of childhood health, of exposure to parental smoking and missing school for a month due to a health event growing up, in addition to self-reported childhood health. Self-reported childhood health has been used extensively in previous research (Haas, 2008; Haas, Krueger & Rohlfsen, 2012) but as has been noted by others, more work is needed to examine how results vary according to different childhood health measures (Blackwell, Hayward & Crimmins, 2001).

In addressing these questions, our findings provide knowledge about the long-term outcomes of poor childhood health. Doing so enables consideration of policies and interventions not only targeted at improving health, but also with the potential for reducing social disadvantage more broadly, as poor health and disability are important constraints to educational attainment, employment participation,

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relationships and family formation (Goodman, Joyce, & Smith, 2011; Haas, Glymour & Berkman, 2011).

Childhood health and health differentials in later life

There are three potential ways in which early life health might be associated with later-life health. The first, consistent with a cumulative disadvantage process (Ferraro & Shippee, 2009; Haas, 2008) suggests that poor childhood health is associated with conditions that accumulate over the life course and leads to lower levels of physical functioning in later life, as well as predicting faster functioning decline over time. A second possible scenario is that poor health in childhood is persistent, but not worsening, over the life course (Kelley-Moore & Ferraro, 2004), leading to variations in later-life health outcomes that mirror differences in childhood health. Under this scenario the health gap remains constant over time. A third scenario is an 'age as leveler' hypothesis (Beckett, 2000), whereby the effects of childhood health on later-life physical functioning diminish over time and the physical functioning of individuals with differing levels of childhood health converge.

A cumulative (dis)advantage process (DiPrete & Eirich, 2006; O'Rand & Hamil-Luker, 2005; Willson, Shuey, Elder & Glen, 2007) suggests that early advantage or disadvantage may compound over time leading to divergence in outcomes in later life (Dannefer, 2003). The concept has a long history (Merton, 1968) and has been used by stratification scholars to explain widening gaps in various outcomes including education, employment and wealth, as well as health (Ferraro, Schafer & Wilkinson, 2016; Haas, Glymour & Berkman, 2011). Concepts of accumulation of risk and cumulative (dis)advantage are evident in the large body of previous work documenting long-term persistence of health status over the life course, and negative impacts in adulthood in a range of in-utero and childhood health exposures (Ferraro & Shippee, 2009; Haas, 2008). In terms of cumulative processes leading to diverging health outcomes, the potential pathways linking childhood health with adult health may include differences in academic achievement (Jackson, 2015), school readiness (Kull & Coley, 2015), and educational and socioeconomic attainments in adulthood (Goodman, Joyce & Smith, 2011; Haas, 2006; Haas, Glymour & Berkman, 2011; Palloni, 2006). The idea is not just that early gaps in (dis)advantage are maintained over time, but rather that the effects compound over time leading to increased divergence in outcomes. For instance, one study by Haas (2008) found that while both childhood and adulthood characteristics influence the level of physical functioning in a sample of U.S. older adults, retrospective rating of childhood health, along with childhood family socioeconomic status was also associated with a faster rate of functional health declines over time.

Examinations of health disparities in later life have also found that the inequality related to early life conditions may remain stable over time (Ferraro & Farmer, 1996; Kelley-Moore & Ferraro, 2004; McDonough & Berglund, 2003). For example, McDonough and Berglund (2003), drawing on data from the Panel Study of Income Dynamics report that histories of poverty (captured between 1967–1982) were related to variations in levels of self-rated health in later years (captured between 1984–1996). They do not find however, that it was related to cumulative health disadvantage. In other words, the gap in self-rated health remained constant over time between those who reported a history of poverty versus those who did not report a history of poverty. Comparing disability trajectories of African Americans and White Americans, Kelley-Moore and Ferraro (2004) also found evidence of 'persistent inequality' in health, whereby the disability gap remained stable across the two groups over time.

Finally, in contrast to ideas from cumulative disadvantage or 'persistent inequality', researchers have also advanced the 'age as leveler' idea (Beckett, 2000; House, Kessleret & Herzogal, 1990; House et al., 1994; Kim & Durden, 2007). This approach suggests that rather than diverging or remaining constant, health differences between the advantaged and the disadvantaged may converge in later life. In her study examining how age modifies the association between education and health, Beckett (2000) showed that even accounting for mortality selection, there is convergence in educational differentials in health conditions and functional impairment in later life. One explanation offered was that although the advantaged may be able to postpone morbidity and health declines, the ageing process reduces such differentials over time, contributing to an equalization of health risks. Another explanation was that in the country under study, the U.S., the availability of universal healthcare for older adults may reduce differentials in access and quality of health care for adults at this life stage.

Observed associations between childhood health and later life health outcomes may also depend on the measure of childhood health. Given the availability of three childhood health measures in our dataset, we are able to consider associations between later-life physical functioning and 1) self-rated childhood health, 2) exposure to parental smoking and 3) prolonged absence from school as a child due to a health issue. While the association between self-rated childhood health and later-life physical functioning has been established (Haas, 2007; Haas, 2008), it is likely that exposure to parental smoking and prolonged absence from school as a child due to a health issue may be associated with a higher likelihood of experiencing a chronic condition in adulthood (Blackwell, Hayward & Crimmins, 2001; Dratva et al., 2016; Lovasi, Roux, Hoffman, Kawut, Jacobs & Barr, 2010; White, D'Aloisio, Nichols, DeRoo & Sandler, 2017), which may in turn result in lower physical functioning in later life (Seeman & Chen, 2002). In their study, Blackwell, Hayward and Crimmins (2001) used one of the same measures as the current study, whether respondents may have missed one month or more of school due to a health condition as a child. From respondents who reported in the affirmative, they further distinguished between different types of childhood illnesses and ailments, ranging from serious, life-threatening childhood diseases to skin conditions. The authors find that while any childhood illnesses and ailments were related to higher odds of reporting having cancer, cardiovascular conditions, lung disease, and arthritis/rheumatism, the strongest association was found for childhood infectious diseases. In a similar vein, exposure to parental smoking as a child may result in lower physical functioning in later-life through adulthood chronic conditions. Exposure to tobacco smoke in childhood has been linked to early emphysema (Lovasi et al., 2010), adult asthma and respiratory symptoms (Skorge, Eagan, Eide, Gulsvik & Bakke, 2005) lung function decline in midlife (Dratva et al., 2016), as well as risks of breast cancer (White et al., 2017). In turn, many chronic conditions are linked to poorer physical functioning (Seeman & Chen, 2002).

Arguably, the association between childhood health and later-life physical functioning may also vary by country context. Most other studies have focused on the United States (Haas, 2008), the United Kingdom (Jackson, 2015), or Europe (Haas & Oi, 2018). Given differences in a number of country-level factors however, including in healthcare systems and health care population coverage, it is not clear that earlier findings apply to the Australian context. The availability of universal publically-funded healthcare in Australia is in sharp contrast with the U.S., where no such policy exists (Blendon, Schoen, DesRoches, Osborn, Scoles & Zapert, 2002). This social safety net for adults may ameliorate ongoing accumulation of health disadvantages of individuals who experienced poor childhood health.

Variations by gender and ethnic/migrant status

It is also important to consider how the association between childhood health and later life physical functioning varies across social groups. For example, existing research has established that health trajectories vary by gender, income, educational attainment, ethnicity and race (Maddox & Clark, 1992). Maddox and Clark (1992) showed variations in functional impairment trajectories drawing on data from the U.S., with levels of functional impairment higher for men than women and also accelerating at a faster pace for men than women over time.

Impairment was also higher for those with less education and lower income, though differences in impairment over time were only observed by income status. Drawing on the Health and Retirement Study in the U.S., Montez and Hayward (2011) found that non-Hispanic white women who reported poor childhood health had significantly increased mortality, though this association was not observed for non-Hispanic white men. The authors reported that a large portion of this association was explained by adult educational attainment, stating that it may be possible that while their sample of white men had more adulthood opportunities to compensate for poor childhood health (such as through achieving higher educational attainment), comparable compensatory pathways were less available for white women. Studies examining racial/ethnic and nativity health disparities in later life in the U.S. have also often found that the disparities can be explained through health and socioeconomic disadvantages of their respondents in early life and adulthood (Haas, Krueger & Rohlfsen, 2012; Haas & Rohlfsen, 2010).

Given likely differences in experiences of racial/ethnic minorities and migrants in Australia compared to the U.S., it is unclear whether findings from the U.S. translate to Australia. While stark health disparities exist between native-born Indigenous and non-Indigenous Australians (Marmot, 2005), about three in ten residents in Australia are born overseas (Australia Bureau of Statistics, 2016a). Similar to findings from the U.S., studies of Australian migrants report a 'healthy migrant effect' (Hamilton 2015), with the health of migrants upon arrival better on average than that of the native-born. The categorization of migrant groups in Australia in academic studies and in data collection is also different from the U.S., focusing on whether the source country is primarily English speaking or non-English speaking (Biddle, Kennedy & McDonald, 2007; Jatrana, Pasupuleti, & Richardson, 2014). Studies find that immigrants from non-English speaking countries have relatively better health than their counterparts from English-speaking countries upon arrival (Biddle, Kennedy & McDonald, 2007). Immigrants who arrive in Australia as children (before age 14) also do not have different health profiles as immigrants who arrive as adults (Biddle, Kennedy & McDonald, 2007). It has also been found however that the health advantages of immigrants to Australia declines with time of residence in the country (Jatrana, Pasupuleti, & Richardson, 2014).

While health disparities may exist across sub-groups, it is less clear whether the association between childhood health and later-life physical functions varies by gender, migrant status, or Indigenous status in Australia. The prevalence of limitations differs by gender and age. A survey by the Australian Bureau of Statistics finds that although on average the prevalence of limitations are similar for men and women, with 18.6% of women and 18% of men reporting a disability in 2015, gender differences are observed at different age groups (Australian Bureau of Statistics, 2016b). For example, the most pronounced difference between men and women was at older ages for profound and severe limitations, with 40% of women aged 75 and over reporting such limitations versus 25% of older men. In contrast, higher proportions of males between the ages of 5 and 14 reported a disability, at 12%, compared to females, at 7%. Variations also exist by country of birth, with 18.6% of Australian-born persons reporting a disability, compared to 21% for migrants from an English speaking country and 15.8% for migrants from non-English speaking countries (Australian Bureau of Statistics, 2016a).

Moderation effects by gender and ethnic/migrant status might suggest that social institutions, including the health safety net for adults in Australia might vary in its treatment of sub-groups of people with poor childhood health. It could also indicate variation in relative access to compensatory resources (such as education) in adulthood and later life. Another reason for any observed moderation effects may be variations in health-seeking behaviours across sub-groups of individuals with poor childhood health, resulting in differences in later-life physical functions. This paper therefore investigates whether the association between childhood health and later-life functional health is moderated by gender or migrant/ethnic status in the Australian context.

Research questions

This study uses high quality longitudinal data to examine the associations between childhood health and later-life physical functioning, as well as specifying and testing two potential moderators that might explain variation in any observed associations. By investigating up to 15 waves of data, we are able to examine both the association between early life health and physical functioning at age 50 and over, and the association between early life health and trajectories of physical functioning in later life.

In sum, we address three main research questions:

1) Is there an association between childhood health and the level of physical functioning at age 50 and over?
2) Is childhood health associated with the rate of change in physical functioning over time, at age 50 and above?
3) Are the observed associations between childhood health and physical functioning at age 50 and over moderated by gender and/or ethnic/migrant status?

Data

This study examines data from waves 1 to 15 (year 2001 to 2015) of the Household, Income and Labour Dynamics in Australia (HILDA) Survey. The HILDA Survey is an ongoing panel study that started in 2001 with a representative sample of Australian households at the first year of data collection. Surveys were collected from individuals aged 15 and over living in the same households. The sample is designed to be representative of the non-institutionalized resident population of Australia with some minor exceptions, including very remote areas and diplomatic personnel of other nations. Data collection for the HILDA Survey combines a self-complete questionnaire and computer-assisted face-to-face interviews. The study aimed to capture a wide range of information on economic and personal well-being, labour market dynamics and family life. It also contains a number of questions asking respondents to recall retrospectively their childhood conditions, which are fielded at waves 9 and 13. Response rates are generally very high, exceeding 90% since wave 3 and 95% since wave 8. Among those who complete the main interview, approximately 90% return a self-completion questionnaire, which includes the health outcome that we consider here. For more information on the study, including a detailed description of the sample design and following rules, please see Watson and Wooden (2007).

Given the focus of this paper on later-life physical functioning, observations where the respondent was under 50 years of age were excluded. Due to the data collection window for the HILDA study and the age restriction, respondents included in the analysis were born between 1902 and 1965. The retrospective childhood health measures are collected at either waves 9 and 13 and are missing for around 25% of cases. This is primarily due to failure to interview at either of these waves, as approximately 99.6–99.8% of interviewees at these waves provide valid responses to the childhood health questions. Previous research on attrition in the HILDA data suggests that successful interviews are more likely among those who are older, Australian born, not suffering from serious long term health conditions, more highly educated, working shorter hours, and either separated or widowed (Watson & Wooden, 2009). Other variables either have no missing data where the information is collected through the main interview, or roughly 9% in cases where data is collected through the self-complete component of HILDA. To accommodate missing data, we used multiple imputation by chained estimates (MICE) (Little & Rubin, 2002; van Buuren, 2007; White, Royston, & Wood, 2011) with $m = 50$ imputed datasets. Estimates based on multiple imputation rest on the assumption that data are 'missing at random' (MAR), meaning that *conditional on the variables included in the imputation model*, the probability of missingness is

assumed to be unrelated to other factors that may bias the estimates of interest (Little & Rubin, 2002). The MAR assumption cannot be tested empirically, but it can be made more credible by building a suitably rich imputation model. We therefore included all person-characteristics that have previously been found to predict missingness, as well as all variables appearing in the final analysis models in the imputation model. The final analytic sample is a subset of respondents at age 50 and above, of 11,051 unique individuals, and 82,103 person-waves (an average of 7.4 complete waves of data per person).

Measures

Dependent variable

The dependent variable comes from the physical functioning subscale of the SF36-Health Survey (Ware & Sherborne, 1992). The SF-36 has been widely validated and shown to produce valid and reliable results. This scale is comprised of ten questions, such as "Does your health limit you in the following activities? And if so, how much?….(i) Vigorous activities, such as running, lifting heaving objects, participating in strenuous sports, (ii) Moderate activities, such as moving a table, pushing a vacuum cleaner, bowling or playing golf, (iii) Lifting or carrying groceries…etc." Possible response options are: (1) "Yes, limited a lot", (2) "Yes, limited a little", and (3) "No, not limited at all". In line with standard scoring (Ware, Kosinski, & Gandek, 2000), responses are summed across the items and scaled to run from 0 (representing the poorest health) to 100 (best health).

Independent variables

There are three primary independent variables used to measure childhood health. *Self-rated childhood health* comes from responses to the question: "These next questions ask about your health during childhood that is, before you turned 15 years of age. How would you describe your health during that time? Was it excellent, very good, good, fair or poor?" Before analysis we standardize values of this variable to have mean 0 and standard deviation 1.[1] *Exposure to parental smoking* comes from responses to the question "Were any of your parents or guardians smokers at any stage of your childhood?" *Missed school due to health problems* comes from responses to the question "When you were growing up, did you ever miss a month of school because of a health problem? That is, were you absent from school for 30 or more consecutive days?" These measures represent different aspects of childhood health, as reflected in the correlations between them. In particular, parental smoking is effectively uncorrelated with missing school due to poor health (r = 0.03) and self-rated childhood health (r = 0.02), while there is a moderate correlation between self-rated childhood health and missing school (r = 0.33).

Control variables include gender, age, birth cohort (in 5 year intervals), number of siblings, whether the respondent was the oldest sibling, ethnic-migrant status (Australian born, not Indigenous; Indigenous (Aboriginal and Torres Strait Islander peoples, ATSI); migrant, from an English-speaking country; migrant, from a non-English-speaking country), whether the respondent was living with both biological parents at age 14, whether the respondent's father was ever unemployed for 6 months or longer when the respondent was a child, and father's occupational prestige in childhood. Thus, the models adjust for a range of demographic factors and socio-economic position in childhood that may confound the relationships between childhood health and later-life health.

Measures of adult socio-economic status, marital status, social support, and health behaviours are used to investigate whether direct effects of childhood health on later-life physical functioning persist net of adult risk factors. Variables in this group include the respondent's smoking history (never smoked; former smoker; current smoker), amount of alcohol consumed on a typical drinking occasion (never consumed alcohol; no current alcohol consumption; 1–2 standard drinks; 3+ standard drinks), frequency of moderate or intense physical activity (not at all; less than once a week; 1 to 2 times a week; 3 times a week; more than 3 times a week; every day), perceived social support (scale comprised of 10 items; e.g. 'I often feel very lonely'; 'There is someone who can always cheer me up when I'm down'), relationship status (married; in a de facto relationship; divorced, separated or widowed; single, never married), highest level of education (below school Year 12, school Year 12, professional qualification, degree or higher), age first left school, employment status (employed, unemployed, not in the labour force), and the respondent's subjective financial prosperity ('Very poor' to 'Prosperous').

Analytic method

Physical functioning is modelled using growth models, with the repeated observations nested within individuals over time (Singer & Willet, 2003). Substantively, our modelling strategy enables investigation of how childhood health is associated with the individual's average level of physical functioning, and also their rate of change in physical functioning over time. The models are specified as follows:

$$PF_{it} = \pi_{0i} + \pi_{1i}(AGE-50)_{it} + \boldsymbol{\beta X_{it}} + e_{it} \quad (1)$$

With level 2 equations:

$$\pi_{0i} = \gamma_{00} + \boldsymbol{\gamma_0 Z}_{0i} + \zeta_{0i} \quad (2)$$

$$\pi_{1i} = \gamma_{10} + \boldsymbol{\gamma_1 Z}_{1i} + \zeta_{1i} \quad (3)$$

In these equations, PF_{it} is the observed value of physical functioning for individual i at time t. Each individual's trajectory of physical functioning is defined by a random intercept (π_{0i}) and yearly rate of change (π_{1i}). Because ($AGE - 50$) is used in place of raw age, π_{0i} is interpretable as the expected value of PF for a particular person when aged 50 (bearing in mind that this may fall outside the range of the observed data in many cases). The individual specific random intercepts and rates of change may depend in turn on person-level covariates $\boldsymbol{Z}_{0i}$ and $\boldsymbol{Z}_{1i}$ with fixed parameters $\boldsymbol{\gamma_0}$ and $\boldsymbol{\gamma_1}$. Time-varying covariates $\boldsymbol{X_{it}}$ are also included in the modelling. Level 1 (e_{it}) and 2 (ζ_{0i}, ζ_{1i}) residuals are assumed normally distributed with mean zero. In analyses reported here, an unstructured covariance structure is used for the random effects, permitting the random intercept and random rate of change to be correlated. The key independent variables for analysis (child health) are person-level variables, and therefore enter into $\boldsymbol{Z_0}$ and $\boldsymbol{Z_1}$.

Analysis proceeds by fitting a series of models that vary the predictors included in $\boldsymbol{X}$, $\boldsymbol{Z_0}$, and $\boldsymbol{Z_1}$. Initial models include only childhood health in the intercept equation, allowing the average level of physical functioning in later life to depend on childhood health. Subsequent models extend upon this by making the rate of change in physical functioning depend on childhood health, adding controls for demographics and early life family and socio-economic status, and finally adding adult risk factors. In short, our modelling strategy entails estimating 'raw' associations between early life health and later-life physical functioning, adjusting for measured confounders of these relationships, and investigating whether significant 'direct' effects of childhood health on adult health persist net of adult risk factors.

[1] Because standardization occurs prior to imputation, values reported in the descriptive table deviate slightly from these values. Prior to standardization, the childhood SRH measure ranges from 1 (Excellent) to 5 (Poor), with mean 1.68 and standard deviation 0.96. Corresponding non-standardized values for other continuous variables in the analysis are mean 40.63, SD 21.08, range 0–100 for father's occupational status, mean 5.39, SD 1.01, range 1–7 for perceived social support, and mean 3.84, SD 0.78, range 1–6 for subjective financial prosperity.

Results

Descriptive statistics for the analytic sample are presented in Appendix Table A1. Time-invariant measures are summarized at the person level, while time-varying measures are summarized in terms of person-years. Seventy percent of respondents reported that at least one of their parents smoked at some stage during their childhood, while 12% had missed a month of school consecutively due to poor health. Around two-thirds of respondents were born in Australia and were non-Indigenous, the majority were currently married and had children, and about half had ever smoked cigarettes. Slightly more than half the sample observations (56.4%) corresponded to periods when the respondent was not in the labour force. The most common educational category was less than completed secondary school (41.9%), followed by vocational qualifications (30.8%) and university degree (19.1%).

The patterning of our childhood health measures by background demographic and early life factors is presented in Appendix Table B1. For the purposes of this descriptive analysis only, we use quintiles of fathers' occupational status and dichotomize the self-reported childhood health measure so that 'excellent' 'very good', and 'good' are combined in one category while 'fair', or 'poor' are combined in the other. Depending on the particular indicator of childhood health, a number of distinct differences emerge, although it is notable that parent smoking and self-rated health are associated with a wider range of background characteristics, whereas missing school was significantly related only to ethno-migrant background. First, migrants from a non-English speaking country are less likely to have missed a month of school, and both Indigenous and non-English speaking background respondents have higher rates of exposure to parental smoking. Despite this, there are only minimal differences in self-reported childhood health on the basis of the respondent's ethno-migrant background. Both parent smoking and self-reported childhood health show clear socio-economic gradients with father's occupational status and the respondent's own educational attainment.

Table 1 shows the main results for our analysis of the associations between childhood health and physical functioning in later life. Full results including parameter estimates for the control variables are available on request from the authors. Models 1 to 6 deal with one indicator of childhood health, either with or without the interaction between the measure of childhood health and age. Models 7 and 8 include all three indicators of childhood health status simultaneously, and 9 and 10 extend the results by controlling for early life factors and mid-life factors respectively. We report non-standardized coefficients in all cases.

Model 1 estimates the association between parent smoking and physical functioning, controlling only for age in years. Age is zero-centred at 50, meaning that the constant may be interpreted as the expected value of physical functioning for a 50 year old individual whose parents did not smoke. The parameter estimates show that physical functioning is poorer for older respondents (β = -0.95, $p < 0.001$) and that respondents whose parents smoked when they were children had poorer health (β = -1.65, $p < 0.01$).

Model 2 adds the interaction between age and parent smoking status. Including the interaction changes the interpretation of the parameters slightly – in particular, the 'main effect' of parent smoking now represents the estimated health deficit when respondents are aged 50. In comparison to model 1, the results show that the main effect of parent smoking is now non-significant (β = -1.26, n.s). The interaction between parent smoking and age is also non-significant (β = -0.04, n.s.). The size of the health gap related to parent smoking is therefore

Table 1
Random coefficient growth model estimates of the relationship between child health and later life physical functioning.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Age	-0.95***	-0.93***	-0.95***	-0.95***	-0.95***	-0.95***	-0.95***	-0.93***	-0.90***	-0.70***
	(0.02)	(0.03)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.03)	(0.04)	(0.03)
Parent smoking (PS)	-1.65**	-1.26#					-1.52**	-1.11#	-0.67	-0.53
	(0.53)	(0.66)					(0.53)	(0.65)	(0.64)	(0.56)
Missed school poor health (MS)			-5.55***	-5.65***			-3.44***	-3.87***	-4.15***	-3.28***
			(0.81)	(0.98)			(0.87)	(1.05)	(1.03)	(0.90)
Self-report child health (SRH)					-2.39***	-2.19***	-2.01***	-1.76***	-1.33***	-0.80**
					(0.25)	(0.31)	(0.27)	(0.33)	(0.32)	(0.28)
PS * Age		-0.04						-0.04	-0.07	-0.05
		(0.04)						(0.04)	(0.04)	(0.04)
MS * Age				0.01				0.04	0.04	0.01
				(0.06)				(0.06)	(0.06)	(0.06)
SRH * Age						-0.02		-0.02	-0.02	-0.03
						(0.02)		(0.02)	(0.02)	(0.02)
Constant	86.10***	85.83***	85.59***	85.61***	84.87***	84.88***	86.36***	86.13***	92.86***	82.91***
	(0.47)	(0.54)	(029)	(0.30)	(0.28)	(0.28)	(0.48)	(0.55)	(1.08)	(1.17)
Variance components										
Std. dev (π_{0i})	21.61	21.61	21.51	21.51	21.47	21.47	21.42	21.42	20.68	17.27
	(0.28)	(0.28)	(0.28)	(0.28)	(0.27)	(0.27)	(0.27)	(0.27)	(0.27)	(0.25)
Std. dev (π_{1i})	1.07	1.07	1.07	1.07	1.07	1.07	1.07	1.07	1.05	0.89
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
Corr(π_{1i}, π_{1i})	-0.50	-0.49	-0.49	-0.49	-0.50	-0.50	-0.50	-0.50	-0.50	-0.49
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.02)
Early life controls	N	N	N	N	N	N	N	N	Y	Y
Mid-life controls	N	N	N	N	N	N	N	N	N	Y

N (persons = 11,051); N (person-years = 82,103). Non-standardized coefficients. Standard errors in parentheses.
*** $p < 0.001$
** $p < 0.01$
# $p < 0.1$
* $p < 0.05$

Table 2
Moderators of the relationships between child health and adult health.

	Ethno-migrant background interactions			Gender interactions		
Age	-0.89***	-0.94***	-0.94***	-0.89***	-0.94***	-0.94***
Parent smoking (PS)	-1.59*			-0.55		
Missed school poor health (MS)		-6.81***			-4.45***	
Self-report child health (SRH)			-2.27***			-0.91*
Ethno-migrant background						
1st Gen. immigrant, English speaking countries (ESB)	0.51	1.22	1.68**	1.83**	1.70**	1.70**
1st Gen. immigrant, non-English speaking countries (NESB)	-5.98***	-4.36***	-3.52***	-3.71***	-3.80***	-3.46***
Indigenous (ATSI)	-8.10	-8.75***	-7.91***	-7.75***	-7.73***	-7.89***
ESB * PS	1.74					
NESB * PS	3.57**					
ATSI * PS	0.52					
ESB * MS		3.71#				
NESB * MS		5.87**				
ATSI * MS		7.00				
ESB * SRH			0.94			
NESB * SRH			2.14**			
ATSI * SRH			2.10			
Sex						
Female	-2.46***	-2.38***	-2.37***	-2.21**	-2.16***	-2.36***
Female * PS				-0.39		
Female * MS					-1.82	
Female * SRH						-1.54***
Constant	93.07***	92.88***	91.85***	92.35***	92.60***	91.90***
Variance components						
Std. dev (π_{0i})	20.84	20.72	20.71	20.84	20.74	20.70
	(0.27)	(0.27)	(0.27)	(0.27)	(0.27)	(0.27)
Std. dev (π_{1i})	1.05	1.05	1.05	1.05	1.05	1.05
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
Corr(π_{1i}, π_{1i})	-0.50	-0.50	-0.50	-0.50	-0.50	-0.50
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Early life controls	Y	Y	Y	Y	Y	Y
Mid-life controls	N	N	N	N	N	N

N (persons = 11,051); N (person-years = 82,103). Non-standardized coefficients.
* $p < 0.05$.
** $p < 0.01$.
*** $p < 0.001$.

constant with respondent age.

Models 3 and 4 repeat this process, substituting whether the respondent ever missed a month of school due to poor health in place of parent smoking. The results show a substantially larger gap (β = -5.55, p < 0.001). This parameter estimate changes only slightly with the inclusion of the interaction with age, suggesting that the size of the gap attributable to this indicator of childhood health is consistent over time. Correspondingly, the parameter estimate for the interaction between age and missing school due to poor health in model 4 is small and non-significant (β = 0.01, n.s.). Similar results were obtained for self-reported childhood health. Model 5 shows a large negative effect (β = -2.39, p < 0.001). Note that self-reported health is measured on a five-point scale (rather than as a binary for the previous two items). When we allow the effect of self-reported childhood health to depend on the respondent's age (model 6), we again find no significant interaction (β = -0.02, n.s.). For all three measures of childhood health then, the associated gaps in later-life health remain constant with age.

Models 7 and 8 combine the three indicators. The overall pattern of results for each item is unchanged, although there is some attenuation in the size of the parameter estimates, likely due to the overlap in health measurement between missing school due to poor health and self-rated childhood health. The largest difference is for missing school due to poor health, where the estimated effect in model 7 is -3.44, compared to -5.55 in model 3 which does not adjust for self-rated childhood health or parent smoking.

Finally, models 9 and 10 investigate how these results are altered when controlling for early life and mid-life factors respectively. Parent smoking is no longer a significant predictor of physical functioning after adjustment for early life family and socio-economic circumstances (β = -0.67, n.s.), suggesting that the association between parent smoking and later-life health may be explained by some of the 'early life' factors that were included in model 9. Supplementary analysis (available on request from the authors) where we entered each of the 'early life' factors one at a time identified that this is primarily attributable to controls for fathers' occupational status. Controlling for own educational attainment in isolation is also sufficient to render this parameter non-significant. The estimated effect for self-rated childhood health also declines substantially in magnitude (β = -1.33, p < 0.001) but remains significant. The effect for missing school due to poor health in fact *increases* marginally when conditioning on early life factors (β = -4.15, p < 0.001). In model 10 we see that adding terms for adult risk factors including mid-life socio-economic position, marital status, social support, and health behaviours further reduces the effect of self-rated childhood health (β = -0.80, p < 0.01), accounts for roughly one-fifth of the effect of missing school (β = -3.28, p < 0.001) and makes little difference to the (already small) parameter for parents' smoking (β = -0.53).

We also investigated several potential background moderators of the associations between early life health and later-life physical functioning, including respondent's ethno-migrant background and gender. The results of these analyses are presented in Table 2.[2] To aid

[2] In supplementary analysis, the same set of mid-life controls included in model 10 in Table 1 were also included in the moderation analysis. Results (not shown) indicate no change to the pattern of moderation presented in Table 2.

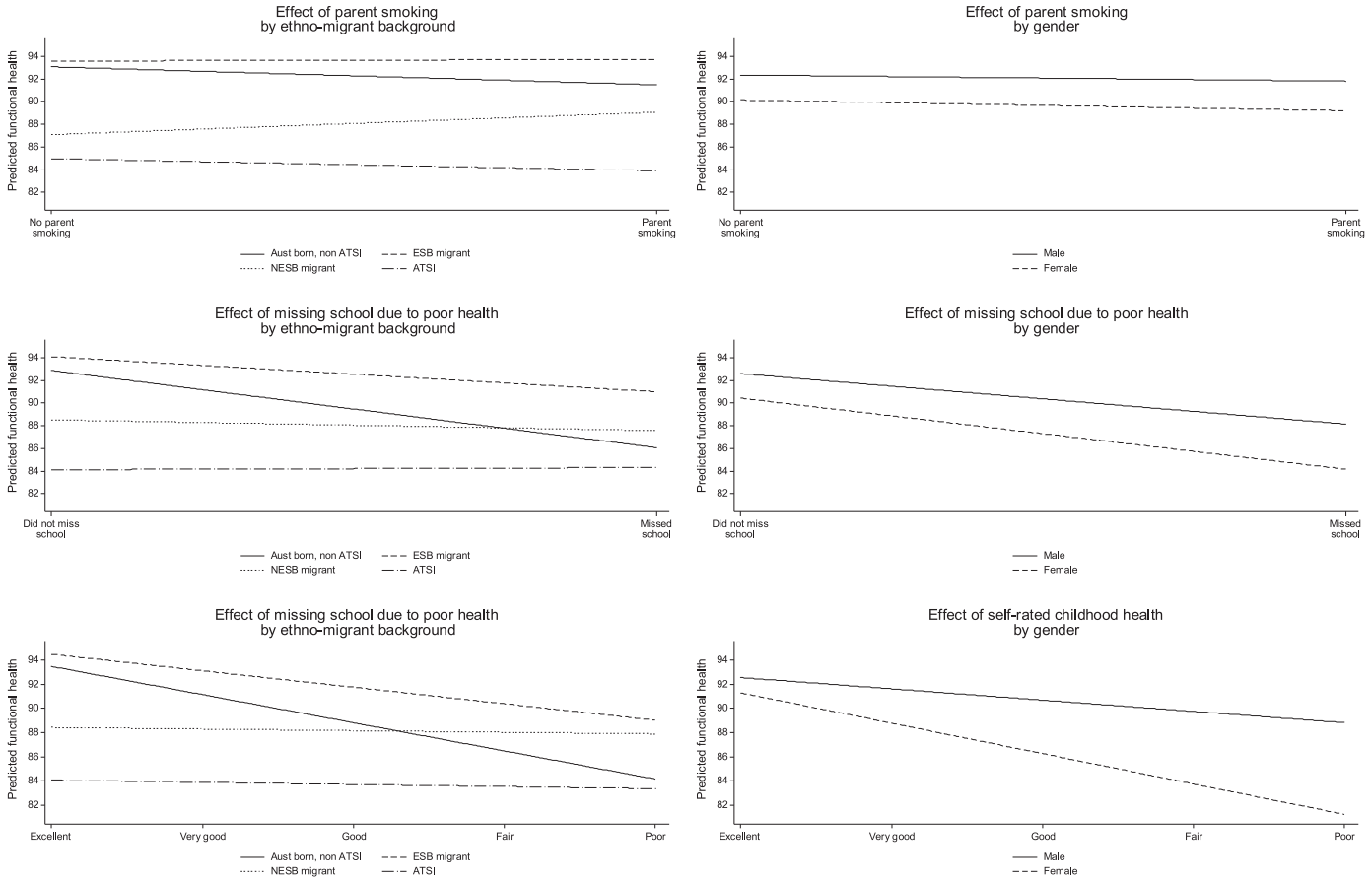

Fig. 1. Moderation of the relationship between child health and adult health.

interpretation of the results, the estimated effects of early life health on later life health are plotted separately by ethno-migrant background and gender in Fig. 1. For ethno-migrant background, we consistently find that the effects of childhood health are smaller among migrants from non-English speaking countries than they are among Australian born non-Indigenous respondents. Migrants from English speaking countries and Indigenous respondents were generally indistinguishable from the reference group in this regard. This is consistent with existing research of a healthy migrant effect (Biddle, Kennedy, & McDonald, 2007; Hamilton, 2015), whereby the migration process selects from a more healthy, rather than unhealthy, population. The fact that we do not find differences for migrants from English-speaking countries suggests that the pool of migrants, and/or the selection process of migration, might vary across different groups of migrants from different countries, though further research is necessary in order to draw a more decisive conclusion. Finally, we find that for the self-rated childhood health measure only, there is a stronger association between early life and later-life health among women than among men. Potentially, this could be due to gender differences in adult opportunities to compensate for poor childhood health (Montez & Hayward, 2011).

Sensitivity analysis

Although retrospective reports of childhood health have generally been found to be valid and reliable (Haas, 2007), there are nonetheless some concerns regarding potential reporting biases and respondents' ability to accurately describe their health many years earlier (Vuolo & Staff, 2013). In particular, a recent analysis by Vuolo and Staff (2013) showed that poor performance on a working memory test was associated with more change (positive and negative) in respondents' ratings of childhood health. These findings suggest that respondents with poor memory function may provide biased or unreliable reports of childhood health. To address this concern we conducted supplementary analyses where we controlled for a measure of working memory. Working memory was measured at wave 12 using a backwards digit span task, with respondents asked to repeat numbers of increasing length in reverse order. Performance on the memory test was found to have a strongly positive relationship with functional health net of other covariates in our final model, but adjusting for this measure did not meaningfully alter our findings. We also tested interactions between memory and childhood health, on the grounds that there may be a comparatively stronger relationship among those with good memory owing to more reliable reports of childhood health in this group. Encouragingly, we found no evidence of significant interactions between memory scores and reports of childhood health in predicting functional health, suggesting that this is not the case in our data. Results from this analysis are available on request from the authors.

Conclusion

This study contributes to our understandings of the association between childhood health and later-life physical functions. Drawing on a nationally representative longitudinal dataset, with three retrospective measures of childhood health, we examined their associations with levels of physical functional health in later life, at age 50 and over in Australia. We hypothesized that childhood health may be associated with later-life health in one of three ways, consistent with expectations from (1) cumulative disadvantage theory, (2) a 'persistent inequality' approach, or (3) the 'age as leveler' approach. In response to our first research question, our findings show that poor childhood health is associated with lower levels of physical functioning in later life. However, in response to our second research question we find no association

between childhood health and the rate of functioning decline over time, supporting the 'persistent inequality' interpretation. We note that this finding is different to findings from Haas (2008), which drew on a dataset from the U.S. to show that poor childhood health is associated with both lower levels of functioning health, as well as a faster rate of functional decline in later life. One potential explanation for this variation in findings might be due to differences in the social safety net across the two countries, with the availability of universal healthcare in Australia likely to reduce the accumulation of disadvantages to those who reported poor childhood health. Note however, that though functional health differences due to childhood health are not exacerbated in later life, neither are they eliminated. Future research that is able to consider the association between childhood health and later-life physical functions in other contexts would be useful to contribute knowledge on the degree to which country-level institutional factors and policies reduce the long-term effects of early life adversity (Haas & Oi, 2018). Doing so could also potentially inform programs and interventions that could eliminate health disparities that arise in early childhood.

Our third research question concerns variations in the association between childhood health and later-life physical functions across different demographic groups within Australia. Here, we find that the association is stronger for women than men. While the interaction parameters are negative across all three measures of childhood health, it was found to be statistically significant only for self-rated childhood health. This might suggest that the childhood health measures may be capturing different aspects of health. Arguably, self-rated childhood health is a broad measure of health, encompassing both physical and mental health. In contrast, exposure to parental smoking may result in higher likelihoods of developing a chronic condition in adulthood (Lovasi et al., 2010; Skorge et al., 2005; White et al., 2017), while school absenteeism due to poor health in childhood may reflect a chronic illness in childhood (Blackwell, Hayward & Crimmins, 2001). What this suggests is that different aspects of childhood health may be associated with later-life physical functions through different pathways. Nevertheless, this finding of gender differences is similar to results presented by Montez and Hayward (2011), which showed an association between poor childhood health and mortality for non-Hispanic white women, but not men.

We also found that poor childhood health was more weakly associated with later life physical functioning among migrants from a non-English speaking background, compared to the non-Indigenous Australian born population. The pattern of findings is consistent across all three of our measures of childhood health. A likely explanation is that prospective migrants whose poor childhood health has continued into adulthood are less likely to migrate to Australia, and the migrant population is therefore positively selected in this regard. We note that a number of studies based in North America finds immigrants face a number of barriers in health care access, including insurance coverage, language barriers and barriers to information, as well as showing variations in health-related beliefs (Clough, Lee & Chae, 2013; Kalich, Heinemann & Ghahari, 2016). While these barriers are likely to be present in the context of Australia as well, universal healthcare coverage in Australia may mitigate health disparities due to health insurance coverage. Further, while language barriers and barriers to information also likely exist to some extent in Australia, we note an overwhelming majority of migrants (85%) live in an urban area, and in the two largest cities of Sydney and Melbourne where there may be more resources in the form of health information in multiple languages and informal social networks that may mitigate such barriers (Australia Bureau of Statistics, 2014). Our identification of a weaker association between poor childhood health and later-life physical functioning for migrants from a non-English speaking background may therefore be a conservative estimate, as presumably migrant health would be better in the absence of any barriers they may experience in Australia.

While our study makes a number of contributions, there are also some limitations. First, our measures of childhood health are imperfect as they do not indicate the specific age at which the respondents may have experienced poor health. Rather the survey questions prompted the respondents to consider their full childhood period before age 15. Future research that is able to consider potential variations accounting for the specific age at which individuals may have experienced poor health in childhood, as well as potential variations in their associations with later-life health, would be valuable. Second, our measures of childhood health are retrospective rather than prospective reports, and therefore may be subject to recall bias. One study by Haas (2007) however, reports that retrospective self-rated childhood health is a robust indicator. In their study, Vuolo et al. (2014) also made recommendations to reduce bias associated with retrospective rating of childhood health. Following from their recommendations, we conducted sensitivity analysis with respondents' working memory scores, and found that adjusting for memory and interactions of memory with child health did not alter our findings. Of note is that we would also expect our other two measures of childhood health (parent smoking, and missing school) to be less affected by recall errors, as parental smoking and missing school are specific events which may be less subject to recall bias. It is also important to note that there is likely to be considerable heterogeneity within categories of our childhood health measures – for instance our parental smoking measure does not capture any information regarding the duration or intensity of smoking. Finally, our sample is likely to be subject to mortality selection, whereby we are only able to observe respondents who have survived long enough to enter the study. Thus, our results are likely to underestimate the true association between poor childhood health and later-life health. This is a limitation faced by similar studies on this topic (see Haas & Oi, 2018).

Our paper has nevertheless advanced existing knowledge, while pointing to important future directions of research. For instance, we explored and found heterogeneity in the associations between childhood health and later-life physical functions, suggesting differences in vulnerability to poor childhood health. Future research that is able to establish and unpack variations in the pathways between childhood health and later-life functions would be able to provide deeper insights into this life course process. Such research may also inform programs that could lessen the impacts of poor childhood health. Further, drawing on existing research on childhood disadvantage and later-life health (Beckett, 2000; Ferraro & Farmer, 1996; Ferraro & Shippee, 2009; House et al., 1990, 1994; Kelley-Moore & Ferraro, 2004; Willson, Shuey, Elder & Glen, 2007), we have also provided important insights into how early life disadvantage – in this case childhood health – may be related to outcomes in later life.

To summarize, the current study adds to the existing body of knowledge by considering the association between childhood health and later-life physical functioning in a specific context – Australia – that has been overlooked thus far in empirical work. Most of the previous research utilizes data from the United States (Haas, 2008), the United Kingdom (Jackson, 2015), or Europe (Haas & Oi, 2018), and to our knowledge, we are the first to examine this research question in Australia. Given cross-national variations in the healthcare system, as well as differences in safety net policies and levels of support for those who are in poor health, it is important to consider whether associations observed in other countries are also found in Australia. Australia has, arguably, a stronger and more supportive health care system that countries such as the United States, including universal health care coverage, which may mitigate some of the long-term outcomes of poor childhood health. Nevertheless our results broadly replicate established findings from other countries, showing that poorer self-rated childhood health is associated with worse health in later life (Haas, 2008; Haas & Oi, 2018). But, as noted, we do not find evidence that childhood health explains health trajectories in later life, a finding that is inconsistent with the only other study we know which has examined this, using data from the United States (Haas, 2008). Potential explanations for this may be cross-national differences in healthcare systems, though this is an

empirical question to be tested by further research investigating how variations in national infrastructure, policies and programs, may modulate the effects of poor childhood health. Future research that is able to combine both a life course perspective, incorporating dynamics and mechanisms over a long period of time, as well as examination of variations across institutional and national contexts may therefore be especially beneficial, and would be of value for academic and policy purposes.

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Ethics approval

Ethics approval not required.

Appendix

See Appendix Tables A1 and B1.

Table A1
Descriptive statistics.

Variable	Mean/ Percent	Standard deviation	Percent imputed
Outcome			
Physical functioning	71.7	26.6	9.2%
Child health measures			
Parents smoked			
No (ref.)	30.0%		25.0%
Yes	70.0%		
Missed a month of school due to poor health			
No (ref.)	88.0%		24.9%
Yes	12.0%		
Self-rated childhood health	-0.1	0.99	24.9%
Controls (time invariant)			
Sex			
Male (ref.)	47.3%		0
Female	52.7%		
Ethno-migrant background			
Aust-born non-Indigenous (ref.)	68.5%		
English-speaking background migrant	13.9%		0
Non-English speaking background migrant	16.1%		
Aboriginal or Torres Strait Islander	1.5%		
Birth cohort			
1902–1919	1.5%		0
1920–1924	3.4%		
1925–1929	6.6%		
1930–1934	8.6%		
1935–1939	10.6%		
1940–1944	13.3%		
1945–1949 (ref.)	17.7%		
1950–1954	17.4%		
1955–1955	13.4%		
1960–1965	7.5%		
Father unemployed for 6 + months			
No (ref.)	89.1%		6.2%
Yes	10.9%		
Father's occupational status	0.0	1.01	4.3%
Respondent eldest child			
No (ref.)	64.8%		0
Yes	35.2%		
Number of siblings (top-coded at 5 +)	2.8		0
Lived with both biological parents at 14			

(*continued on next page*)

Table A1 (*continued*)

Variable	Mean/ Percent	Standard deviation	Percent imputed
No (ref.)	16.6%		0
Yes	83.4%		
Highest level of education achieved			
University degree (ref.)	19.1%		0
Vocational qualification	30.8%		
Completed secondary	8.1%		
Less than completed secondary	41.9%		
Number of children ever had			
Zero	10.8%		0
One	9.5%		
Two or more	79.7%		
Controls (time-varying)			
Age	64.0	10.33	0
Social support	-0.01	1.00	
Subjective financial prosperity	-0.01	1.00	
Marital status			
Married	62.4%		
De facto	6.1%		
Divorced/separated/widowed	26.4%		
Never married	5.1%		
Labour market status			
Employed	42.3%		
Unemployed	1.3%		
Not in the labour force	56.4%		
Smoking			
Never smoked	49.1%		8.9%
Former smoker	37.5%		
Current smoker	13.3%		
Alcohol consumption volume			
Non-drinker	21.3%		9.2%
1-2 standard drinks	53.1%		
3+ standard drinks	25.6%		
Frequency of physical activity			
Not at all	16.7%		8.0%
Less than once a week	14.4%		
1 to 2 times a week	21.1%		
3 times a week	14.5%		
More than 3 times a week	21.0%		
Every day	12.3%		

Table B1
Associations between childhood health and background factors (percent).

	Missed school – poor health		*Parent smoked*		*Self-rated health*[(a)]	
	No	Yes	No	Yes	Good	Poor
Sex [(d)]						
Male	89	11	29	71	95	5
Female	87	13	31	69	93	7
Ethno-migrant background [(b) (c)]						
Australian born, non-Indigenous	88	12	31	69	94	6
1st Gen. immigrant, English speaking countries	86	14	18	82	94	6
1st Gen. immigrant, non-English speaking countries	91	9	39	61	94	6
Indigenous	85	15	20	80	94	6
Birth cohort [(d)]						
1902–1919	86	14	31	69	91	9
1920–1924	90	10	36	64	95	5
1925–1929	89	11	35	65	94	6
1930–1934	88	12	31	69	93	7
1935–1939	85	15	27	73	91	9

(*continued on next page*)

Table B1 (*continued*)

	Missed school – poor health		*Parent smoked*		*Self-rated health*[a]	
	No	Yes	No	Yes	Good	Poor
1940–1944	89	11	29	71	93	7
1945–1949	87	13	26	74	94	6
1950–1954	88	12	30	70	94	6
1955–1959	89	11	31	69	95	5
1960–1965	89	11	31	69	95	5
Father unemployed 6+ months when growing up [c] [d]						
No	88	12	31	69	94	6
Yes	86	14	25	75	92	8
Father's occupational status when growing up (quintiles) [c] [d]						
1st (disadvantaged)	87	13	25	75	93	7
2nd	88	12	31	69	94	6
3rd	88	12	26	74	93	7
4th	89	11	30	70	94	6
5th (advantaged)	89	11	36	64	95	5
Respondent oldest child growing up? [c]						
No	88	12	31	69	94	6
Yes	88	12	28	72	94	6
Number of siblings						
None	87	13	25	75	94	6
1	87	13	30	70	93	7
2	88	12	30	70	94	6
3	89	11	32	68	95	5
4	88	12	29	71	94	6
5+	88	12	30	70	93	7
Respondent living with both biological parents at 14? [d]						
No	87	13	31	69	92	8
Yes	88	12	30	70	94	6
Respondent's highest level of education [c] [d]						
University degree	89	11	35	65	96	4
Vocational education and training	88	12	30	70	94	6
Completed secondary	90	10	31	69	93	7
Less than completed secondary	87	13	28	72	93	7
Total	**88**	**12**	**30**	**70**	**94**	**6**

(a) Self-rated childhood health is dichotomized for the purposes of this descriptive table only, 'Good' is equivalent to the 'excellent', 'very good', or 'good' response categories, and 'poor' is equivalent to 'fair', or 'poor'. (b) bivariate association with 'missed school' significant at $p < 0.05$. (c) bivariate association with 'parent smoking' significant at $p < 0.05$. (d) bivariate association with 'self-rated health' significant at $p < 0.05$. Responses sum to 100% for each measure of child health within rows of the table.

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