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Health across early childhood and socioeconomic status: Examining the moderating effects of differential parenting

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ABSTRACT

Variations in parenting within the family (i.e. differential parenting) are associated with various domains of child adjustment, whereby disfavoured siblings exhibit poorer social and emotional outcomes. To date there is no research examining the effects of differential parenting on children's general health, or the way in which differential parenting interacts with socioeconomic markers to predict general health over time. The present study assessed 501 Canadian families at 2 time points separated by 18 months. Differential maternal negativity predicted worse health 18 months later. Moreover, the association between maternal education and child health was strongest when children were also exposed to high levels of differential negativity. Findings indicate that multiple forms of social disadvantage (i.e. between families and between siblings) can operate independently or in a cumulative fashion to predict health across early childhood.

Introduction

Prior to conception and continuing throughout infancy and childhood, the pathways towards human health and well-being are influenced by the family socioeconomic context (Conger & Donnellan, 2007; Repetti, Taylor, & Saxbe, 2007; Schofield et al., 2011). However, socioeconomic status (SES) is not the only aspect of family life that plays a critical role in the development of health inequities (Shokonoff, Boyce, & McEwen, 2009; Wilkinson & Marmot, 2003). Interpersonal environments and emotional climates within the family can influence child health directly, and can buffer or exacerbate the effects of SES (Bradley & Corwyn, 2002). Researchers have focused on the ways in which between-family variations in parent–child relationships impact the association between SES and child development (e.g. Burchinal, Roberts, Zeisel, Hennon, & Hooper, 2006; Werner & Smith, 2001). However, there have not been any studies examining the way in which within-family variations in parenting moderate the effects of SES on child health. This within-family variation, or *Differential Parenting*, refers to sibling differences in rearing environments, whereby one child receives more warmth and affection, or hostility and negativity, relative to his or her sibling. Although differential parenting is more likely to occur under settings of socioeconomic risk and contextual stress (Henderson, Hetherington, Mekos, & Reiss, 1996; Jenkins, Rasbash, & O'Connor, 2003), no studies have examined the way in which these factors combine to predict early measures of general child health over time.

Differential parenting has a negative impact on child psychosocial, behaviour and emotional outcomes (Boyle et al., 2004; Burt, McGue, Iacono, & Krueger, 2006; Caspi et al., 2004; Conger & Conger, 1994; Jenkins et al., 2009; Pike & Kretschmer, 2009; Richmond & Stocker, 2009). These findings can be considered through the lens of *distributive justice*, where emphasis is placed upon the conditions in which the goods and harms within a social group are fairly distributed in order to maximize well-being for all individuals (Deutsch, 1985). Issues of distributive justice "occur not only at the societal level but also in intimate social relations" (p. 1. Deutsch, 1985), such as within-family relationships. Existing studies on differential parenting have largely focused on child psychosocial health. Thus, the first goal of the present study was to examine how differential parenting contributes to child general health beyond the influence of SES. Moreover, as many studies have shown that risks combine in the prediction of child well-being (Jenkins, 2008), the second study goal was to examine if differential parenting moderated the effects of SES on child health. We hypothesized that exposure to social *and* familial disadvantage would predict the worst health outcomes. Such findings may illustrate that the "struggles and power imbalances" influencing health within society (p. 1, Hofrichter, 2003) can

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interact with sources of inequality within the immediate family context.

SES, stress and child health

The SES-health relationship can be considered through social causation principles, whereby the conditions of low SES influence health outcomes; and social selection principles, whereby less healthy persons experience downward social mobility (see Conger & Donnellan, 2007; Schofield et al., 2011). Although there is evidence for both relationships, the current study is an evaluation of social causation. Consistent with this, researchers have argued that SES influences child health via three pathways: a) inequitable allocation of *resources* like nutrition, healthcare, housing, and education; b) *stress reactions* caused by parenting, environmental hazards, adverse life events, violence, and neighbourhood problems and c) *health behaviours* like tobacco, alcohol and illicit substance abuse and exercise (Bradley & Corwyn, 2002; McEwen & Seeman, 1999; McEwen & Stellar, 1993; Repetti et al., 2007). Following improvements in our understanding of socioeconomic gradients (Hertzman & Boyce, 2010; Keating & Hertzman, 1999) and the physiological consequences of adverse rearing environments (Rutter, 2002; Rutter & O'Connor, 2004), researchers now suggest that biological stress reactions may be the most important link between child health and contextual risk (Caserta et al., 2008; Evans, 2003).

Parenting and child health

Similar to the effects of SES, there is unequivocal evidence citing the importance of early family environments in child developmental health (Repetti et al., 2007). The effects of parenting on child health may be best conceptualized by examining the associations between parental behaviour and the human stress or emotional system (Jenkins, 2008). Often emerging as a function of negative parenting, there are health consequences associated with the chronic activation of these biological stress pathways (Boyce, 2007; Repetti et al., 2007).

The effects of poor parenting and family adversity on various indicators of child health have been documented. One study examined the impact of parental sensitivity and parental conflict in kindergarten on cardiovascular health during middle-school (Bell & Belsky, 2008a). Early parental warmth predicted a variety of outcomes including heart rate and diastolic and systolic blood pressure, even when controlling for SES. Parenting quality has been linked to early asthma onset, as well. In a prospective cohort study of children with a genetic risk of asthma, problems with caregiving, postpartum depression, and low maternal support during infancy were associated with an increased risk of onset before age 3, and again before age 8 (Klinnert et al., 2001). Parenting behaviour has also been linked to child stress hormones. Children of mothers who express higher levels of warmth and involvement have steeper diurnal cortisol rhythms, a pattern that is thought to be indicative of better health (Pendry & Adam, 2007). Thus, similar to the effects of SES, one possible mechanism by which parenting may be related to child health is through the chronic and persistent activation of the biological stress response systems.

Differential parenting as a source of inequality

Parenting can be viewed as a form of social capital: it is a resource – in the form of a relationship – that predicts developmental well-being and success in society across the lifespan (Coleman, 1990). Not unlike the inequitable distribution of economic capital within nations, social capital (i.e. parenting resources) can be inequitably distributed within families (Boyle et al., 2004). Most studies examining parenting and child health measure a single parent–child dyad. Such research cannot reveal the complex ways in which parenting operates within a family.

Jenkins (2008) notes that measured environments can be divided into the family-level and child-specific. Factors that all siblings experience similarly operate at the family-level, (e.g. family conflict, maternal depression, or divorce). Conversely, there are experiences that differ for each sibling, (e.g. unique peer or school context). Dimensions of parenting can be conceptualized using family-level and child-specific distinctions. The family-level average of a particular parenting construct is conceptualized as *Ambient Parenting (AP)*, referring to the amount of that dimension present in the household atmosphere (Jenkins et al., 2009). The child-specific deviations from this family average is referred to as *Differential Parenting (DP)*, and can be conceptualized as the amount of negativity or positivity a child experiences relative to the family average. The fact that siblings are more dissimilar than similar after controlling for genetic effects is partially attributable to non-shared environmental influences like differential parenting (Turkheimer & Waldron, 2000). There is a large literature citing the effects of differential parenting on various psychosocial, behavioural and emotional outcomes, including delinquency, sibling relationships, aggression, and emotional disturbance (cited above). Seeing as there are similar biological pathways underlying the parental effects on child mental and physical health (Repetti et al., 2007), it is likely that such associations exist between indicators of general child health and differential parenting.

There are multiple factors that play into the emergence of differential parenting. Though we have cited literature on the effects of differential parenting on child psychosocial functioning, it is also true that children with behavioural or emotional problems elicit higher levels of differential treatment. Parents are reactive to the aggressive behaviour patterns of their children (Granic & Patterson, 2006) and this pattern may be greater under situations of socioeconomic stress (Jenkins et al., 2003). In other words, differential parenting both impacts child behaviour (Burt et al., 2006) and arises from sibling differences (Richmond, Stocker, & Rienks, 2005). While child characteristics certainly contribute to the emergence of differential parenting, the focus of the current study is on the later health consequences of this differential treatment, regardless of its origin. That is, we are interested in the health consequences of differential parenting: a family process that likely emerges through a complex interaction between factors at parent and child levels, and environmental risk factors.

The moderating effect of parenting

Environmental risks have been found to operate contingently in the prediction of children's mental health. Rutter's cumulative risk model (1983, 1993) suggests that individual risk factors transmit minimal harm when present in isolation; multiple risks, however, confer great harm when they combine in a cumulative fashion. Evans (2003) has shown that the aggregation of risks, including poverty, single parenthood, and low parental education, is also associated with heightened cardiovascular and neuroendocrine dysfunction and increased body fat.

Poor quality parent–child relationships may leave children vulnerable to the consequences of other environmental risks. For example, Simons and colleagues (Simons et al., 2006), showed that African American boys exposed to high levels of racial discrimination only exhibited antisocial, violent and delinquent behaviour if parents were unsupportive. Similarly, in a sample of adopted children, Kriebel and Wentzel (2011), showed that the highest levels of behavioural problems were experienced by those with high levels

of pre-adoption risk and low levels of child-centred parenting in the adoptive home. The effects of biological risk factors (including blood lead levels) are also moderated by parenting quality in the prediction of child cognitive outcomes (Hubbs-Tait et al., 2009). Taken together, this body of research suggests that the effects of non-parenting risk factors are most pronounced under situations of negative parenting.

It should be noted, however, that risks may combine with one another in a fashion other than that suggested by the cumulative risk model. The Blaxter Hypothesis (1990) suggests that negative health behaviours exert greater influence in *high* SES populations because these persons have "further to fall" and because other factors may be more important for low SES groups. There is evidence in support of the Blaxter Hypothesis, though this pattern is often observed among adults (e.g. Schafer, Ferraro, & Williams, 2011). Given the outcome of interest and the developmental population in question, the current study proposed and tested a cumulative model. Consistent with the literature cited above, it was hypothesized that low SES and high differential parenting would combine to create the poorest health outcomes.

In summary, the first goal of this study was to test the hypothesis that differential parenting predicts later child health, over and above socioeconomic status and ambient parenting. The second goal was to test the hypothesis that the children most susceptible to the negative influence of SES were those who were disfavoured by parents.

Methods

Participants

Participants for the current study are from the Intensive sample of the Kids Families and Places Study (http://kfp.oise.utoronto.ca/). All of the women giving birth to infants in the cities of Toronto and Hamilton, Ontario (Canada) between April 2006 and September 2007 were considered for participation. Families were recruited through a program called Healthy Babies Healthy Children, run by Toronto and Hamilton Public Health, which contacts the parents of all registered newborn babies within several days of the newborn's birth. Healthy Babies Healthy Children is a universal, voluntary program that offers support to all parents and their children during the transition to parenthood. Inclusion criteria for the Intensive component of the Kids Families and Places Study (which involved longitudinal follow-up and intensive measurement) included the presence of an English-speaking mother, a newborn >1500 g, 2 or more children < 4 years and families agreeing to be filmed in the home. The project was funded and approved by the Canadian Institutes of Health Research.

Thirty-four percent of families approached in Toronto agreed to take part. At Time 1 (T1; infants were 2 months old), 501 families took part in the intensive sample data collection and these families were followed up at T2. Reasons for non-enlistment included inability to contact families and refusals. We compared our sample with the general population of Toronto and Hamilton using 2006 Census Data, limiting the census to women between 15 and 54 years. Additionally, since mothers with newborns are more likely to be partnered, statistics for marital status were obtained from the Canadian National Longitudinal Survey of Children and Youth, a nationally representative sample of children 0–11 years old (Statistics Canada, 2008). Families were of similar size, and income. Families from the Kids Families and Places Study had higher education levels, immigrant status was lower (57.7% vs. 47.6%) and mothers were more likely to be partnered (see Meunier, Bisceglia, & Jenkins, in press) in our sample vs. the general population. In the current investigation, data for the dependent variable was only collected at T1 and T2 for sibling 2 (i.e. closest to newborn). Thus, sibling 2 is the focus of this investigation, though all siblings in the family to a maximum of 4 were-assessed for parenting, permitting an accurate calculation of differential parenting (DP). There were 501 Sibling 2's who participated in the KFP at T2. At Time 2122 Sibling 2's (24%) were missing dependent measures.

Measures

Covariates, socioeconomic indicators and parenting behaviour were measured at T1. Maternal report on child health was collected at T1 and T2.

Covariates

The following demographic measures and covariates were collected from the mother: child gender (girl = 1), child age, ethnicity (Asian = 1, African-Canadian = 1, European = reference), mother immigrant status (non-immigrant = 1), enrolment of child in childcare (yes = 1), presence of asthma (yes = 1), family status (single family = 1, step family = 1, intact family = reference), mother working or studying out of the home (fulltime or part time employment or student = 1).

Previous studies have found that rates of asthma and obesity are higher among boys and African-Americans, and lower among children of Asian heritage, relative to their white counterparts (Stingone, Ramirez, Svensson, & Claudio, 2011). Children with asthma are more likely to be hospitalized with influenza (Miller et al., 2008), though incidence of respiratory problems declines across childhood (Matricardi et al., 2008). Moreover, children of immigrant parents have poorer general health (Huang, Yu, & Ledsky, 2006). Maternal employment and single parenthood are also associated with lower levels of child health (Bradley & Corwyn, 2002). Age dispersion amongst siblings was controlled for (standard deviation within families), as age differences may be associated with a normative level of differential treatment (Kowal & Kramer, 1997; Meunier et al., in press).

Low birth-weight and maternal depression have also been linked to maternal reports of health problems amongst children (Turney, 2011). In the present study, birth-weight was reported by the mother. In accordance with convention (e.g. Costello, Worthman, Erklani, & Angold, 2007), we dichotomized birth-weight so that children less than 2.5 kg (5.5 pounds) were considered "low birth-weight". Maternal depression was assessed using the Center for Epidemiological Studies Depression Scale (CES-D, Radloff, 1977), which is self-report instrument for non-clinical populations. Using the past week as a recall period, 20 depressive symptoms are evaluated on a 4-point scale (higher scores mean more depression). Internal consistency was good ($\alpha = .84$).

Socioeconomic variables

The Kids Families and Places study sample has a high proportion of immigrants. As there is a more modest association between income and education in immigrant samples (Leyendecker, Harwood, Comparini, & Yalcinkaya, 2005), these constructs were kept separate. *Income and Assets* was measured as a composite. Parents responded to the following questions: "how many rooms do you have in your house"; "Do you own or co-own this home/apartment/unit, even if still making payments: yes = 1, no = 2"; "Do you own or co-own a car, even if still making payments: yes = 1, no = 2". These questions, in addition to a question pertaining to annual income, were standardized and coded so that all variables were going in the same direction, where higher scores were

indicative of higher SES. Items had an internal consistency of $\alpha = .80$. *Maternal Education* was assessed by asking mothers the number of years of formal education they have completed, excluding kindergarten.

Positive and negative parenting

Parenting was assessed using self-report measures derived from the Canadian National Longitudinal Survey of Children and Youth. Mothers were asked to rate a variety of statements describing affection, positive interaction, punishment and hostility in the parent–child relationship using a 5-point Likert Scale (1 = never, 5 = many times each day). Items, adapted from Strayhorn and Weidman's (1988) Parent Practices Scale, have been repeatedly subjected to factor analyses by the National Longitudinal Survey of Children and Youth, now in its 8th measurement wave, resulting in 4 psychometrically sound dimensions: Warmth/Involvement, Hostility/Ineffectiveness, Consistency and Rationality. The current investigation focused on the Warmth/Involvement dimension (5 items called parental *Positivity*) and the Hostile/Ineffective dimension (5 items called parental *Negativity*). These measures were specifically adopted due to their widespread utilization and validation in the multidisciplinary study of parenting, being associated with indices of child adjustment and family-level adversity in the theoretically expected ways (Browne, Odueyungbo, Thabane, Byrne, & Smart, 2010; Ho, Sand, & Jenkins, 2008; Kohen, Leventhal, Dahinten, & McIntosh, 2008; Peters et al., 2010; Strohschein, Gauthier, Campbell, & Kleparchuk, 2008; Thurston et al., 2010; Willms, 2002, pp. 71–104). In previous studies these scales have also provided valid measurement of differential parenting (Boyle et al., 2004; Jenkins et al., 2003).

Examples from the positivity scale include 'how often do you and your child laugh together?', and 'How often do you and he/she talk or have fun with each other for 5 minutes or more?' Internal consistency for this scale was good ($\alpha = .79$). Examples from the negativity scale include 'how often do you get angry with your child?', and 'How often do you get annoyed with him/her for saying or doing something he/she is not supposed to?' Internal consistency for this scale was also good ($\alpha = .80$). Consistent with precedence, the parenting scores for each child were derived by taking the mean of all items for positivity or negativity, creating a range of possible scores from 1 to 5.

Though only sibling 2 was included in the primary analysis, parents reported on parenting for of all their children to a maximum of 4. Based on established methodology, this permitted the assessment of ambient parenting (AP) and differential parenting (DP) (Jenkins et al., 2009). First, AP was calculated by taking the family average for positivity and negativity. Higher scores on AP indicate higher average levels of either positivity or negativity in the family. DP was calculated by subtracting the family average from the individual raw score. Higher scores on DP indicate that children are experiencing more of that parenting dimension, relative to the siblings. For positivity, higher scores would suggest the child is favoured. For negativity, higher scores would indicate the child is disfavoured. Both AP and DP are conceptually different and must be included in statistical models.

Child health

General child health was the primary outcome of interest. Mothers responded to the following question at baseline and 18 month follow-up: In general, would you say this child's health is (1) excellent, (2) very good, (3) good, (4) fair or (5) poor. Similar ordinal measures of child health have been employed in other survey-based epidemiological studies (Chen, Martin, & Matthews, 2006a,b).

Data analysis

Continuous study variables were summarized using means and standard deviations. Frequencies and percentages for categorical variables are given. Correlations amongst continuous predictor variables were examined using Pearson's correlation coefficients. Ordinal regression using cumulative probability logits was used to examine the relationship between the predictors and the outcome. In addition to the covariates, ratings of child health were controlled for at T1. Thus, odds ratios can be interpreted as odds of worse ratings of child health, relative to other children at T2, irrespective of T1 health. Odds apply to all cumulative ordinal comparisons on the health measure (1 vs. 2–5, 1–2, vs. 3–5, etc.), which were assumed to be equivalent, though this assumption was empirically tested using the test of parallel lines (not shown). Child health at T1 was centred at 3 ("good health"). All other continuous variables were transformed into *Z*-scores so estimates could be interpreted as the odds associated with a 1 standard deviation increase in the predictor. Categorical variables are treated as dummy variables, as outlined above. All analyses were conducted using SPSS 19.

Model building took place in two steps. First the main effects of parenting (ambient and differential) were entered into the regression equation while controlling for all covariates. Second, two interactions were entered into the equation: income and assets*DP, and maternal education*DP. This process was conducted separately for positivity and negativity. Model fit was evaluated using the likelihood ratio test.

Results

Missing data

There was a moderate amount of missing data on some variables (0–28%). Recommendations laid out by Graham (2009) were utilized for the handling of missing data. Descriptive statistics and a correlation matrix of continuous variables are reported using data derived from Estimation Maximization (Little & Rubin, 1987, 2002). Multiple Imputation, as described by Schafer (1999) and Rubin (1987), was utilized for hypothesis testing. Simulation studies have demonstrated that in Multiple Imputation performs adequately even when samples are small ($N = 50$) and when there is 50% missing data. Five datasets were generated using the Multiple Imputation program within SPSS 19 and we report the pooled estimates.

Analyses

Descriptive statistics for continuous and categorical predictor variables are presented in Tables 1and 2. Table 3 shows the correlation between variables in the model. At T1, 335 (66.9%) mothers

Table 1
Descriptive statistics of continuous predictor variables at Time 1.

Variable	Min	Max	*M*	SD
Child age	0.58	4.33	2.58	0.76
Sibling age SD	0.29	7.39	2.07	1.04
Maternal depression	0.00	40.00	9.47	7.24
Income and assets	−1.93	1.40	0.07	0.81
Maternal education	8.00	22.00	15.27	2.67
Ambient positivity	3.00	5.00	4.47	0.44
Differential positivity	−0.40	0.93	0.03	0.13
Ambient negativity	1.40	5.00	2.81	0.55
Differential negativity	−1.13	0.70	−0.02	0.15

Note: SD = within-family standard deviation. $N = 501$.

Table 2
Descriptive statistics of categorical predictor variables at Time 1.

Variable	*N*	%
Girl	245	48.9%
African/Black	46	9.2%
Asian	133	26.5%
Mother non-immigrant	268	53.5%
Low birth-weight	29	5.8%
Child asthma	30	6.0%
Attending childcare	318	63.5%
Single parent family	32	6.4%
Step family	25	5.0%
Mother out of home	38	7.6%

$N = 501$.

reported that their children had excellent health, 114 (22.8%) reported very good health, 46 (9.2%) reported good health and 6 (1.2%) reported fair health. At T2, 312 (62.3) had excellent health, 136 (27.1) had very good health, 43 (8.6) had good health, 8 (1.6) had fair health and 2 (0.4%) had poor health.

Results for maternal negativity are presented in Table 4. Model 1 was a significant improvement over the null model, which fitted only threshold estimates (-2 *Log Likelihood* $= 802.90$, $\chi^2(18) = 145.16$, $p < .001$). Child health at T1 was a significant predictor of health at T2. A one unit increment towards poorer health ratings at T1 increased the odds of having poorer health at T2 by a factor of 2.93. Maternal education was a significant predictor of T2 health, as well. Controlling for child health at T1, a one standard deviation increment in maternal education at T1 corresponded to odds of poorer health at T2 that were 30% lower. Finally, differential negativity was associated with higher odds of having poorer health. A one standard deviation increment in differential negativity at T1 corresponded to odds of poorer health that were 49% higher.

In Model 2 the income and assets*DP and maternal education*DP interactions were evaluated. The income and assets*DP interaction was non-significant so it was removed, while the effect of maternal education on child health was significantly moderated by DP. The resultant model was a significant improvement over Model 1 (-2 *Log Likelihood* $= 797.31$, $\chi^2(1) = 5.58$, $p = .018$). The maternal education*DP interaction is plotted in Fig. 1. The effect of maternal education on child health is strongest for the disfavoured sibling, or when there are high levels of differential negativity.

There were no significant main effects for ambient or differential positivity, and no significant interactions of differential positivity with income or maternal education. Consequently, results for positivity have not been presented in table format. Supplementary analyses, including the non-significant positivity results, are available on the personal website of the second author (http://jenkinslab.wordpress.com/).

Table 4
Ordinal regression output indicating odds of worse scores in mother reported child health at T2 when controlling for T1 scores.

Variable	OR (95%CI)	OR (95%CI)
Model 1 and Model 2	Model 1	Model 1
T1 child health	**2.93 (2.08, 4.12)**	**2.98 (2.12, 4.21)**
Girl	1.03 (0.67, 1.58)	1.02 (0.67, 1.57)
Child age	0.37 (0.12, 1.18)	0.39 (0.12, 1.25)
African/Black	0.23 (0.04, 1.25)	0.20 (0.04, 1.13)
Asian	1.54 (0.83, 2.86)	1.51 (0.81, 2.82)
Non-immigrant	0.75 (0.43, 1.31)	0.73 (0.42, 1.30)
Low birth-weight	1.41 (0.41, 4.79)	1.44 (0.41, 5.01)
Child asthma	1.56 (0.62, 3.90)	1.55 (0.62, 3.90)
Attending childcare	1.18 (0.74, 1.89)	1.16 (0.73, 1.85)
Single parent family	0.90 (0.28, 2.91)	0.96 (0.30, 3.14)
Step family	1.33 (0.39, 4.52)	1.49 (0.44, 5.05)
Sibling age difference	0.93 (0.59, 1.46)	0.92 (0.58, 1.46)
Mother out of home	0.72 (0.29, 1.81)	0.72 (0.28, 1.82)
Maternal depression	1.12 (0.88, 1.43)	1.11 (0.86, 1.43)
Income and assets	0.82 (0.51, 1.32)	0.80 (0.50, 1.30)
Maternal education	**0.70 (0.54, 0.91)**	**0.69 (0.54, 0.89)**
Ambient negativity	0.96 (0.77, 1.20)	0.97 (0.77, 1.21)
Differential negativity	**1.49**[a] **(1.00, 2.24)**	1.35 (0.90, 2.03)
Maternal education*Differential negativity		**0.72 (0.53, 0.97)**

Note. Significant effects at 0.05 are in bold. OR = Odds Ratio, CI = Confidence Interval.
[a] $p = .05$.

Discussion

The goals of the current study were to test two hypotheses: 1) differential parenting will predict later child health and 2) the interaction between socioeconomic status and differential parenting will predict later child health. Both hypotheses received support in the case of differential maternal negativity, but not for differential positivity. Higher levels of differential negativity were associated with higher odds of poorer health while controlling for a variety of confounding variables and SES. This finding contributes to existing research that has demonstrated robust associations between parenting experiences and child health (Bell & Belsky, 2008a,b; Belsky, Bell, Bradley, Stallard, & Stewart-Brown, 2007). Children who are disfavoured within a family have poorer health status 18 months later, compared to children who are favoured.

Beyond the effects of absolute levels of parenting (which were controlled for and non-significant in the current study), differential negativity may influence child behaviour by creating an affectively negative environment that is characterized by a lack of security, competition, fear and anxiety (Jenkins & Greenbaum, 1999). Similar to the negative consequences of social competition and inequality within countries (Wilkinson & Pickett, 2006), the differential allocation of psychological harm within families has a negative impact on disfavoured children, not just in terms of their emotional and behavioural development, but also in terms of their general health

Table 3
Pearson correlations between continuous study predictor variables at Time 1.

		2	3	4	5	6	7	8	9
1	Child age	**0.41**	**−0.11**	**0.09**	0.03	0.01	−0.03	**0.14**	**0.15**
2	Sibling age SD		0.07	**−0.12**	**−0.15**	**−0.28**	**0.42**	**0.15**	−0.07
3	Maternal depression			**−0.27**	**−0.25**	**−0.18**	0.03	**0.30**	−0.08
4	Income and assets				**0.42**	**0.10**	**−0.09**	**−0.19**	0.06
5	Maternal education					0.05	**−0.14**	**−0.13**	0.06
6	Ambient positivity						**−0.20**	**−0.20**	0.05
7	Differential positivity							0.03	**−0.34**
8	Ambient negativity								0.02
9	Differential negativity								

Note. Significant effects at 0.05 are in bold. SD = within-family standard deviation. $N = 501$.

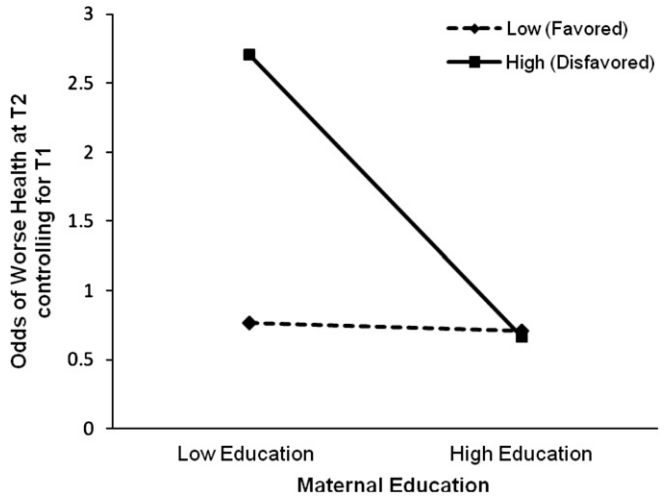

Fig. 1. The effect of maternal education on odds of worsening in mother reports of child health is moderated by differential maternal negativity.

and well-being. Like the effects of absolute levels of parenting demonstrated in other research (Repetti et al., 2007), and the effects of absolute socioeconomic level (Bradley & Corwyn, 2002) and socioeconomic inequality (Wilkinson & Pickett, 2006), effects of differential parenting are likely operative through the stress response. As target children receive more negativity than siblings, and experience themselves in a position of relative disadvantage, stress reactivity is likely to be high. Non-human primate research has demonstrated the physiological consequences of relative disadvantage, where inferior members of dominance hierarchies have poorer health outcomes (Sapolsky, 2005). This trend extends to humans. However, the effect of differential maternal negativity on child health also operates in a broader context of socioeconomic variability, functioning to moderate the effects of environmental risk as indexed by maternal education.

Maternal education confers risk to children through a variety of mechanisms, including increased rates of preterm and small-for-gestational-age births (Luo, Wilkins, & Kramer, 2006), exposure to environmental stressors such as tobacco smoke (Wills, McNamara, & Vaccaro, 1995), inadequate nutrition, neighbourhood stress exposure, and limited access to medical care (Bradley & Corwyn, 2002). It is likely that these environmental risks have the most impact in situations where children are also treated more negatively than their siblings, exacerbating physiological stress. Conversely, the absence of this disfavoured position within the family may buffer the consequences that typically coincide with low maternal education.

Though the psychophysiology of stress reactivity is not focus of this study, the biological mediating mechanisms responsible for the connection between contextual risk and child health should be mentioned. Two primary neurological pathways regulate the emotional stress response (see Boyce, 2007; Repetti et al., 2007). One is the corticotropin releasing hormone (CRH) system, which is comprised of the hypothalamic-pituitary-adrenocortical (HPA) axis and the amygdala system and its interconnections. The other neurological stress pathway is the locus coeruleus-norepiniphrine (LC-NE) system, comprised of the noradrenergic cells in the brainstem and their various connections (Boyce, 2007). Through a cascade of mediating steps, both of these systems are activated in response to stressful stimuli resulting in the release of stress hormones, including cortisol, and the characteristic “fight or flight response”. These responses serve vital adaptive functions, though chronic activation can have negative health consequences. For example, chronic HPA activation can lead to increased levels of plasma CRH and hypercortisolism (Repetti et al., 2007) which, over the life course, correspond to the emergence of insulin resistance, hypertension, atherosclerosis, compromised immunity and bone loss (Boyce, 2007). It is via these mechanisms that stressful conditions “get under the skin” and impact developmental health (Hertzman & Boyce, 2010).

There was no main effect of ambient maternal negativity on child health. This is unexpected given the existing research on parenting and health in early childhood (e.g. Bell & Belsky, 2008a,b). However, studies of parenting and child general health have focused on parenting between families rather than within families. The effects of ambient parenting and differential parenting may have been somewhat confounded in studies that do not have a sibling design. Additional research is required to answer this question. It is noteworthy that both ambient and differential positivity were not found to predict child health. Although there are replicated associations between both negativity and positivity with developmental outcomes, it has been argued that negativity is more closely related to both contextual risk factors and child functioning (Berg-Nielsen, Vikan, & Dahl, 2002).

Limitations and future directions

A few limitations must be mentioned. First, the reliance on within-informant data raises the possibility of shared-method variance bias. The longitudinal, auto-regressive approach reduces this risk, though future studies of differential parenting and child health should rely on multiple informants and biological markers of child health. Secondly, although multiple siblings are enrolled in the Kids Families and Places Study, the burden on parents in answering questions necessitated decisions about which constructs to measure for all children (mental health) and which would be limited (general health). Thirdly, 34% of approached respondents were included in the Kids Families and Places Study, raising representativeness concerns. Our sample is higher functioning than the general population, which should be kept in mind when making generalizations from the current findings. Fourthly, although the use of single item measures is widespread in survey studies (e.g. Chen et al., 2006a,b), this may not be optimal based on the domain-specific model of measurement error, where more items result in greater construct validity; however, single item scales have performed as well as multi-item scales in terms of convergent and discriminant validity (Gardner, Cummings, Dunham, & Pierce, 1998).

Results from the current study have implications for the investigation of social inequality. These findings illustrate the importance of considering multiple forms of inequality and multiple social locations in which inequalities are manifested. The principles of distributive justice are not limited to economic situations within geo-political entities; rather, they extend to any situation in which goods or harms are distributed across units of organization (i.e. countries, families, schools, workplaces, etc.) in a fashion that differentially impacts psychological, physiological, economic or social well-being (Deutsch, 1985). Thus, it is important to apply principles of distributive justice when conceptualizing family relations, and to ensure that parent–child dyads are considered in terms of the broader family environment.

Researchers have articulated the importance of considering multiple sources of capital when examining the determinants of child development (financial, social and human; Bradley & Corwyn, 2002). Consistent with this earlier research, findings from this study suggest that different forms of disadvantage multiply to predict child development. Moreover, inequality from multiple social locations can interact across levels of organization (within

society and within families). It seems that developmental outcomes are most deleterious when individuals are disadvantaged in multiple settings. Stated differently, a favourable relative position in a proximal setting (i.e. the family) can buffer the negative consequences of disadvantage in distal settings (i.e. society). It is important to understand inequality within a multiplicative framework of this kind. More generally, findings from this research illustrate the importance of integrating concepts from developmental psychology (differential parenting or disadvantage within the family) into the population health literature (disadvantage at the societal level), since both of these processes impact health in an interactive fashion. Researchers should continue to apply within-family methodology to understand health inequalities in a global economic environment characterized by increasing gaps between the rich and the poor, and a shrinking middle class.

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Appendix. Supplementary data

Supplementary data related to this article can be found online at doi:10.1016/j.socscimed.2012.01.017.

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