Working paper

The socio-economic gradient in Allostatic Load. Results from the PIAMA birth cohort study

Abstract

How early in life is the socio-economic gradient in health observed? This study investigates this question by focusing on childhood circumstances related to the parental socio-economic position (SEP) and their influence on Allostatic Load (AL) at the age of 12. Using biomarkers collected by the PIAMA population-based birth cohort study in the Netherlands when the respondents were around age 12 (N=1079), we examine the relationship between the parents' education level and the children's AL level and we assess whether this relationship differs between boys and girls. Our results illustrate that parental education matters for both boys and girls, but only when we consider fathers, i.e., the AL score of children of fathers with a high education level was around 5 percent lower than the score of children of low educated fathers. No association with AL was found for maternal education.

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SUMMARY BOX

What is already known on this subject?

The social gradient in health is a result of a lifelong process of exposure to socially patterned life circumstances. The resulting chronic stress is thought to influence the body at the physiological level, a process known under the name of allostatic load. Allostatic load is multi-system construct and a framework of understanding the “long arm of childhood” using a life-course lens.

What does this study add?

This study shows that gender health differences can be traced back to childhood. The allostatic load score of children of fathers with a high education level was around 5 percent lower than the score of children of low educated fathers. No association with AL was found for maternal education.

INTRODUCTION

It is nowadays widely acknowledged that the social gradient in health observed in western societies is a result of a lifelong process of exposure to socially patterned life circumstances [1, 2]. This process starts even before birth[3] and is shaped by circumstances linked to the parental household that have long lasting effects on the health of children and future adults through impacting children's health, their adult socio-economic position and finally, the health status during adulthood and old age [4-6]. Some of the mechanisms behind this "long arm of childhood" are the material and social context during childhood as well as the parental lifestyle and health knowledge, which are all related to the position that parents have in the general socio-economic hierarchy [7].

Although a large body of research has provided ample evidence for the importance of the socio-economic position (SEP) of parents for the health differences during adulthood [7], less is known about the beginnings of this process during childhood. One of the reasons for this lack in the literature is the availability of data, i.e., in order to estimate the importance of the childhood circumstances for the development of health inequalities later in life, most studies use retrospective questions that are collected from samples of adult population [4, 6]. However, in recent years, the accumulation of data collected from cohorts following children from birth through childhood and onwards has allowed a more thorough examination of questions regarding the early origins and the development of health inequalities during childhood [8]. In the present study, we also take this route, by utilizing high quality data collected by the PIAMA study, a Dutch birth cohort study [9], in order to better understand if and how socioeconomic health disparities develop during childhood.

Our approach in this study is to examine one of the mechanisms that is proposed to explain the existence and persistence of health inequalities between socio-economic groups, i.e., the chronic stress exposure. According to this mechanism, the social position over the life course is characterised by differential exposure to environmental and social risks, which results

in accumulated chronic stress and material deprivation [10, 11]. In turn, chronic stress exposure is thought to influence the body at the physiological level, a process that is known in the literature under the name of allostatic load (AL) [12]. AL is a comprehensive empirical and theoretical framework that articulates how chronic stress "gets under the skin" and leads to the development of disease [13]. Its critical assumption is that non-lethal cues from the social environment (e.g., experiences of violence, material insecurity, etc) become stressors and trigger the body's flight or fight reaction [14]. In physiological terms, this process implies the activation of the sympathetic–adrenal–medullary (SAM) and the hypothalamic–pituitary–adrenal (HPA) axis [15], affecting neuroendocrine, inflammatory, metabolic and cardiovascular physiological systems [13]. AL framework emphasizes the importance of chronic stress exposure, i.e., it is the repeated stress response activation or lack of adaptation to the stressors that leads to the "wear and tear" of the body [16] Furthermore, AL is a multi-system construct because the chained reaction of psychological processes spread across multiple interconnected biological systems.

In order to accommodate the above theoretical statements, this study takes a life course perspective on biosocial pathways [17] by examining the association between the parental SEP, as measured by the level of education of both mother and father at the child's age of 1 year, and AL level when the child was around 12 years of age. It is reasonable to expect that the living environment of parents with low SEP is filled with more risks and threatening cues, which, in time, could trigger the stress reaction of the child, and result in higher AL.

By taking this approach our study adds to the literature twofold. First, we contribute to the AL literature which is mainly focused on older adults [15], while *we specifically look at children aged 12*. In fact, the literature that examines whether chronic stress exposure relates to AL in children's samples is very thin, and the majority of studies were conducted in the US [18, 19]. The results of these studies provide support for the idea that worse childhood circumstances

relate to higher levels of AL [20]. While the mechanisms linking parental SEP with childhood AL are universal [21], the question remains whether these findings that came from a specific institutional and cultural setting are also found in another setting, i.e., the Netherlands. The US and the Netherlands differ in their family and child-supportive policies, which are much more generous in the Dutch context [22], one aspect that could moderate the relationship between low parental SEP and AL. In addition, the Netherlands is a much more egalitarian country in terms of income distribution, with an active approach and policy to reduce spatial segregation of the poor [23]. These factors could imply that the environmental and social risks linked to low parental SEP could be lower than in the US, and thus the question is whether we can find the same detrimental effect on children's AL. Second, we will also examine *gender differences in the effect of parental SEP on children's AL*, an aspect that is largely disregarded in the literature. However, in the light of the gendered patterns of health inequality during adulthood and old age that are consistently brought forward by scholars [10], it is highly relevant to examine when these gender differences begin.

METHODS

The data used in this study was collected as part of the Prevention and Incidence of Asthma and Mite Allergy (PIAMA) study, a Dutch population based birth cohort study whose design and content was extensively described elsewhere [9]. The study started with 3963 children who were born in 1996/1997, 3541 children were still in the study at age 11, and 3202 of these children were invited for a clinical examination at age 12 (n participants: 1511). During this examination, weight, height and blood pressure were measured and blood samples were collected and used to compute our measure of AL.

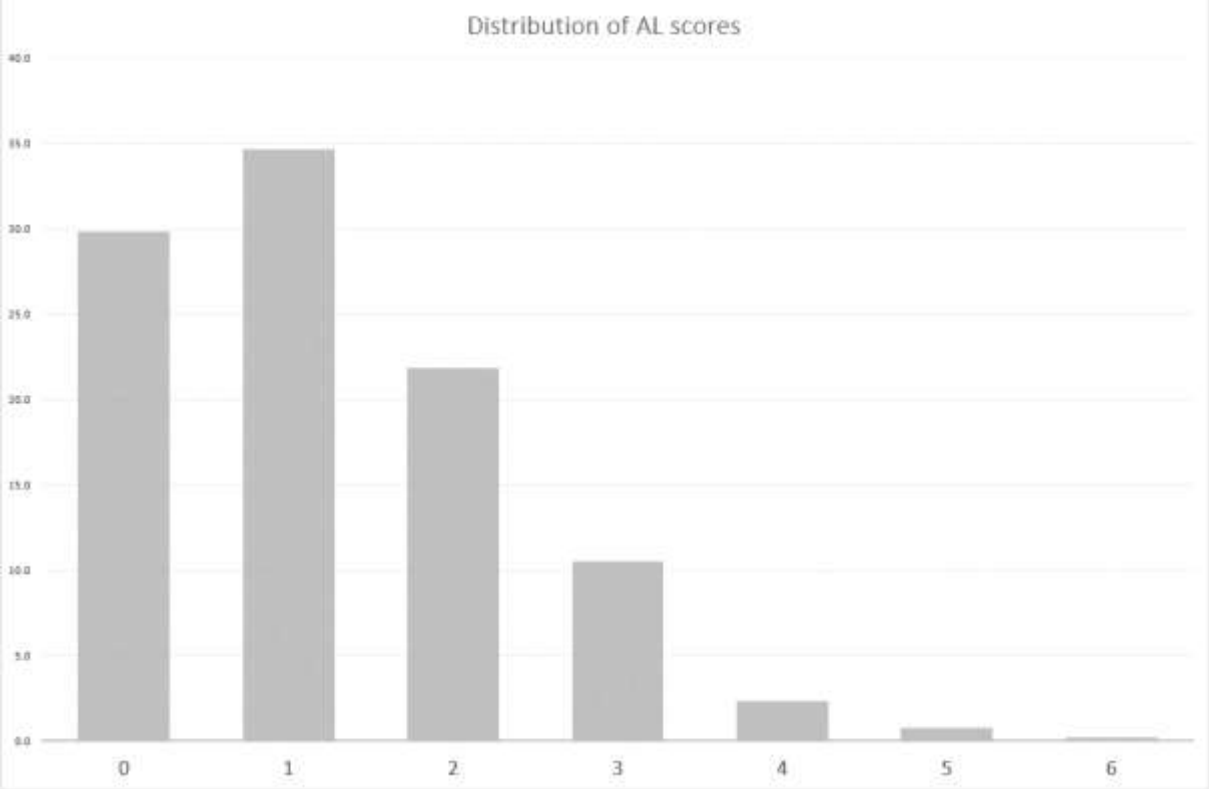
In order to examine the socio-economic gradient in AL, we retrieved the education level of the mother and of the father, as reported when the child was 1 year old. No information on continuing education was collected, however, given that the mean age of the mother at the time

of child birth was around 30, we do not believe that this will create bias in our education level measure.

In order to accommodate one of the undisputed features of AL, i.e., as a measure of multisystem dysregulation it should include biomarkers of the different physiological systems that are thought to be affected by chronic stress exposure, our dependent variable was calculated as a sum scale covering indicators measuring the metabolic, cardiovascular and inflammation systems. Based on the availability of measures, we selected the following biomarkers (associated physiological systems between parentheses): systolic and diastolic blood pressure (cardiovascular), total IgE (inflammation), HDL cholesterol to total cholesterol ratio (TC/HDLC ratio), Glycated haemoglobin (HbA1c), and BMI for sex and age standardized (metabolism). Information on the procedure and measurements protocol is presented elsewhere [24].

We followed recent research and calculated the AL index based on population distribution cut-off points that identify the health risk category (coded 1) followed by a simple summative operation [25]. For each of the 6 biomarkers we determined cut-off points that identified the health risk category as the highest quintile, a stricter cut-off point than the usual highest 25 percent used in the literature. We followed the work of Seeman and colleagues [14] and we did not define gender specific cut-off points because we wanted to compare genders based on a common definition. The AL measure was computed as a sum score that had a valid outcome when all 6 health risk variables had valid information (valid cases: 1193). The distribution of the AL scores is presented in Figure 1.

Figure 1. Distribution of allostatic load scores in the sample



Education of the mother and father differentiated between low (ref.cat.) (primary school, lower vocational or lower secondary education), intermediate (intermediate vocational education or intermediate/higher secondary education), and high (higher vocational education and university). The gender of the respondent differentiated between boys and girls (1 respondent who was classified as transgendered at a later follow-up was excluded from the analyses). As control variables we first accounted for the occupational status of the mother and father collected when the child was 1 year old and we differentiated between being employed (ref.cat.), self-employed, housewife(man), and other situations. Next, we accounted for housing quality, using the presence of mould/ dampness stains in the bathroom, living room, kitchen, parents' bedroom, or the child's bedroom as indicators of bad housing quality (ref.cat. is housing quality: good). Other control variables accounted for the child's general health in the past 12 months as reported by parents (excellent / good vs moderate / poor (ref. cat.)), the ethnic background (children whose parents were both born in The Netherlands, vs western / not western ethnic minority (ref.cat.)), premature birth (under 37 weeks of gestation), weight at birth (in grams), and smoking indoors when child was 11 years old (yes vs no (ref.cat.)). Table 1 summarizes descriptive information on all the variables in the analyses. After exclusion of participants with missing values on one or more of the covariates, our working sample had 1079 respondents.

TABLE 1. CHARACTERISTICS OF THE STUDY POPULATION (N=1079)

	Min / Max	Mean (%)	Sd.
AL	0/6	1.24	1.13
Education mother: low	0/1	17.7	
Education mother: intermediate	0/1	41.6	
Education mother: high	0/1	40.7	
Education father: low	0/1	19.8	
Education father: intermediate	0/1	33.0	
Education father: high	0/1	47.1	
Gender: boy	0/1	50.8	
Dutch	0/1	93.2	
Occupation mother: employed	0/1	65.3	
Occupation mother: self-employed	0/1	4.5	
Occupation mother: housewife	0/1	25.0	
Occupation mother: other	0/1	5.1	
Occupation father: employed	0/1	87.1	
Occupation father: self-employed	0/1	10.5	
Occupation father: houseman	0/1	0	
Occupation father: other	0/1	1.6	
Housing quality: good	0/1	67.7	
Child's health: good / excellent	0/1	98.4	
Premature birth	0/1	4.4	
Birth weight (grams)	1370 / 5000	3553	526
Indoor Smoking	0/1	16.7	

Note: figures calculated for the sample that participated in the medical examination, after listwise deletion of missing values.

We assessed the relationship between parental SEP and children's AL by employing a linear regression model. In order to accommodate the fact that the AL measure is skewed, we followed recent research and estimated all the effects with robust standard errors [26]. In addition, as a sensitivity analysis, we also checked the robustness of our results by estimating bootstrapped standard errors, as well as log normalizing the AL index and estimating an OLS regression. The results were similar and we only report the effects with robust standard errors. We also computed a measure of AL that accounted for the unequal number of biomarkers that cover each of the 3 physiological symptoms, but our results did not change.

We started by estimating the effects of the mother's and father's education separately and only controlled for gender and ethnicity (Model 1 and 2), then we added the education measures together in the same model (Model 3), and in the next step we added the remaining control variables (Model 4). We re-estimated Models 3 and 4 with a measure of education that recorded the highest level of education between the parents in order to address the effects of potential education inequality between the parents, i.e., it could be that fathers most often had the highest education in the couple (results available from authors). Furthermore, since the examination of gender differences is central to this study, we estimated Model 4: 1) by adding interaction terms with the education of the mother and father (Model 5 and 6) and 2) separately for girls and boys (Model 7(g) and 8(b)).

RESULTS

Most of the children (93%) in our study population had two parents who were born in the Netherlands; nearly all fathers and 70% of the mothers were (self)employed and more parents were highly educated than low educated. The mean AL score was 1.24 on the scale from 0 to 6. This figure indicates that the level of presumed physiological deregulation due to chronic stress exposure was low in our sample of 12 years old children (see Table 1).

TABLE 2. SELECTED REGRESSION COEFFICIENTS (ROBUST STANDARD ERRORS) FOR THE ASSOCIATION OF PARENTAL EDUCATION AND GENDER WITH AL (N: 1079)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7(g)	Model 8(b)
Constant	1.36 (.16)	1.42 (.16)	1.43 (.16)	1.85 (.38)	1.97 (.39)	1.84 (.38)	2.34 (.50)	1.5 (.56)
Education mother (low ref.)								
intermediate	-.09 (.10)		-.03 (.10)	-.02 (.10)	-.23 (.14)	-.02 (.10)	-.23 (.15)	.21 (.14)
high	-.14 (.10)		.04 (.11)	.08 (.11)	-.09 (.16)	-.08 (.11)	-.11 (.17)	.28 (.15)
Education father (low ref)								
intermediate		-.05 (.10)	-.05 (.11)	-.06 (.11)	-.05 (.11)	-.06 (.14)	.01 (.15)	-.12 (.15)
Education father: high		-.30 (.10)	-.32 (.11)	-.32 (.10)	-.31 (.11)	-.31 (.14)	-.22 (.16)	-.42 (.15)
Gender: boy	.17 (.07)	.17 (.07)	.17 (.07)	.16 (.07)	-.15 (.16)	-.17 (.16)		
Interactions								
boy* edu. mother: intermediate					.42 (.19)			
boy* edu. mother: high					.33 (.19)			
boy* edu. father: intermediate						-.00 (.20)		
boy* edu. father: high						-.03 (.19)		

Notes: Model 1, 2, and 3 controls for ethnic background, Model 4 controls for ethnic background, occupation of mother and father, housing quality, child's health, premature birth, birth weight, and indoor smoking. Model 5 and 6 add interactions between gender and education level of parents. In Model 7(g) and 8(b) we re-estimated Model 4 in the girls' and boys' sample, respectively

The results of our analyses are summarized in Table 2. From Model 1 to 4 we found that parental education was relevant, but only for the children of fathers with a high education level – they had a significant lower level of AL in comparison to their counterparts with fathers with a low level of education. This conclusion did not change when we included the education of the father and of the mother in the same model or when we accounted for all the individual-level control variables. When we re-estimated Model 4 with a measure of education that records the highest education level between the parents, we found that children in families where at least one of the parents was high educated had a significantly lower level of AL than children in families where parents were low educated (coef. -.23, p=.046). In addition, Model 1 to 4 also revealed that gender was relevant – boys had significantly higher mean level of AL than girls.

Is parental education relevant more for boys or for girls? Based on Models 5 to 8, we found that especially boys of high educated fathers had lower AL, however, the difference with the effect of high educated fathers for girls was not statistically significant (effect interaction -.03, p = .88). Regarding the effects of mother's education on AL for girls and boys, these were not significant (see Models 7(g) and 8(b)), but the interaction with gender was significant, suggesting that boys with mothers with intermediate education have significantly higher AL than girls whose mother have intermediate education.

DISCUSSION

In the present paper we set off to investigate the socio-economic gradient in AL and its role in shaping gendered inequalities in health during childhood. Based on the results of our analyses, the following main conclusions can be derived.

First, we argued that low parental SEP goes hand in hand with a higher probability of their children to be exposed to chronic stressors, resulting in the systemic physiological deregulation that is encompassed by the AL measure. Our results support this idea, even though the relationship between parental SEP and children's AL was found only for the education of

the father, i.e., the AL score of children with high educated fathers was around 5 percent lower than the score of children with low educated fathers. Longitudinal research in older samples has shown an increasing AL trend with age that was steeper when life course accumulation of risks was higher [27]. Our results suggest that the divergent trend in AL starts already during childhood. Future research employing a longitudinal design and measuring levels of AL throughout childhood through adulthood and old age could shed light on this so called "long arm of childhood" mechanism.

A possible explanation of why fathers's education mattered and not that of the mothers is the potentially higher involvement of the fathers with high education in the upbringing of their children, which could result in less marital stress and more supportive environment. However, due to the lack of measures in the data, this explanation remains speculative. Next, fathers' higher education can be linked to higher levels of income, while mothers' high education level may not translate into higher levels of income due to for instance part time work. Within the limits of the available measures we controlled for the level of material resources of the mother and father by including the occupation status, and the effects found for education were not explained away. Future research is needed in order to examine whether the effect of father's education on the children's level of AL is context specific and what are the explanations behind it.

Second, our results suggested that the beneficial health effect of having a high educated father was stronger for boys than for girls, however, the formal interaction term did not support this conclusion. It is uncertain whether this pattern hinted by our data becomes stronger in time. Regarding the education of the mother, our results did not reveal a clear pattern. From research following children to adulthood, we know that maternal education matters for the level of AL during adulthood of both men and women, but through different pathways [28]. Research is

warranted in order to understand how these effects and mechanisms unfold in time from childhood to adulthood and older age.

We also note that we found gender inequalities in health as measured by AL to exist at the age of 12, i.e., boys had significantly higher level of AL than girls. This finding reflects some results from adult samples [26], however, the gender differences in AL are not conclusive in all studies – most often men have higher level of AL than women, however, the differences are not always significant. Gendered health differences during adulthood reflect different normative trajectories in work and family domains that are followed by men and women [29], however, for children at the age of 12 these considerations do not apply. However, our results suggest gendered patterns of exposure to chronic stressors during youth. Future research focused on identifying the underlying stressors is warranted.

Our study has a few limitations, mainly related to the lack of more detailed measures of parental SEP. However, research has shown that in the Netherlands, education level is increasingly important for the attained occupational status and associated wealth in later life [30] and therefore it is a good measure of parental SEP. Next, selective loss-to-follow-up and/or refusal to participate in the medical examination could have biased our results. Exploration of non-response patterns showed that children of high educated fathers were more likely to participate. This implies that we have fewer children of low educated fathers in our working sample than there were in the PIAMA baseline population of n=3963. Given their low level of AL, our results are more likely underestimating the effect of the parental education on children's AL. And lastly, the data does not include biomarkers that cover all the physiological systems thought to be involved in the AL process. However, we expect this limited coverage of the spectrum of physiological systems to result in an underestimation of the level of AL, which would imply that our conclusions still hold.

In conclusion our study contributes to the understanding of the origins and development of health inequalities across the life course by showing that parental education of the father matters for boys and girls alike, i.e., AL score of children of fathers with a high education level was lower than the score of children of low educated fathers. Furthermore, our study also shows that gender health differences can be traced back to childhood, i.e., boys had higher levels of AL than girls.

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