

National Child Development Study

NDCDS

user support group

working paper 48

The Association of Slow Growth in
Childhood with Family Conflict

Scott Montgomery

City University & Royal Free Hospital, London

Mel J Bartley

City University & University College London

Richard G Wilkinson

University of Sussex

November 1996

Social Statistics Research Unit

City University * Northampton Square * London EC1V 0HB.

National Child Development Study User Support Group

Working Paper No: 48

**The Association of Slow Growth in Childhood
with Family Conflict**

Scott M Montgomery

University Department of Medicine, Royal Free Hospital School of Medicine & Social
Statistics Research Unit, The City University

Mel J Bartley

Department of Epidemiology and Public Health, University College London Medical School
& Social Statistics Research Unit, The City University

Richard G Wilkinson

The Trafford Centre For Medical Research, University of Sussex, Brighton

November 1996

**Social Statistics Research Unit
City University, Northampton Square, London EC1V 0HB**

Summary

Background - Having previously observed that slow growth in childhood was associated with subsequent labour market disadvantage, we have attempted to determine if family conflict and other markers of childhood adversity (which may have adverse psychological consequences) are associated with slow growth to age 7 years.

Methods - 6,574 males and females born between 3 and 9 March 1958, who were members of the British National Child Development Study (NCDS) were used in these analyses. Slow growth at age 7 years indicated by short stature defined as the lowest fifth of the height distribution.

Findings - 31.1% of children who had experienced family conflict were of short stature, compared with 20.2% of those who had not, representing relative odds of 1.79 (95% CI 1.39 - 2.30). After adjustment for social class, crowding, sex and (genetically) pre-determined height (using fully attained adult height) the relative odds were slightly reduced to 1.62 (95% CI 1.18 - 2.23). 44.0 % of children from the most crowded households were of short stature compared with 16.4% of those from the least crowded. The unadjusted relative odds were 3.99 (95% CI 2.94 - 5.41), and after adjustment for the potential confounding variables, they were 3.07 (95% CI 2.08 - 4.51). Low social class was also a risk for short stature at age 7 years, but this was not significant after adjustment for the other confounding factors.

Interpretation - Family conflict and household crowding during childhood were independently associated with slow growth to age 7 years.

Introduction

There have been reports of psychosocial and material influences on height and growth^{1 2 3}. We decided to investigate evidence of material and psychosocial circumstances on slow growth in childhood among members of the National Child Development Study (NCDS): the 1958 British birth cohort.

Longevity and upward social mobility are strongly and independently related to height, and height appears to be an important indicator of social variation in health⁴. The strength of the relationship between height and social mobility led to the heights of Civil Servants in the Whitehall Study being more closely related to achieved occupational grade than to their social class of origin⁵. It was once widely assumed that findings such as these indicated that height was a marker for some physiological advantage, and also that the tall were upwardly mobile perhaps because of a human tendency to be impressed by stature. However, these interpretations are unlikely to represent a full explanation, as it has been shown that unemployment in early adulthood, which is a good marker for social mobility, was much more closely related to slowed growth to age 7 years than to fully-attained adult height⁶. This suggested that some feature of the childhood environment may influence both early growth rate as well as labour market success (and therefore social mobility).

Methods

The data used were taken from the National Child Development Study (NCDS)^{7 8 9 10}, a continuing national longitudinal study of approximately 16,000 subjects living in Great Britain who were born between 3 and 9 March 1958. There have been six data collection sweeps at birth and at ages 7, 11, 16, 23, and 33 years. Due to sample attrition and exclusion of cases with incomplete data, these analyses were limited to 3169 males and 3405 females. The cohort has nevertheless remained largely representative although the most disadvantaged groups are under-represented¹¹.

Height at age 7 years was measured by school doctors to the nearest inch. The bottom fifth of the height distribution (estimated separately for males and females), containing the shortest children, was used as a binary variable in the analysis. Height at age 33 years was measured to the nearest centimetre by social research interviewers using portable stadiometers. Height at age 33 years was used as a continuous variable. A binary measure of family conflict was taken from a questionnaire completed by the cohort member's Health Visitor at age 7 years. The Health Visitor reported family difficulties due to domestic tension, divorce, separation or desertion. The Registrar-General's social class based on the father's occupation when the cohort member was 7 years of age was used to indicate socio-economic conditions. Cases were excluded where father's occupation was not recorded, including where the father was not resident in the household. Crowding was used as an indicator of social disadvantage¹² to provide a more accurate measure of standard of living¹³ than social class alone. The

measure of crowding was based on the number of persons per room, excluding kitchens and bathrooms, when the cohort members were age 7 years. This was divided into four categories: up to 1 persons per room (ppr); over 1 and up to 1.5 ppr; over 1.5 and up to 2 ppr; and over 2 ppr. The last category represents the greatest relative disadvantage.

All analyses were conducted using SPSS¹⁴. The relationship between family conflict, family circumstances and growth to age 7 years was examined by cross tabulation and multiple logistic regression. Height at age 7 years was divided at the bottom fifth to form a binary dependent variable; a similar measure was in previous analyses⁶. We adjusted for height at 33 years, modelled as a linear variable, in the multiple logistic regression model. This model was also adjusted for social class, crowding, and sex. All independent variables, with the exception of adult height at age 33 years, were modelled as binary dummies. Cases with incomplete data were excluded from the analysis.

Results

A total of 299 (4.5%) cohort members had been exposed to family conflict as indicated by the Health Visitors report. The height range at age 7 years for boys in the shortest fifth was 1.02m to 1.17m, with a mean of 1.15m. The range for other boys was 1.19m to 1.52m, with a mean of 1.25m. For short girls the range was 0.99m to 1.17m with a mean of 1.14m; for the taller girls the range was 1.19m to 1.55m with a mean of 1.24m. A total of 1358 (20.7%) boys and girls were in the short stature group.

Of children who experienced family conflict, 31.1% were in the bottom fifth of the height distribution at age 7 years, compared with 20.2% of those who didn't experience it. The relative odds of being in the lowest fifth of the height distribution amongst those who experienced family conflict were 1.79 (95% CI 1.39 - 2.30), when compared with those who had not experienced family conflict. After adjustment for the potential confounding variables, including adult height, the relative odds were slightly reduced to 1.62 (95% CI 1.18 - 2.23), indicating an independent relationship between slow growth and family conflict.

In social class V, 30.3% of children were in the short stature group, compared with 11.7% in class I, with relative odds of 3.29 (95% CI 2.25 - 4.82). After adjustment for the potential confounding variables the strength of this relationship was significantly diminished and the relative odds for short stature amongst children from social class V families were reduced to 1.45 (95% CI 0.90 - 2.32), when compared with children from class I. Thus, after adjustment, the relationship between social class and height at age 7 years was no longer statistically significant.

There was a clear gradient of association between crowding and short stature at age 7 years. 44.0% of those from the most crowded households were of short stature while only 16.4% of those from the least crowded households were in the shortest height fifth. The unadjusted relative odds for being in the bottom height fifth for those in the most crowded households were 3.99 (95% CI 2.94 - 5.41), when compared with those from the least crowded

households. Adjustment for the potential confounding variables slightly reduced these relative odds to 3.07 (95% CI 2.08 - 4.51).

Discussion

It has previously been observed that 7 year old boys with slowed growth were far more likely to experience adult unemployment, even after adjustment for socio-economic background, educational attainment and other risk factors for unemployment⁶. It is hypothesised that environmental factors, including family conflict, has dual consequences: slowing growth and also affecting psychological development. Whilst those with slowed growth in childhood do not necessarily become the shortest adults, their poorer psychological development may result in subsequent labour market and other disadvantage. This paper has focused on environmental influences, in the form of family conflict, on growth rate to age 7 years. We have attempted to adjust for genetically determined height (by adjusting for fully attained adult height) and thus focusing on slowed growth.

A child's height at age 7 years is the result of the interaction between genetic and environmental influences¹⁵. In a study of 7569 English and Scottish primary school children Smith *et al*¹⁶ found that the socio-economic differences in height are established in early childhood. However, it is known that only a relatively small proportion of those of short stature at age 7 years are of short stature as adults¹⁷, indicating significant variation in growth rates. Here, we are concerned with slowed growth as this may be a better indicator of environmental effects than persistent short stature. Our analysis is likely to have

underestimated the influence of family conflict and socio-economic circumstances on growth in childhood. This is because we adjusted for fully attained adult height: family conflict and socio-economic circumstances can also result in shorter fully-attained adult height¹⁸. This 'over-adjustment' was necessary to identify slowed growth rather than short stature which may have been determined genetically.

Our results show independent relationships between slower growth in childhood and family conflict and crowding. This confirms the finding from Swedish data, based only on retrospective recall, that height is related independently to economic hardship in childhood and to "family dissension"³. It is plausible that such influences are likely to influence emotional as well as physical development. These developmental consequences may themselves affect health and social mobility later in life.

The relationship between household crowding and the risk of being in the lowest fifth of the height distribution at age 7 is powerful and graded. This may be because crowding is a good indicator of low income and poor housing conditions and this is reflected in slower childhood growth. Frequent sleep disturbance may be another mechanism through which growth is affected in crowded households. Growth hormone is released during periods of deep sleep and its secretion rate would fall if sleep were disrupted¹⁹. The secretion of other hormones required for normal physical development is also stimulated by growth hormone²⁰. Conditions that are more likely to result in disturbed sleep, such as excessive noise, shared rooms, or beds, would reduce growth hormone secretion and may result in slowed growth.

Family conflict during childhood has been shown to be a risk factor for subsequent psychological ill health²¹ and it has also been suggested that slow growth in infancy may be associated with adult depression²². Factors affecting growth rate may influence future physical health, as slow growth in infancy has been associated with increased risk of adult coronary heart disease²³. Psychological stress may be responsible for these processes as this can result in raised levels of the endogenous opioid, beta-endorphin²⁴, which has been shown to modulate growth hormone release through the mediation of growth hormone releasing hormone (GHRH) in pre-pubertal children²⁵. Chronic levels of stress in children resulting from persistent family conflict may influence growth through the action of beta-endorphin or other growth factor modulators.

Adverse childhood conditions may have dual risks for both poor future health and persistent socio-economic disadvantage, which is itself a risk factor for poor health. Domestic conflict is known to be associated with a number of adverse long-term developmental, physical health, psychological^{26 27 28} and labour market⁶ outcomes.

Conclusion

Slow growth in childhood was associated with family conflict and this was independent of socio-economic circumstances. This may be because slow growth is a sensitive marker of emotional disturbance and chronic stress in childhood. Household crowding was also found to be strongly associated with slower growth, and this may be because it measures

disadvantage as well as possibly having a more direct influence on growth through the disrupted sleep mechanism. By controlling for sex and fully attained adult height we have attempted to adjust for pre-determined genetic influences on height at age 7 years. The association of slow growth in childhood with labour market disadvantage and poorer health in later life may be due to childhood psychosocial and material adversity resulting in poorer physical and psychological development.

Slow growth at age 7 years indicated by the shortest fifth of the height distribution. Relative odds are adjusted for domestic conflict, social class, crowding, sex and adult height.

	n	%	UNADJUSTED			ADJUSTED		
			Relative Odds	95% CI	Sig.	Relative Odds	95% CI	Sig.
Family Conflict								
No	6275	20.2	1.00			1.00		
Yes	299	31.1	1.79	1.39 - 2.30		1.62	1.18 - 2.23	
					0.00000			0.1100
Social Class								
I	403	11.7	1.00			1.00		
II	1050	14.2	1.25	0.88 - 1.77		1.15	0.76 - 1.73	
III _{nm}	705	16.7	1.52	1.05 - 2.18		1.29	0.84 - 1.98	
III _m	2943	23.5	2.32	1.69 - 3.19		1.48	1.02 - 2.16	
IV	1127	21.9	2.12	1.52 - 2.97		1.25	0.84 - 1.88	
V	346	30.3	3.29	2.25 - 4.82		1.45	0.90 - 2.32	
					0.00000			0.0000
Crowding								
Up to 1 pers/room	4020	16.4	1.00			1.00		
over 1 to 1.5 pers/room	1741	22.9	1.51	1.31 - 1.73		1.36	1.14 - 1.62	
over 1.5 to 2 pers/room	631	34.9	2.72	2.27 - 3.27		2.44	1.93 - 3.09	
over 2 pers/room	182	44.0	3.99	2.94 - 5.41		3.07	2.08 - 4.51	

n = 6,574

References

1. Widdowson EM. Mental contentment and physical growth *Lancet* 1951; June 16: 1316-18.
2. Power C, Manor O. Asthma, enuresis, and chronic illness - long term impact on height. *Archives of Diseases in Childhood* 1995; 73 (4): 298-304.
3. Nystrom Peck M, Lundberg O. Short stature as an effect of economic and social conditions in childhood. *Social Science and Medicine* 1995; 41: 733-8.
4. Marmot MG, Shipley MJ, Rose G. Inequalities in death - specific explanations of a general pattern. *Lancet* 1984; 1 (8384): 1003-6.
5. Marmot MG. Social inequalities in mortality: the social environment. In: *Class and health: research and longitudinal data*. edited by RG Wilkinson Tavistock, London: 1986.
6. Montgomery SM, Bartley MJ, Cook DG. & Wadsworth MEJ. Health and Social Precursors of Unemployment in Young Men in Great Britain. *Journal of Epidemiology and Community Health* 1996; 50: 415-422.
7. Shepherd P. The National Child Development Study: An Introduction to the Background to the Study and the Methods of Data Collection 1985. NCDS Working Paper No. 1, Social Statistics Research Unit, City University, London.
8. Ferri E (Ed). *Life at 33*. London: National Children's Bureau, 1993.
9. Butler N. R. and Bonham, D. G. *Perinatal Mortality*, Livingstone, Edinburgh, 1963.
10. Butler N. R. and Alberman, E. D. (eds) *Perinatal Problems*, Livingstone, Edinburgh, 1969.
11. Power C., Manor O. and Fox AJ. *Health and Class: The Early Years*. Chapman and Hall, London, 1991.
12. Essen J., Fogelman K. and Head J. Children's Housing and their Health and Physical Development. *Child: Health, Care and development*. 4: 357-369, 1978.
13. Goldblatt PO. Mortality and Alternative Social Classifications. In: Goldblatt, P. (Ed.) *Longitudinal Study: Mortality and Social Organisation*. HMSO: 163-92, London, 1990.
14. Norusis MJ. *SPSS User's Guide*. SPSS Inc., Chicago, 1990.

15. Eveleth PB, and Tanner JM. *Worldwide Variation in Human Growth*. Cambridge, 1976: 222.
16. Smith AM, Chinn S, Rona RJ. Social factors and height gain of primary schoolchildren in England and Scotland *Annals of Human Biology* 1980; 7: 115-24.
17. Greco L, Power C. and Peckham C. Adult Outcome of Normal Children Who are Short or Underweight at Age 7 Years. *BMJ* 1995; **310**: 696-700.
18. Peck MN, and Lundberg O. Short Stature as an Effect of Economic and Social Conditions in Childhood. *Soc. Sci. Med.* 1995; **41**, 5: 733-738.
19. Preece M. A. Prepubertal and Pubertal Endocrinology. In: *Human Growth*, 2nd edn., Vol. 2. Edited by Falkner J. and Tanner J. M. Plenum Press, London, 1985.
20. Preece M. A. and Holder A. T. The Somatomedins: A Family of Serum Growth Factors. In: *Recent Advances in Endocrinology and Metabolism*, Vol. 2. Edited by O'Riordan J. L. H. Churchill Livingstone, Edinburgh, 1982.
21. Wadsworth MEJ. Early stress and associations with adult health behaviour and parenting. in Butler NR, and Corner BD. (eds) *Stress and Disability in Childhood*. John Wright and Sons, Bristol, 1984: 100-104.
22. Barker DJP, Osmond C. Rodin I, Fall CHD, and Winter PH. Low weight gain in infancy and suicide in adult life. *BMJ* 1995; **311**: 1203.
23. Fall CHD, Vijavakumar M, Barker DJP, Osmond C, and Duggleby S. Weight in infancy and prevalence of coronary heart disease in adult life. *BMJ* 1995; **310**: 17-19.
24. Schedlowski M, Fluge T, Richter S, Tewes U, Schmidt RE, and Wagner TOF. Beta-endorphin, but not substance-p, is increased by acute stress in humans. *Psychoneuroendocrinology*, 1995; 20, No. 1: 103-110.
25. Pugliese MT, Abdenur J, Fort P, and Lifshitz F. The relationship between beta-endorphin and the growth-hormone (GH) response to GH releasing hormone in prepubertal children. *Endocrine Research*, 1992, **18** no 1: 41-50.
26. Wadsworth M, Maclean M, Kuh D, Rodgers B. Children of divorced and separated parents: summary and review of findings from a long-term follow-up study in the UK. *Family Practice* 1990; 7: 104-9.

27. Wadsworth MEJ. Early stress and associations with adult health, behaviour and parenting; in: Stress and disability in childhood. Edited by Butler NR, Corner BD. Wright, Bristol 1984.

28. James O. Juvenile violence in a winner-looser culture. Free Association Books 1995.