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Socioeconomic position in childhood and cancer in adulthood: a rapid-review

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ABSTRACT

Background The relationship of childhood socioeconomic position (SEP) to adult cancer has been inconsistent in the literature and there has been no review summarising the current evidence focused solely on cancer outcomes.

Methods and results We performed a rapid review of the literature, which identified 22 publications from 13 studies, primarily in the UK and northern European countries that specifically analysed individual measures of SEP in childhood and cancer outcomes in adulthood. Most of these studies adjusted for adult SEP as a critical mediator of the relationship of interest.

Conclusions Results confirm that childhood socioeconomic circumstances have a strong influence on stomach cancer and are likely to contribute, along with adult circumstances, to lung cancer through cumulative exposure to smoking. There was also some evidence of increased risk of colorectal, liver, cervical and pancreatic cancers with lower childhood SEP in large studies, but small numbers of cancer deaths made these estimates imprecise. Gaps in knowledge and potential policy implications are presented.

INTRODUCTION

Substantial evidence supports the notion that adult chronic diseases are not determined solely by exposures and events in adult life.^{1–3} Beyond genetic susceptibility, exposures and the social circumstances of early life begin a process that extends throughout the lifespan to influence adult disease. Epidemiologists take a life-course approach to the study of physical and social hazards during gestation, childhood, adolescence, young adulthood and midlife that can affect adult chronic disease risk and health outcomes. This well-established approach aims to identify the underlying biological, behavioural and psychosocial processes that operate across the lifespan.^{2–4} Aetiological factors may act during critical periods of development, with or without additional later life influences, or they may act through the accumulation of risk through various pathways.^{5–6} This approach focuses our attention on the importance of the early environment on human biological and psychological development and on the timing of a range of exposures during this critical period, including those associated with adverse socioeconomic circumstances.⁴

There is evidence that social determinants can confer a disadvantage, starting before birth and accumulating throughout life.³ For example, social disadvantage may lead to the adoption of suboptimal diets or physical activity patterns that may persist into adulthood.⁷ Poor education and learning opportunities in childhood may lead to

cognitive deficits⁸ and lower socioeconomic position in adulthood,⁹ which is in turn associated with most forms of adult ill health.³

Substantial epidemiological evidence supports the view that circumstances associated with adverse socioeconomic position (SEP) early in life have effects on overall adult morbidity and mortality^{10–12} and especially on cardiovascular disease.^{13–15} For adult cancers, however, the evidence for meaningful associations is inconsistent. Better evidence is needed to clarify the relationship of early life disadvantage with adult cancers, in order to develop interventions to prevent cancer and reduce inequities in cancer outcomes. Better understanding is needed of the pathways through which social determinants in early life influence behaviour or have direct effects on cancer incidence and mortality.

To understand the current evidence in this area, we performed a rapid review of the literature on the association of early life SEP and adult cancer incidence and cancer mortality. Specifically, we sought to summarise what is currently known, the quality and the strength of the evidence and limitations to further exploration of this relationship. Our work draws on previous systematic reviews of childhood SEP and all major adult health outcomes,^{16–17} but focuses only on cancer outcomes. The review did not try to capture the literature on the relationship of childhood SEP with behaviours (eg, dietary, alcohol consumption, physical activity and smoking) that could mediate the relationship.

METHODS/LITERATURE SEARCH

Rapid reviews are a streamlined approach to synthesising evidence for decision-making and we sought information to assist in defining new directions for cancer prevention research initiatives.¹⁸ For this review, the PubMed database was used to search for relevant literature (<http://www.ncbi.nlm.nih.gov/pubmed>). We used the PRISMA guidelines to document our search strategy.¹⁹ The search terms used were ‘Child* AND Socio* AND Cancer Incidence’ (publication date 1994–present day), which yielded 1712 publications. ‘Child’ captured articles with children up to age 19 years. The inclusion of ‘George Davey Smith’, whose work with colleagues in this area has been extensive, netted an additional 725 publications (no publication date restrictions were used) for a total of 2437 publications. Duplicates were removed and the final list of titles was screened for relevance.

When screening the titles, those that did not consider socioeconomic factors mentioned these factors without alluding to any cancer outcome, cause-specific mortality or other diseases or behaviours related to cancer (eg, smoking) were



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excluded and the abstracts of the remaining papers, all in English (n=84), were reviewed. When reviewing the shortlisted abstracts, those that indicated that the paper did not discuss socioeconomic factors in childhood or over a life course were excluded together with papers that did not mention cancer outcomes. Full-text articles were obtained for those papers considered to be relevant (n=40). At this stage, a further review was conducted of the content. Papers were included where there was specific mention of socioeconomic factors at the individual level (not ecological correlations) over a life period and the effect on cancer with or without other health outcomes in adult life was assessed. Twenty-two papers from 13 studies met all the inclusion criteria for the review (see online supplementary appendix for the PRISMA flow diagram).

Information from these 22 papers was extracted and placed in a table (see online supplementary appendix table 1) showing key study design highlights. A second table summarised the main findings (see online supplementary appendix table 2). The papers were grouped by study and the country in which it was conducted. The research design of the studies varied significantly, meaning that it was not possible to conduct a meta-analysis.²⁰ We thus provide a narrative synthesis of the findings.

RESULTS

The rapid review was based on 22 peer-reviewed articles from 13 studies that came from studies of European and North American populations. We grouped them by region first from UK, then Norway and Sweden and finally by individual studies from Finland, Denmark, the Netherlands, France and the USA (see online supplementary appendix, table 1). The earliest publication date among them was 1996 and the most recent date was 2012.¹⁷

Study designs

A number of study designs were used, of which the most common were cohort studies constructed with retrospectively collected data on childhood circumstances, although several studies in Scandinavian countries were cohorts created with linkages of population registries to census data that contained information on childhood SEP, usually the father's occupation.^{11 21–24} One Danish study used adoption records to compare the effects of biological and adoptive father's occupation with adult cancer.²⁵ There was one early case-control study.²⁶ Most studies focused on both men and women, but three included only women^{12 27 28} and three only men.^{15 24 26} Entry for cohort studies was at times already during adult life (eg, university students¹⁴) and at other times at birth or early in the life when SEP was determined from parents or linkage with various administrative or other data sources at the time. Follow-up to incidence or mortality was accomplished through linkage with cancer registries or other national health databases.¹² The most common statistical models for cohort analyses were Cox proportional hazard models.

Most of the studies recruited individuals in the 1940s and 1950s, although some were initiated earlier in the century, such as the Boyd Orr cohort,^{29 30} and a few younger cohorts have been initiated since then.^{22 24 31}

Measurement of SEP

The measurement of SEP in these cohort studies was usually taken from a self-report of the study participant as part of a survey at entry to the study. However, in other studies, SEP was assessed in childhood through a national health database and

linked to mortality outcomes determined from national death indices.²² The most common measure of SEP in childhood was father's occupation, used by 18 of the 22 papers we located for review. Of the four that did not use father's occupation, one Finnish study used occupation of the head of the household²⁴ and the three others papers from the same study in Norway used various measures of living conditions during childhood.^{11 21 23} Other measures included parenteral education, either father or mother, number of siblings,³² overcrowding (number of persons per room), car ownership or maternal marital status. Rarely were there data on family or household income.^{23 33} This information was usually collected at the time of a baseline interview from the participating adult or from linking to population registry data.³⁴ Adult SEP, when ascertained, was usually based on the occupation, education or deprivation level of the study participant as an adult or of the main wage-earner in the family. In other studies reviewed but not included, education was used to reflect childhood SEP,³⁵ but previous research has pointed out that its meaning is ambiguous, since it may capture aspects of both childhood and adult SEP.^{16 36}

Other variables

In addition to SEP, the included studies collected data on a range of participant characteristics. Some studies included health behaviours like tobacco use, alcohol consumption and physical activity, physical examination data on body mass index, height and weight, blood pressure, lung function, psychosocial measures of stress and laboratory measurement of blood lipids. However, the inclusion of such variables was far from consistent and did not lend itself to separate analysis in this review. Results for childhood SEP and cancer were usually reported as unadjusted for adult cancer, adjusted for adult SEP and adjusted for adult SEP with covariates such as those listed above.³⁶

Cancer incidence and mortality

Most studies used cancer mortality from national death indices as their outcomes, although three drew on cancer incidence data^{26 28 31} and one used life expectancy.³³ The results of most studies focused on either overall cancer mortality or site-specific cancer mortality for the most common cancers, which were of the lung, breast, colon and rectum, prostate and stomach. Larger studies published more recently were able to assess the relationship of childhood SEP with less common cancers like liver, pancreas, cervical, melanoma, brain, lymphoma and leukaemias, but seldom with sufficient numbers to precisely estimate risks.^{22 23 31 37}

Overall results

These studies documented that individuals experiencing poorer socioeconomic circumstances during childhood carry a higher risk of overall mortality, independently of adult socioeconomic position.^{16 17} This was documented for cohorts in the UK, northern Europe and the USA. Interestingly, increased mortality after childhood deprivation was also observed in some younger cohorts,^{22 31} where it might be expected that these cohorts' experienced better conditions during childhood than previous generations despite the shorter follow-up resulting in fewer deaths. The risk associated with lower childhood socioeconomic position was, not surprisingly, partly mediated by adult socioeconomic position and adult risk factors³⁶ and most recent studies have adjusted for adult SEP in their analyses.

In relation to cancer, 9 of 11 studies found no significant relationship between low childhood SEP as measured by father's or

head of household's occupation and a higher risk of total cancer mortality (see online supplementary appendix table 2),^{24 31 38} although there were two exceptions.^{22 37} In seven analyses that only categorised cancer outcomes as either smoking or non-smoking related, only the smoking-related cancers were related to childhood SEP.^{10 21 24 25 33 38 39} There was no association between childhood socioeconomic characteristics and later death from non-smoking-related cancers in four analyses of two studies from the Oslo Mortality Study and the Boyd Orr Cohort in England and Scotland.^{21 23 33 34}

However, for specific cancers, some of which were non-smoking related, there were associations with a variety of sites with different degrees of strength and statistical significance. Thirteen analyses reported on lung cancer mortality and childhood SEP, as measured by fathers' occupations, more siblings or exposure to worse housing conditions during childhood (see online supplementary appendix table 2), and all but two^{30 31} showed elevated risks, which were attenuated by adjustment for adult socioeconomic circumstances. Stomach cancer was also consistently related to childhood SEP but independent of adult circumstances in eight analyses.^{11 12 14 22 23 32 36 40} Large studies in Norway and the Netherlands found a higher risk of large bowel and rectal cancer among those who had the poorest housing conditions during childhood or fathers of lower occupational status, partially mediated by adult SEP,^{11 31 40} but no relationship to colorectal cancer was found from a study in England and Scotland.¹⁴ Mortality was not elevated from prostate cancer or malignant melanoma in a large study from Norway.²³ Breast cancer incidence would be expected to be less common in lower SEP children as they reach adulthood, but there is evidence from one study that breast cancer mortality is higher in lower childhood SEP groups.²⁸ Liver cancer was significantly associated with lower childhood SEP independent of adult SEP in the only study where it was examined.²² The larger studies in Norway, Sweden and the Netherlands were able to analyse less common cancers like those of the pancreas, cervix, ovary and brain, lymphoma and leukaemia, but most of these estimates were imprecise given the small numbers of cases. Exceptions were cancer of the cervix showing a significant 77% increased risk with lower childhood SEP in Norway⁴⁰ and pancreatic cancer with a significant 23% increase in the large Swedish study.²²

In summary, childhood socioeconomic circumstances have a strong influence on stomach cancer and are most likely to contribute, along with adult circumstances, to lung cancer through cumulative exposure to smoking. There was some evidence of a relationship with colorectal cancer in two studies and with cancers of the liver, cervix and pancreas in single large studies. With the exception of breast and melanoma cancers, which are inversely related to SEP in childhood, other cancers tended to suggest direct relationships with low early life SEP, but small numbers of cancer deaths made these estimates imprecise.

DISCUSSION

Main findings

This rapid review of the available literature up until 2014 was focused on epidemiological studies of large cohorts in Europe and North America. Despite their generally large size, the numbers of cancer outcomes in most studies were limited. Thus, precise estimates of risk could only be made for the most common cancers or those for which the relationship to childhood SEP was especially strong. Suggestive findings for lower incidence cancers from some of the larger, more recent studies have not been confirmed. When all cancers were considered

together, the relationship to childhood SEP tended to be weak or non-existent. Given that total cancer mortality includes a combination of diseases resulting from very different aetiological processes, some associated with high SEP and others with low SEP, this result should not be surprising.

The four most common cancers in these countries are those of the breast, prostate and lung and colorectal cancers, which together account for about 48% of all cancer deaths.^{41 42} Other cancer outcomes examined in the largest cohorts in this review^{22 40} were stomach, cervical, liver, pancreas, brain, lymphomas, leukaemias, and melanoma cancers. Consistent with past reviews, childhood SEP, usually measured by father's occupation, was related in most studies to lung cancer mortality in adulthood, although attenuated by adjustment for adult SEP.^{16 17}

Stomach cancer mortality was also related to childhood SEP and unaffected by control of adult SEP as has been documented previously.^{16 17} Colorectal cancer mortality was reported to be related to childhood SEP in three studies.^{14 23 31} Breast cancer mortality was inconsistently related to childhood SEP measured by father's occupation, with some studies finding lower childhood SEP to be associated non-significantly with lower mortality^{12 40} and one with higher mortality³⁷ or no relationship.³¹ One study in the USA found that mother's education was related to increased breast cancer incidence, while father's education was significantly associated with decreased breast cancer mortality.²⁸ Prostate cancer associations were also inconsistent.^{14 23 31}

Biological mechanisms and their social determinants

Mortality was the most common outcome because it is the most available. Since mortality is related both to incidence and additional factors associated with access to and the quality of care introduced by the healthcare system, it is difficult to differentiate from mortality data the factors responsible for the onset of cancer (ie, causation) and those additional factors associated with survival once a cancer is diagnosed and treated.

Lung cancer was related more to adult SEP than childhood SEP, suggesting that smoking behaviour associated with SEP in adult life is the more proximal cause. However, given the residual influence of childhood SEP, the mechanism for lung cancer may still be related to the early establishment of smoking behaviour in lower income families. Smoking is heavily socially patterned in the developed countries where these studies took place and research in the UK has illustrated that children whose parents smoke are three times more likely to become smokers themselves. Children who grow up in less affluent households are more likely to have smoking parents and thus become smokers.⁴³ Research on the accumulated influence of SEP on the smoking behaviour of individuals over the life course is important in enhancing our understanding of these links between childhood circumstances and health behaviour later in life.¹⁶

For stomach cancer, the mechanism is likely to be a more direct effect related to exposure to and acquisition of *Helicobacter pylori* infection in less advantaged families during childhood.^{12 14 22 23} This is consistent with the absence of any attenuated effect of adjusting for adult SEP. Breast cancer tends to be associated with higher adult SEP and associated reproductive factors like earlier age of menarche, later age of menopause, later age at first pregnancy and lower parity for breast cancer, all of which are associated with the higher status of father's occupation in childhood.⁴⁴ A possible mechanism for colorectal cancer could be the relationship of childhood SEP to dietary

practices and healthcare screening access issues associated with colorectal cancer mortality.

In their first review of the literature, Galobardes *et al*^{16 17} noted that there was 'relatively little investigation of *how* early life circumstances influence adult health'. Adverse conditions in infancy and childhood can influence health in later life directly (latent effects), or cumulatively, or by their influence on subsequent life course pathways.^{4 33} However, adult SEP is closely related to that in childhood, suggesting strong socioeconomic pathways.¹² Power *et al* provide evidence on representative data for six countries linking child and adult socioeconomic status to health risk behaviours such as smoking and obesity. The associations of adult SEP with cancer-related health behaviours like tobacco use, alcohol consumption, diet, reproductive and sexual behaviour have long been appreciated.⁴⁵ More recently, the various possible mechanisms by which SEP may influence cancer-related behaviour have been explored in more detail⁴⁶ and studies have suggested that they may explain a substantial proportion of all-cause mortality associated with SEP.⁴⁷ Taken together, these studies suggest that childhood conditions influence subsequent health risk directly as well as through their influence on socioeconomic status and health behaviours later in life.³³

Gaps in knowledge and research needed

This review suggests a number of steps to be taken to further our understanding of the role of childhood SEP on adult cancer.

The first is that it may be time to reanalyse some of the large studies that have contributed so far to this literature. It has been 6 years since the last analyses of some of the larger data sets. Additional deaths will have accumulated permitting more precise measurement of effects for common cancers and the beginnings of better estimates for the less common sites. Questions remain about whether childhood SEP has substantial effects beyond those of lung and stomach cancer. Additional data on the other major cancers will help clarify the nature and extent of the influence of childhood SEP on adult cancer.

Second, consideration should be given to establishing a consortium of large cohorts with appropriate data that could be harmonised to produce even larger sample sizes. This type of consortium has been established in genetic epidemiology and has produced insights into important common variants for the most prevalent cancer sites.⁴⁸ Such existing large consortia provide models for the logistics, governance, procedures and protocols. However, more so than for genetic studies, harmonisation of life course cohorts across countries, time periods, measures of SEP and other factors (including, importantly, good measurement of health behaviours and changes through time of these behaviours) would present substantial challenges and strong institutional support would be needed.

Third, additional analytic efforts are needed that categorise cancer sites into useful groupings for understanding the potential role of childhood SEP. One category is obviously whether the cancer is related to tobacco smoking behaviour. However, additional questions remain about the role of other behaviours that may be established in childhood related to SEP such as dietary practices, physical activity and sunlight exposure. Still other relatively common cancers are strongly associated with infections like *H. pylori* for stomach cancer, hepatitis B for liver cancer and human papillomavirus for cervical and oropharyngeal cancers. Other cancer sites have unclear aetiologies and any relationship to childhood SEP may provide clues to their origins (eg, pancreas, brain gliomas and lymphomas).

Fourth, further studies are needed on how to assess SEP or what aspects of SEP need to be measured in childhood in order to understand better how it affects adult cancer. Is it material wealth and associated access to goods, education and housing, or is it the stress of disadvantaged environments that leads to suboptimal cognitive functioning, coping skills or both?⁴⁹ Also, more work needs to be carried out to understand the relationship of childhood to adult SEP and the various trajectories up and down the social ladder over the life course.^{10 24} How much can we expect to improve outcomes with a focus on improving childhood factors related to SEP?

Finally, what are the mechanisms by which childhood SEP influences adult cancer risk? How do the social determinants of health act to result in higher cancer mortality for specific cancers? More broadly, how do social determinants 'get under the skin'?⁵⁰ The lifecourse perspective gives us some guidance in tackling this question by asking whether there are direct (latent) effects (eg, *H. pylori* for stomach cancer) or accumulated exposures (eg, tobacco use and lung cancer).⁴ Considering in more depth the possible mechanisms by which low SEP can influence cancer-related behaviours is also needed.⁴⁶

One of the mechanisms that may be relevant here is the association of lower SEP with less development of successful 'executive functioning' in childhood. Executive functioning occurs in the prefrontal cortex and theoretically controls one's ability to counter the negative influences of environments that encourage poor health behaviour, like smoking, overeating, excessive consumption of alcohol and physical inactivity.⁸ In turn, there is evidence that the social clustering of these behaviours can explain much of the life expectancy differences between the most deprived and the most advantaged sectors of society.⁵¹ The strength of this control network appears to be linked to early environmental conditions and more advantaged populations.⁸ Although there are as yet no data to link weak 'executive control' to the onset of adult chronic disease and cancer in particular, the modulation of behaviours known to mediate cancer risk is an important area for further study. There may well be possibilities for interventions in early development that could strengthen the innate capacity of self-control to the benefit of reducing cancer risk and an array of other adult chronic diseases.⁵²

The role of genetics in the relationship between childhood SEP and adult cancer seems remote, but our evolving understanding of how epigenetic mechanisms can influence gene expression and resulting developmental phenotypes suggests an additional potential mechanism. The field of behavioural epigenetics is still experimental⁵³ but can potentially explain how suppression of gene expression, by methylation, for example, may be influenced by diet and other environmental factors including the stresses associated with disadvantage itself.⁵⁴ This, in turn, may produce individual differences in behaviours that could lead to cancer in adult life.⁵⁵ This notion, which is being studied intensely, opens up thinking about how the influence of genetics may not be determined solely by inheritance or somatic mutation, but by modifiable environmental influences.⁵⁴ Thus, the opportunity to consider interventions to enhance environmental and social conditions may also have application in understanding the relationship of childhood SEP and adult cancer.^{55 56}

Potential policy implications

Stronger evidence that childhood SEP is related to cancer incidence and mortality in adulthood would add to what is already known about its relationship to other adult chronic non-communicable

diseases like heart disease and diabetes and advance arguments for policy steps to support early childhood development. It is well documented that measures like life expectancy, disability-free life expectancy and age-standardised mortality increase in a gradient with measures of social class.³ Research directed at expanding the evidence for cancer outcomes across the social gradient is needed to bolster these findings.

Policies to improve environments to make healthy behaviours 'easier' (such as reducing the supply or availability of tobacco and alcohol, increasing the price and controlling the marketing of unhealthy products) are clearly important for cancer prevention both for children and adults. These need to be combined with individual level interventions (such as smoking cessation services and support to improve diets or increase physical activity) for cancer prevention in adults.⁵⁷ However, policy may also need to extend past what we currently can recommend and extend to improving the circumstances of early life development.

Childhood development starts from the womb and continues through the early preschool years into early schooling. Children, given the opportunity for healthy options, strong family and social supports, will develop cognitive skills and executive functions that lead to healthier behaviours that will most likely reduce their risk of cancer in adulthood. It is a much longer term strategy than improving mammogram rates and reducing tobacco use, but needs to be considered alongside existing frameworks for cancer prevention. Such a societal strategy is a sound investment in the future and likely to have a profound effect on the population burden of cancer as well as other chronic diseases common in industrialised societies.

Specific priorities for enhancing early development have been advanced by the Marmot Review, *Fair Society and Health Lives*,³ and include priorities to reduce inequalities in the early development of physical and emotional health, ensuring high-quality maternity services, parenting programmes and early education, and building resilience skills in the young. A research programme is needed to examine more deeply the impact of

policies and practices of this sort on learning skills and early behaviours in young people across the social gradient. Evidence already exists that behaviours related to tobacco and alcohol use, diet and physical activity and sun and workplace and environmental exposures are responsible for upwards of 40% of cancer incidence.^{58–59} Policy steps to improve early life development could translate into behaviours that influence cancer incidence and mortality in adulthood.

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What is already known on this subject

Substantial epidemiological evidence supports the view that circumstances associated with adverse socioeconomic position (SEP) in early life have effects on overall adult morbidity and mortality, especially for cardiovascular disease. The evidence for meaningful associations with adult cancer is inconsistent.

What this study adds

This rapid review updates the literature on the relationship of childhood SEP and adult cancer and finds consistent evidence for lung cancer mortality, which is attenuated by adult SEP and for stomach cancer mortality. Weak, inconsistent or no relationships were found for colorectal, prostate, breast, liver, pancreatic and other cancers. Overall, this review provides additional suggestive evidence of the influence of childhood social circumstances on adult cancer and identifies gaps in need of future research.

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