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Will our children be healthy adults? Applying science to public health policy

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ABSTRACT – Cardiovascular disease is predicted to be a leading cause of death and disability worldwide for the foreseeable future. Observational studies link a variety of prevalent early life experiences (for example, smoking in pregnancy, child poverty) to increased risk of adult cardiovascular disease. Experimental animal studies suggest plausible causal relationships. However, there has been little consideration of how to use this wealth of information to benefit children's futures. Policy documents have drawn on research evidence to recognise that early experience influences life chances, the development of human capital, and long-term health. This has led to a general policy emphasis on prevention and early intervention. To date, there are few examples of the evidence base being useful in shaping specific policies, despite potential to do so, and some examples of policy misunderstanding of science. Minor changes to the perspectives of epidemiological research in this area might greatly increase the potential for evidence-based policy.

KEY WORDS: cardiovascular disease, early life, public health, public policy

Introduction

This paper considers what public health science can contribute in helping the state achieve the global aspiration for a healthy next generation. Recent figures predict the top 10 causes of death worldwide for the year 2030.[1] For developed countries, the top five causes are ischaemic heart disease, cerebrovascular disease, lung cancer, diabetes mellitus and chronic obstructive pulmonary disease. These are also in the top 10 causes of life lived with disability in developed countries and, with the exception of lung cancer, in the top 10 causes of deaths in developing countries. Thus, at a population level, these diseases will determine if our children will be healthy, long-lived adults both in the UK and elsewhere.

This paper will focus on cardiovascular disease, both ischemic heart disease and cerebrovascular diseases, and their risk factors, for illustration. Cardiovascular disease is predicted to be the leading cause of deaths and a leading cause of lost disability adjusted life years worldwide for the foreseeable future.[1]

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Evidence linking early life to adult cardiovascular disease

Epidemiological studies have taken various approaches to studying links between early life and later health. A large set of studies, primarily aimed at understanding aetiology, have assessed the relationship between bio-medical measures such as birth weight and adult cardiovascular disease.

Figure 1 illustrates results from one such study, using birth records from the early part of the 20th century in Hertfordshire.[2] Men whose births were reported in these records were traced through the death registration system and their cause of death linked to their birth weight. There is a 37% increase in risk of mortality from coronary heart disease from the highest to the lowest birth weight group. For premature mortality (death before 65 years), the gradient is even steeper. Furthermore, this relationship has been confirmed in a recent meta-analysis.[3]

Other studies have assessed the relationship between early health-related behaviours and cardiovascular risk. For example, smoking in pregnancy might increase long-term cardiovascular risk in offspring in at least two ways. First, living with two parents who smoke increases the risk of a child subsequently becoming a smoker themselves threefold, compared to living with non-smoking parents.[4] Second, smoking during pregnancy is associated with an increased risk of subsequent obesity, itself a risk factor for cardiovascular disease. A recent meta-analysis estimated that the risk of overweight was about 50% higher in the offspring of women who had smoked during pregnancy and this did not appear to be due to confounding by sociodemographic factors.[5]

A further focus is on how the circumstances of individuals' early lives affect health and disease risk later. For example, several studies have shown that growing up in poorer circumstances is linked to higher risk of adult cardiovascular disease, regardless of adult socioeconomic position. Furthermore, the later mortality disadvantage of being in a low socioeconomic position in early life is not confined to older birth cohorts. The relationship between poor circumstances early in life and later cardiovascular mortality is also seen in more recent cohorts, who did not, in general, suffer from under-nutrition or other extremes of material deprivation in childhood.[6]

Early socioeconomic disadvantage might be linked to cardiovascular disease through a number of different mechanisms, including early exposure to tobacco, unhealthy diets and other so-called lifestyle determinants. Recent research has also linked early childhood adversity, including maltreatment, with adult cardiovascular health. For example, in a large study from California using retrospectively reported data, risk of ischaemic heart disease increased with increasing numbers of reported adverse childhood experiences (for example, being the victim of domestic violence, living with a parent who misused alcohol), independent of conventional adult cardiovascular risk factors.[7] This suggests a specific role for childhood adversity in the genesis of ischaemic heart disease.

Mechanisms, debates and challenges

Two prominent conceptual models describe how early exposures might be linked to adult cardiovascular disease.[8] In the first model, often called 'programming', factors act at a critical early period, such as in utero, permanently changing physiological processes or structures in ways which subsequently lead to poor adult health. In an alternative model, early life circumstances are the start of a cumulative experience of adverse factors, which, over time, leads to increased risk of poor adult health – this model is often referred to as an 'accumulation' or 'chains of risk' model, but again, it depends on early exposures.

Although scientific paradigms may separate programming models from chains of risk, they are not mutually exclusive.[9] For example, smoking in pregnancy may set up a chain of risk for adult smoking whereby a child lives in a household where a parent smokes, takes up smoking as a young person, and whose smoking habits persist, increasing their cardiovascular risk. Additionally, smoking in pregnancy may act at a critical period in fetal development, and, through a programming effect not yet elucidated, increase the risk of adult obesity, also a cardiovascular risk factor (Fig 2).

Although there is much evidence linking early life to adult cardiovascular disease, areas of debate remain. These include the causal nature of the relationship, and particularly what some measures of exposure, such as birth weight, indicate. There is also debate over the size of association and therefore the potential for change if early life exposures could be modified. The precise mechanisms underlying the relationships are not yet

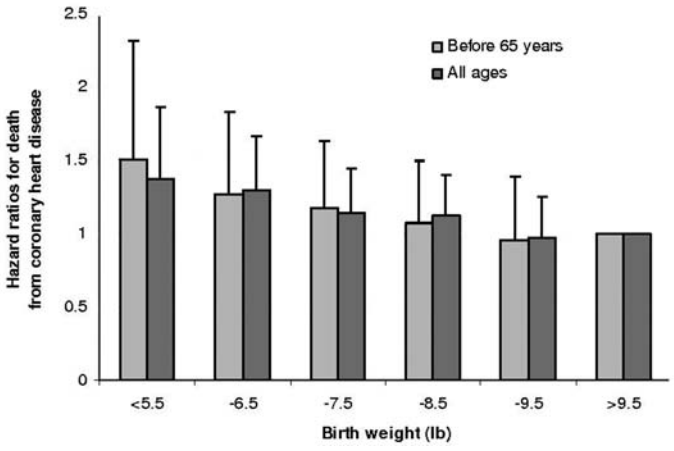

Fig 1. Hazard ratios (95% confidence intervals) for death from coronary heart disease by birth weight (n=10636 men, born 1911–1930, Hertfordshire, UK). Based on data from reference 2.

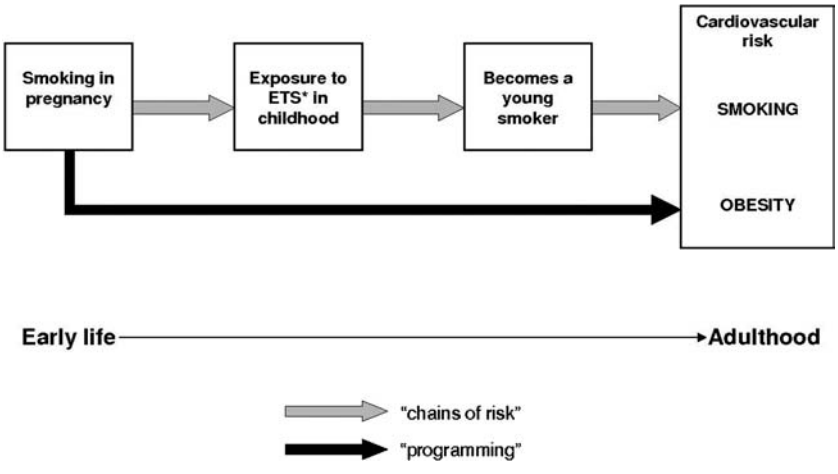

Fig 2. Conceptual model linking early life exposures with adult cardiovascular risk. ETS = environmental tobacco smoke. Adapted from reference 8.

detailed, although a vast literature in experimental animals (not reviewed here) indicates plausible mechanisms.[10–12] Finally, some argue that public health intervention in early life to prevent adult disease cannot be justified until causality is proved. One traditional way of assessing whether exposure is causally related to outcome is to demonstrate that experimental manipulation of the exposure leads to change in the outcome. This reveals a Catch-22 situation. Testing of public health interventions cannot be justified without proof of causality, but proving causality requires demonstrating changes following experimental intervention in human populations.

Many of the early life exposures that are linked to cardiovascular disease are highly prevalent. For example, in 2005, 32% of mothers in England who had recently given birth reported smoking in the 12 months before or during pregnancy.[13] Over 34,000 children were the subject of a child protection plan at the end of March 2009.[14] A staggering 2.9 million children (23% of UK children) live in households with an income below 60% of contemporary median income.[15] Furthermore, many of these risk factors cluster, meaning that children are multiply disadvantaged.

So, at the beginning of the 21st century, there is much evidence linking (highly prevalent) early life factors to adult cardiovascular disease, a disease likely to continue to place a high burden on this and future generations. Substantial research evidence linking early life interventions to cardiovascular risk is unlikely in the foreseeable future. How then can the wealth of current information be used sensibly to benefit children's futures?

Using the evidence in policymaking

Over the last 12 years, policy documents have drawn on a wide body of research evidence, including that linking early life experience to adult cardiovascular disease, to recognise that early experience is likely to be influential in determining life chances, in promoting human capital and economic development, and in improving lifelong health. This has led to an emphasis, at least in policy rhetoric, on primary prevention, on early intervention and so forth. For example, in New Labour's first white paper on health, *Saving lives: our healthier nation*, it was recognised that:

> *People's health can be strongly influenced by patterns of ill-health which can flow down the generations. For mothers and young children, the importance of improving health to break these patterns is clear.*[16]

The maternity standard of the National Service Framework for Children, Young People and Maternity Services includes a similar phrase:

> *the care and support provided for mothers and babies during pregnancy, childbirth and the postnatal period has a significant effect on children's healthy development and their resilience to problems encountered later in life.*[17]

However, in moving from an overarching policy aim, that of breaking intergenerational transmission of ill health, to how such an aim might be delivered through health services, the link between evidence and policy action has broken. This factual statement on the consequences of service delivery cannot be justified from the scientific evidence.

But might evidence be useful in more specific circumstances? Take the example of policies which aim to promote the nutrition of expectant mothers and infants. As well as immediate benefits to health, part of the rationale of such policies is to promote adult health.

In a nutshell, the research evidence shows that maternal nutritional state, size at birth and adult disease are interlinked. But the implications of these links for dietary change in pregnancy are uncertain, and include the possibility that some changes in diet in pregnancy might be harmful to short- or long-term health of the offspring. Breastfeeding is linked to short-term benefits for infant health and possibly long-term benefits too.[18] Can this evidence be useful in policymaking?

The new Healthy Start Programme, focused on disadvantaged families, has indeed embraced the need for earlier support of nutrition (www.healthystart.nhs.uk/). Its predecessor, the Welfare Food Scheme, started in infancy and provided a perverse incentive to bottle feed. The new policy starts in pregnancy, provides at least equal incentives to breastfeed and has been implemented in a way that should facilitate earlier contacts with antenatal services. Though it is rarely possible to prove the influence of research on final policy, Healthy Start was developed following a rigorous evidence synthesis, which included research on the links between early nutrition and adult disease.

By contrast, the Health in Pregnancy Grant (announced in 2007 but scrapped in 2010) gave a lump sum of £190 to all pregnant women but not until after 25 weeks gestation. When launched, ministers indicated that this new policy would promote maternal nutrition and so tackle the UK's high rates of low birth weight. However, the evidence suggests that nutritional change at this late stage of pregnancy would make little difference to birth weight, not least for the many low birth weight babies born before term. The danger of policy misinterpretations of the evidence is that they may divert focus and resource from interventions which might address low birth weight and its sequelae.

Why use science in policymaking?

One could argue that science on long-term benefit is not needed to advance the case for promoting mother's and children's health. That could be done on the basis of short-term effects, compassion or human rights.

But failure to consider long-term outcomes limits the potential for beneficial change. Furthermore, better use of research might lead to increased understanding of the specific actions needed in early life for short- and long-term benefits, or at least better understanding of the trade-offs that might have to be made.

Consider a hypothetical example. In the UK, a main plank of family anti-poverty strategies is to encourage employment of one or both parents. The evidence base could be used to

predict what might happen to a child's short- and long-term health under this policy scenario. Suppose, for example, that a lone mother goes back to work soon after the birth of her baby to escape poverty. She may increase her income (likely to be beneficial to her baby's short and long-term health),[19] but she is less likely to breastfeed (not beneficial).[20] Her baby may receive high quality professional childcare, and that should lead in the long-term to health and other benefits.[21] But she will have less time for her baby (not beneficial), and she may feel more stress, she may find attachment to her baby more challenging, and so on.

If, on the other hand, the policy scenario is one of increased welfare benefits, once again, household income should increase. The evidence suggests that breastfeeding is now more likely but the baby and indeed mother might not benefit from access to childcare. There will be more time for parenting but time and money are not the only stresses on lone parents, and social isolation might now play a part, with resulting uncertainties on stress and attachment.

So, under both scenarios, the short- and long-term net effects on health of the infant are difficult to predict. However, the use of evidence enables consideration of how alternatives might operate and therefore how benefits might be maximised and potential harms mitigated.

Developing science for use in policymaking

Policymakers tend to be risk averse, cost conscious, and often need to demonstrate achievement in short timescales. If they are to use research on the possible long-term outcomes of current action, they need it presented in ways which allow them to balance short- and long-term costs and benefits, and to recognise uncertainty. The following are suggestions for augmenting current research to increase its use in policymaking.

Current research often uses physiological or disease specific outcome measures, reflecting a focus on aetiology. For example, there are studies of the relationship of birth weight with many causes of death but none with life expectancy or quality of life, outcomes valued by the population at large and by policymakers. Outcomes might also include consideration of costs, harms and co-benefits, particularly in evidence syntheses.

Reporting of research and especially evidence syntheses might take better account of context. For example, mothers and young children who might be exposed now to risks that will influence their later adult health may be different physiologically, genetically, culturally and socially than those on whom research has been reported. Furthermore, the prevalence, nature and intensity of exposures are likely to depend at least partly on historical time.

Finally, research on mechanisms has tended to focus on biological and individual, rather than societal mechanisms of action. Major policies with long-term aims, such as Sure Start children's centres and increased welfare benefits for families, should be implemented with robust evaluation, including attempts at interim assessment of those long-term aims.[22]

Conclusions

Gavin Milroy talked of 'abounding and conflicting evidence'. The capacity to generate scientific evidence linking early life to adult disease has greatly exceeded the ability to apply it to public policy in anything but the most general way. This risks spending much time and resource in uncovering how and why early life is linked to adult disease without paying proportionate attention to what can be achieved by this knowledge.

Early experience has huge potential to alter adult health. One of the challenges for science and for medicine, as well as for policymakers, is to ensure that it brings only benefits for our children when they grow up.

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