Social and biological pathways linking early life and adult disease

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Evidence is presented for a pathways model linking early life factors and adult disease, which takes account of the inter-relationships between social and biological risks throughout the lifecourse. Few studies, if any, have yet recorded adequate birth to death information which could be used to quantify the effects of different factors and their timing. Hence, there is only limited understanding of the extent to which biological and social risks experienced at different life stages combine to influence adult disease. However, some of the pathways between early and later life are suggested when evidence from earlier stages of the lifecourse is linked to that from studies at older ages, in which adult disease risk factors have been established. Further support for pathway effects is provided by studies showing that health outcomes of early biological insults can depend on the subsequent social and biological environment. Thus, it is argued that adult disease will be more fully understood when account is taken of the combined effects of social and biological risk occurring at different life stages.

Previous papers in this issue illustrate the diversity of relationships between early life factors and adult health. These relationships span a range of health outcomes which, in some instances, manifest several decades later. It is not surprising that there is great interest in early life/adult health relationships, nor that the possible explanations attract a broad range of comment from medical and social scientists. This discussion focuses on the two major and sometimes conflicting models that are available to explain how early life environment influences health in later life.

The first, which can be described as a latency model, emphasises the strong independent effects on health status late in life, of discrete events that tend to occur early in life. Evidence supporting the existence of critical or sensitive periods in brain development is taken to support this model; as are the associations between birthweight, placenta size and weight gain in the first year of life with cardiovascular disease in the fifth decade. It is hypothesised that, in the case of the latter associations, future adult disease is 'programmed' during fetal life and infancy.
The second, which can be described as a pathways model, emphasises the role of early environment on subsequent life trajectories, which in turn influence adult health. In other words the pathways model focuses on the cumulative effect of life events along developmental trajectories, and it thereby implicates conditions of life throughout the lifecycle in adult disease causation. Evidence for differential pathways and related health effects is most widely seen in relation to socioeconomic status, as supported by the findings of longitudinal studies of children, working populations and the elderly which, when overlapped, highlight the enduring impact of social position on health.

For practical or policy considerations, latency and pathway models could be viewed as complementary to one another. After all, there is no reason to suppose that latent factors only act independently. Any early life event which could exert a latent effect may also be the first stage along a lifelong pathway which might have implications for future health. Yet despite the conceptual complementarity, the two explanatory models appear to generate different policy approaches. The latency model leads to an early childhood investment strategy, whereas the pathways model supports strategic investments throughout the lifecycle. Thus, the two models tend to attract different audiences who, in turn, are separated by different world views. Such differences will be difficult to resolve because of methodological problems inherent in lifelong health studies.

The complex relationship between latent and pathway effects is illustrated with data from the 1958 birth cohort study, in which subjects have been followed up most recently to age 33 (see Table 1 for a summary of the study). Table 2 shows the relationship between health in early adulthood and social class of origin. In general, the relationships shown in Table 2 are graded, with improvements in health evident for each increase in the social hierarchy. It could be argued that the relationships with class at birth represent evidence of a latent effect of

Table 1 Summary of 1958 British birth cohort study

<table>
<thead>
<tr>
<th>Year</th>
<th>Birth</th>
<th>Age 7</th>
<th>Age 11</th>
<th>Age 16</th>
<th>Age 23</th>
<th>Age 33</th>
</tr>
</thead>
<tbody>
<tr>
<td>1958</td>
<td>17,773</td>
<td>16,883</td>
<td>16,835</td>
<td>16,915</td>
<td>16,457</td>
<td>16,455</td>
</tr>
<tr>
<td>Data sources</td>
<td>Parents</td>
<td>Parents</td>
<td>School</td>
<td>School</td>
<td>Tests</td>
<td>Test</td>
</tr>
<tr>
<td></td>
<td>School</td>
<td>Medical</td>
<td>Subject</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Target sample*</td>
<td>17,773</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Achieved sample</td>
<td>17,414</td>
<td>15,458</td>
<td>15,503</td>
<td>14,761</td>
<td>12,537</td>
<td>11,407</td>
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</table>

Table 2  Morbidity and symptom prevalence at age 33 by social class at birth

<table>
<thead>
<tr>
<th>Health/morbidity</th>
<th>Sex</th>
<th>I &amp; II</th>
<th>III NM</th>
<th>III M</th>
<th>IV &amp; V</th>
<th>All</th>
<th>Total sample</th>
<th>χ² trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morbidity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poor/fair self rated health (%)</td>
<td>M</td>
<td>17.5</td>
<td>9.8</td>
<td>50.9</td>
<td>21.8</td>
<td>8510</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>17.5</td>
<td>9.5</td>
<td>50.8</td>
<td>22.1</td>
<td>7944</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Limiting longstanding illness (%)</td>
<td>M</td>
<td>5.1</td>
<td>5.9</td>
<td>6.2</td>
<td>7.6</td>
<td>6.2</td>
<td>5057</td>
<td>4.65**</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>4.7</td>
<td>5.7</td>
<td>8.6</td>
<td>6.1</td>
<td>5230</td>
<td></td>
<td>10.72**</td>
</tr>
<tr>
<td>Psychological distress (%)</td>
<td>M</td>
<td>4.2</td>
<td>5.1</td>
<td>7.4</td>
<td>7.7</td>
<td>6.6</td>
<td>5044</td>
<td>13.88***</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>6.3</td>
<td>11.5</td>
<td>12.5</td>
<td>15.4</td>
<td>11.9</td>
<td>5216</td>
<td>39.55***</td>
</tr>
<tr>
<td>Back pain (last 12 months) (%)</td>
<td>M</td>
<td>29.3</td>
<td>29.9</td>
<td>30.1</td>
<td>31.5</td>
<td>30.2</td>
<td>5042</td>
<td>1.11</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>22.7</td>
<td>25.0</td>
<td>27.3</td>
<td>31.9</td>
<td>32.7</td>
<td>5214</td>
<td>21.65***</td>
</tr>
<tr>
<td>Respiratory symptoms ≥ 1 (%)</td>
<td>M</td>
<td>16.0</td>
<td>17.1</td>
<td>21.4</td>
<td>24.7</td>
<td>20.6</td>
<td>4935</td>
<td>26.47***</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>12.4</td>
<td>10.4</td>
<td>17.0</td>
<td>22.0</td>
<td>16.6</td>
<td>5109</td>
<td>40.72***</td>
</tr>
<tr>
<td>Obese (%)</td>
<td>M</td>
<td>7.6</td>
<td>7.5</td>
<td>11.6</td>
<td>14.3</td>
<td>11.0</td>
<td>4984</td>
<td>27.27***</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>10.0</td>
<td>14.3</td>
<td>17.7</td>
<td>17.7</td>
<td>16.0</td>
<td>5137</td>
<td>28.49***</td>
</tr>
</tbody>
</table>

*P < 0.05  **P < 0.01  ***P < 0.001.

aFor a fuller definition of health measures see Power and Bartley.

bScoring 7 or more on the malaise inventory.

cBody Mass Index > 30 (men), > 28.6 (women).

early life social circumstances, but this would be an incomplete explanation because there is no representation of intervening experience in the table. It is possible that social class at birth predisposes the individual to a series of social and biological events that evolve over time, and which are similarly related to social position. If so, social class at birth would be a marker for subsequent experience and the data in Table 2 could, instead, be explained by a pathway model.

Several of the earlier contributions to this issue have focused on factors associated with the latency model. In contrast, this chapter will present evidence highlighting the importance of pathway effects. This evidence is organised in two sections. In the first section, evidence is presented to illustrate how social and biological risk factors for adult disease accumulate differentially for individuals with different social backgrounds. Here we draw on pertinent observational data, in particular that from the 1958 birth cohort study. Further evidence for a pathways model is presented, in a second section, from studies showing that health outcomes of early biological insults can depend on the subsequent social and biological environment. Examples are available, mainly from educational and social intervention studies, demonstrating that early life trajectories can be modified in ways that affect life chances. Deliberate efforts to change the experiences of those on a given ‘trajectory’ can be successful, and thereby change the individual’s life circumstances and health status as time passes.
Social and biological pathways

Table 3  Social and biological risk factors for adult disease, according to social class at birth

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>Social class at birth</th>
<th>Sex</th>
<th>I &amp; II</th>
<th>III M</th>
<th>III M</th>
<th>IV &amp; V</th>
<th>All</th>
<th>Total sample</th>
<th>( \chi^2 ) trend P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birthweight (mean g)</td>
<td>M</td>
<td>3433</td>
<td>3415</td>
<td>3357</td>
<td>3354</td>
<td>3371</td>
<td>7315</td>
<td>444.4***</td>
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</tr>
<tr>
<td></td>
<td>F</td>
<td>3295</td>
<td>3277</td>
<td>3237</td>
<td>3207</td>
<td>3239</td>
<td>7154</td>
<td>&lt;0.05</td>
<td></td>
</tr>
<tr>
<td>Childbirth conditions</td>
<td>M</td>
<td>10.6</td>
<td>21.5</td>
<td>34.1</td>
<td>47.6</td>
<td>31.7</td>
<td>6573</td>
<td>417.4***</td>
<td></td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>9.5</td>
<td>23.3</td>
<td>35.0</td>
<td>46.1</td>
<td>31.8</td>
<td>6242</td>
<td>&lt;0.05</td>
<td></td>
</tr>
<tr>
<td>Parental divorce (%)</td>
<td>M</td>
<td>2.7</td>
<td>5.3</td>
<td>8.8</td>
<td>12.8</td>
<td>8.3</td>
<td>6641</td>
<td>95.5***</td>
<td></td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>3.0</td>
<td>4.8</td>
<td>9.1</td>
<td>13.1</td>
<td>8.5</td>
<td>6305</td>
<td>92.5***</td>
<td></td>
</tr>
<tr>
<td>Height of age 33 (mean m)</td>
<td>M</td>
<td>1.28</td>
<td>2.26</td>
<td>1.87</td>
<td>2.30</td>
<td>1.89</td>
<td>5001</td>
<td>24.92***</td>
<td></td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1.44</td>
<td>1.64</td>
<td>1.82</td>
<td>2.40</td>
<td>1.86</td>
<td>5097</td>
<td>28.62***</td>
<td></td>
</tr>
<tr>
<td>Educational qualifications (%)</td>
<td>M</td>
<td>1.78</td>
<td>1.77</td>
<td>1.77</td>
<td>1.76</td>
<td>1.77</td>
<td>6388</td>
<td>&lt;0.05</td>
<td></td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1.64</td>
<td>1.63</td>
<td>1.62</td>
<td>1.62</td>
<td>1.63</td>
<td>6700</td>
<td>&lt;0.05</td>
<td></td>
</tr>
<tr>
<td>Regular smoking ages 23–33 (%)</td>
<td>M</td>
<td>20.3</td>
<td>23.2</td>
<td>27.2</td>
<td>30.4</td>
<td>26.1</td>
<td>4239</td>
<td>25.48***</td>
<td></td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>17.5</td>
<td>20.4</td>
<td>26.4</td>
<td>35.2</td>
<td>26.0</td>
<td>4542</td>
<td>75.00***</td>
<td></td>
</tr>
<tr>
<td>Job demand/control (%)</td>
<td>M</td>
<td>14.5</td>
<td>17.6</td>
<td>21.8</td>
<td>27.1</td>
<td>21.0</td>
<td>4655</td>
<td>46.88***</td>
<td></td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>25.1</td>
<td>33.9</td>
<td>34.8</td>
<td>37.8</td>
<td>33.5</td>
<td>4942</td>
<td>34.89***</td>
<td></td>
</tr>
</tbody>
</table>

\( a \) Childhood material conditions, indicated by overcrowding ( > 1 person/room) at any two ages at 7, 11, or 16, and sharing or lacking household amenities (either hot water, bathroom, indoor toilet) at any two ages at 7, 11, or 16.

\( b \) Qualifications obtained by age 23, none vs 1 or more.

\( c \) High negative work attributes, such as no ability to vary the pace of work or timing of breaks, monotonous tasks, no requirement for learning new skills.

\( * * * P < 0.001 \)

Risks accumulating over time

Social origins, adult disease risks and health status

To explore the prospect that early life social origins differentiate subsequent life trajectories that are likely to influence adult health, we examine how risks accumulate over time, in relation to social class of origin. This is illustrated in Table 3, which gives the distribution of seven social and biological risk factors according to social class at birth. Risk factors are presented in order of the age at which they occur. Each factor was selected because it had been suggested as having an independent predictive effect on adult disease: birthweight, childhood material circumstances, parental divorce, height, educational attainment, smoking behaviour and demand and control relationships at work (notably work stressors and the range of decision making freedom).

Strong gradients are observed according to social class at birth for each of these factors (Table 3). This confirms that class at birth, whatever else it represents, is systematically associated with factors relevant to adult disease. Furthermore, Table 3 also provides evidence...
that the selected factors accumulate differentially by social group, such
that by adulthood those born into successively lower social classes have
experienced more biological and psychosocial risk; received fewer
educational investments; and have poorer working environments and
worse health behaviours than those with higher social origins. The most
parsimonious explanation of these findings is that pathway effects are
the dominant source of variation in health status in adulthood. Even so,
the relationships in Table 3 may be misleading, merely serving as a
marker for early social origins. Further evidence is needed to disentangle
early and later life factors. For example, if the pathway model were
important, then adult health risk would be more strongly related to
factors which accumulate over time than to social class at birth.

One test of this is provided by a comparison of the relationships for
educational attainment and adult health (Table 4) with those for social
class at birth and adult health (Table 2). It appears that gradients of
health outcome, at this stage in the lifecourse, are stronger in general in
relation to educational attainment than to social class at birth.30
Educational attainment is strongly influenced by social background, but
it is not equivalent to it. Indeed, social measures such as occupational
class and education may be related but they are theoretically distinct,
with occupation representing working and living conditions, and
education representing cultural capital. Thus, health behaviours, such
as smoking and diet, are more closely associated with education than
they are with income and occupation. In the present context, educational attainment is likely to be representing aspects of readiness

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### Table 4  Morbidity and symptom prevalence at age 33 by educational qualifications

<table>
<thead>
<tr>
<th>Health/morbidity</th>
<th>Sex</th>
<th>&gt; A level</th>
<th>A level</th>
<th>O level</th>
<th>&lt; O level</th>
<th>None</th>
<th>All</th>
<th>Total sample</th>
<th>χ² trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>No in each class (%)</td>
<td>M</td>
<td>19.0</td>
<td>22.0</td>
<td>23.8</td>
<td>19.7</td>
<td>15.4</td>
<td>6267</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>18.0</td>
<td>11.3</td>
<td>29.8</td>
<td>25.6</td>
<td>15.3</td>
<td>6270</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poor/fair self rated health (%)</td>
<td>M</td>
<td>6.4</td>
<td>9.9</td>
<td>10.3</td>
<td>18.1</td>
<td>23.4</td>
<td>12.6</td>
<td>4685</td>
<td>119.6***</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>6.5</td>
<td>9.5</td>
<td>10.6</td>
<td>17.1</td>
<td>25.0</td>
<td>13.2</td>
<td>4978</td>
<td>130.9***</td>
</tr>
<tr>
<td>Limiting longstanding illness (%)</td>
<td>M</td>
<td>4.0</td>
<td>3.7</td>
<td>5.8</td>
<td>6.4</td>
<td>12.3</td>
<td>5.9</td>
<td>4711</td>
<td>45.1***</td>
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<tr>
<td></td>
<td>F</td>
<td>4.8</td>
<td>6.7</td>
<td>5.1</td>
<td>5.8</td>
<td>10.9</td>
<td>6.2</td>
<td>5020</td>
<td>13.8***</td>
</tr>
<tr>
<td>Psychological distress (%)</td>
<td>M</td>
<td>2.8</td>
<td>4.2</td>
<td>6.9</td>
<td>8.0</td>
<td>11.6</td>
<td>6.3</td>
<td>4700</td>
<td>58.5***</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>4.5</td>
<td>4.8</td>
<td>10.5</td>
<td>15.0</td>
<td>24.7</td>
<td>11.7</td>
<td>5005</td>
<td>175.8***</td>
</tr>
<tr>
<td>Back pain (last 12 months) (%)</td>
<td>M</td>
<td>23.6</td>
<td>28.9</td>
<td>32.6</td>
<td>33.4</td>
<td>35.0</td>
<td>30.4</td>
<td>4698</td>
<td>50.5***</td>
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<tr>
<td></td>
<td>F</td>
<td>23.1</td>
<td>25.9</td>
<td>26.8</td>
<td>27.9</td>
<td>33.2</td>
<td>27.1</td>
<td>5002</td>
<td>18.4***</td>
</tr>
<tr>
<td>Respiratory symptoms ≥ 1 (%)</td>
<td>M</td>
<td>12.4</td>
<td>16.6</td>
<td>21.3</td>
<td>23.7</td>
<td>34.8</td>
<td>20.6</td>
<td>4605</td>
<td>119.1***</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>10.2</td>
<td>11.1</td>
<td>13.4</td>
<td>20.9</td>
<td>29.5</td>
<td>16.5</td>
<td>4900</td>
<td>121.7***</td>
</tr>
<tr>
<td>Obese (%)</td>
<td>M</td>
<td>6.4</td>
<td>8.9</td>
<td>10.7</td>
<td>13.2</td>
<td>16.0</td>
<td>10.5</td>
<td>4650</td>
<td>45.3***</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>12.5</td>
<td>13.4</td>
<td>14.6</td>
<td>17.1</td>
<td>23.4</td>
<td>13.9</td>
<td>4929</td>
<td>33.8**</td>
</tr>
</tbody>
</table>

*p < 0.05 **p < 0.01 ***p < 0.001

*See footnotes to Table 2
for school, social and behavioural adjustment, social investment opportunities made available, social stability and health-related behaviours. Thus, it is an excellent marker of the ‘healthfulness’ of accumulated childhood experience. That educational attainment is more strongly associated with adult health status than class at birth suggests that pathway effects may be operating.

Others have demonstrated stronger relationships between adult disease and social measures for later life than for measures earlier in life\textsuperscript{33}. However, as Lynch \textit{et al}\textsuperscript{33} caution in the interpretation of their results showing adult mortality to be more closely linked to income in adult life, such evidence does not suggest that socioeconomic conditions in childhood are unimportant. Childhood environment can be regarded as contributing to adult disease through its strong influence on adult socioeconomic circumstances.

**Studies of lifetime exposures**

It could be argued that a more useful approach, and one that more accurately represents the complex relationships between social and biological influences across the lifecourse, is to try to understand how risks experienced at different ages \textbf{combine} to increase risk of disease in adult life. This is a common research strategy in psychological investigation, as reflected for example in Rutter’s\textsuperscript{34} description of ‘chains of risk’. According to this conceptualisation, several, sometimes many, intervening links in a chain are required before long-term sequelae become manifest, and in the absence of some of these connections adverse outcomes may be averted. Others considering relationships over the lifecourse refer to ‘unhealthy life careers’ triggered by childhood living conditions and associated social problems, such as broken family and family conflicts\textsuperscript{9}. In contrast, epidemiological investigations tend to emphasise the independence of risks associated with a particular factor or life stage. But ‘chains of risk’ are also likely to be relevant to other dimensions of adult health other than psychological well-being.

To illustrate, childhood living conditions are known to influence a child’s physical development, as indicated by their height\textsuperscript{35}, which in turn influences adult social position\textsuperscript{36}; then both height and adult social position subsequently affect adult health and mortality\textsuperscript{19}. In another example, socioeconomic conditions affect the probability of exposure to lead \textit{in utero} and in childhood. In turn, children in the lowest social position seem to have a lower threshold of vulnerability to lead’s developmental effects\textsuperscript{37}, which in turn, may lead to academic underachievement as reflected in
lower education attainments, and thereafter a lower educational level; which, in turn, increases the risk of adult mortality.

It would be possible to speculate with many other pathways linking early environment and adult health. As is evident from these examples, though, the arguments about pathways have tended to rely on extrapolating from evidence on early life, to that from studies starting in middle age and following through to death (such as the Whitehall study\textsuperscript{16}). Unfortunately, few studies as yet have adequate information from birth to death that also include data for the intervening years.

However, some evidence exists to suggest that adult health is more fully understood by taking account of life careers or accumulated circumstances than by focussing on any particular period in isolation from other life stages. This can be seen for adult ‘health’ outcomes including mortality. It is illustrated, for example, in studies that show stronger relationships between mortality and longest occupation than between mortality and current occupation\textsuperscript{38}. In this context, longest occupation provides a better proxy for lifetime living conditions than a social measure restricted to a particular life stage. Other investigations of lifetime socioeconomic circumstances demonstrate that mortality risks associated with lower social class origins are to some extent ameliorated by higher socioeconomic position in later life\textsuperscript{39,40}. Nonetheless, Mare’s analysis of mortality and socioeconomic status at several life stages, suggests that the effects of social origins endure through relationships with later experiences, such as education and subsequently with occupational and financial resources\textsuperscript{39}. Even for the shorter period of childhood growth, measures of the cumulative effect of socioeconomic status, as represented by persistent poverty, are related to childhood stunting (low height for age) and wasting (low weight for age), whereas single year income measures are not\textsuperscript{35}. In sum, evidence on combined effects across the lifecourse is sparse, but the approach promises insights concerning the relevance of pathway effects, and thereby should enhance understanding of adult disease.

**Modification of early life insults**

Evidence which shows that early life insults can be altered, and in ways which affect subsequent disease risk is, in effect, support for the significance of a complex interaction between latent and pathway effects. Some of the research showing modification of adult mortality risk associated with early life environment has been discussed in the section above. However, it is from studies of shorter periods earlier in life, particularly those concerned with child developmental trajectories, that evidence on modifying influences is well documented. In the Kauai birth cohort study, for example, it was found that severe perinatal stresses (that
is, complications of pregnancy, labour, and delivery) compromised the physical and psychological development of children from low socioeconomic status families, but was successfully buffered in higher status families. By 20 months of age, the average ‘developmental quotient’ for low socioeconomic status children who had experienced severe perinatal stress was much below similarly stressed children from higher socioeconomic status families. In contrast, low socioeconomic status children who had experienced mild or moderate perinatal trauma were developmentally much closer to their more affluent counterparts. High socioeconomic status, in this case, represents a series of ongoing investments which not only protect healthy children from future risks but can reverse the impact of existing risks. The investment pays off when children reach school age. Those who are less ready, intellectually and emotionally, to cope with the school environment quickly enter a spiral of behavioural acting out and academic failure which leads to early drop out and delinquent behaviour in both boys and girls. Thus, by school entry pathway effects start to predominant.

**Intervention studies**

In addition to such evidence from observational studies, there is now an extensive body of research showing that well-designed stimulation programmes can improve the cognitive and social-emotional development of children. The focus on cognitive and social-emotional functioning is relevant because of its connection to school readiness. School readiness, in turn, is important because the complex web of early academic failure and early school misbehaviour is associated with lack of school readiness and, in turn, strongly predictive of school failure, employability, criminality, and psychological morbidity in young adulthood.

Effective programmes to improve cognitive and social-emotional functioning can begin at a variety of ages, but some start as young as the first few months of life. These are parent/infant stimulation programmes, which feature home-based activities; voluntary involvement of at least one parent; reinforcement of the role of the parent in child development; frequent contact with programme staff; and establishment of positive role models from the local community. It is important to recognise that such programmes demonstrate improved cognitive and social-emotional functioning of children from both the socioeconomic/psychosocial, and, also, the biological (e.g. low birth weight) categories of high risk.

Starting at age 6 months, programmes based in education centres have been shown to be helpful for children in the socioeconomic/psychosocial category of high risk. Moreover, programmes which have been
successful at improving either or both cognitive and socioemotional functioning, in either or both males and females, have been demonstrated up until the onset of puberty. In general, program intensity, length, and parental involvement are positively correlated with success.

As children get older, it becomes more difficult to organise effective programmes. This may be taken as indirect support for the importance of latent effects, and, insofar as human development follows a pattern of ‘critical periods’, this would be a justifiable inference. However, it is also true that, as children age, the range of influences on them broadens, such that classroom and home take up an ever-shrinking fraction of their time and their consciousness. It is, therefore, no surprise that effective programmes which begin in early adolescence, or which sustain the effects of early intervention into that period, are often buttressed by ongoing efforts outside such as, for instance, recreational programmes.

At this point, the relationship between latency, pathways, and the effectiveness of intervention studies becomes more complex. There are two conceptual problems. The first is that a successful intervention study at a given age demonstrates the importance of pathway effects to that age, but, potentially, reinforces the importance of latent effects thereafter. This issue was partially addressed in the previous paragraph, but cannot be fully addressed without evidence from a greater number and variety of intervention studies from mid-adolescence and beyond.

The second issue concerns the relationship between individual coping and social opportunity. Whereas, effective intervention studies designed to improve individual capacities support pathway effects in retrospect, and latent effects in prospect; the role played by available opportunities (for example, education, work, social support, and so on) is a purely pathway effect. Thus, to assess the relative importance of latent versus pathway effects through intervention studies, it is not enough to know whether or not improvement has occurred. It is also important to ask whether or not the investment in individual capacities can fully overcome the restricted opportunities faced by those at socioeconomic disadvantage. The answer to this question, so far, is ‘no’. In other words, providing socioeconomically disadvantaged children with cognitive and socioemotional functioning matching that of more privileged children will improve their life chances, but will not fully overcome the pathway advantages of upper social class.

Conclusion

In conclusion, a pathways model linking early life and adult disease is suggested with evidence from both observation and intervention studies.
As yet the evidence is incomplete since no studies have relevant data, that is of health, health-related behaviour and social factors, throughout the lifecourse from birth to death. Despite this the plausibility of a pathways model is suggested by the strong relationship between an individual’s social origins and their subsequent experience of likely risk factors for disease in later life, as indexed by birthweight, childhood material circumstances, parental divorce, smoking behaviour, educational attainment, adult height, and demand and control relationships at work. Given that the effects of early life insults such as severe perinatal stress, low birthweight and lack of adequate stimulation can be modified to some extent by subsequent circumstances and events, is further support that pathway effects are operating. These studies suggest therefore, that the ongoing role of social investment cannot be overstated.

Prominent among the many research questions that are outstanding, are the following: which early life insults are not ameliorable to change at later stages in the life course? for which adult disease outcomes are early life factors most important? even for the latter, does a pathways model also apply to the development of adult disease? Consideration of such questions, involves looking in particular to the combined effects of factors occurring at different life stages. We argue here that this approach should incorporate inter-relationships between both social and biological explanations for adult disease.

Acknowledgements

Chris Power (Weston Fellow) and Clyde Hertzman are fellows with the Canadian Institute for Advanced Research. The authors are grateful to Sharon Matthews, who is supported by ESRC (award no R000235189), for preparation of data from the 1958 birth cohort study.

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