What is the Relationship between Higher Obtained Education and a Delayed Age at Onset of Dementia?

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The importance of childhood experience and environment for risk of later life morbidity has received increasingly more attention over the last decades. For instance, childhood IQ in several studies was linked to age at onset of dementia [1]. We believe that there are several theoretical models to explain this association, which is important when deciding at which age-group to target health-promoting activities to prevent dementia. Currently there is no treatment for dementia and prevention seems the only option. With increasing pressure on economic resources worldwide, scientific evidence for these models to guide policy is thus important. In this editorial we discuss which of these models is more salient and when interventions should be introduced for maximum and cost-effective results.

The first model discusses how a higher childhood IQ reflects a better education, and suggests that a better education promotes access to more adult socioeconomic resources leading to better health, including a lower dementia risk. Data from the United States [2] show that a high IQ is strongly linked to a higher adult SES. Health and morbidity are strongly linked to adult SES and education. For instance, in the UK, lower classed or manual ('blue collar') workers were twice as likely to die prematurely of all causes than 'white collar' (higher classed office and non-manual) workers between 2001 and 2003 [3]. Model 1 suggests that having a higher IQ as a child leads to having more social and financial resources as an adult, which may be associated with a lower risk for early mortality and morbidity, including dementia.

According to this model, interventions could mainly focus on adulthood providing more resources to reduce morbidity risk. Data suggest that midlife is the most important time to promote healthy lifestyle behaviours to prevent morbidity, such as later life dementia, with a focus on cessation of smoking, reducing obesity, and treating high blood pressure and cholesterol [4]. These midlife risk factors each individually double the risk for dementia in later life (model 2) and many other (vascular) morbidities associated with early mortality.

High blood pressure was also predicted by childhood IQ, but only up to the age of 65 [5]. After this, variation increases. Interestingly, other data showed that high blood pressure was predictive for age at onset of dementia but only 15 years before the onset of dementia (i.e. in midlife). In this study, blood pressure started to decrease in prospective cases 1-2 years before the clinical onset of dementia symptoms [6]. Several studies thus found lower blood pressure in dementia cases compared to controls. Similarly weight -while higher in midlife- is often lower or similar in dementia cases as controls and others found that a decline in total cholesterol levels was a better predictor for age at onset of dementia than midlife cholesterol levels [7]. This indicates a change in risk factors, so for these cardiovascular risk factors (obesity, high blood pressure and high total cholesterol) an early intervention (in midlife) is crucial as once dementia is apparent, these risk factors are no longer treatable and/or no longer pose risk.

If midlife vascular risk factors are important for later life dementia, and access to resources may prevent this effect, it is important to investigate which factors are associated with better midlife socioeconomic status. In model 1 we discussed the potential of childhood IQ or education. However, midlife adult social status attainment is also linked to father’s social class, independent of childhood obtained education and/or IQ [8] (model 3a). This third model thus focuses on parental SES. Having parents with high SES may increase the likelihood of childhood exposure to healthier environments with better access to health care, more varied diets with fruits and vegetables, less physical and environmental stress caused by overcrowding, smoking and other pollutants, more frequent winter-sun holidays (e.g. skiing in winter) to boost vitamin D levels etc. This model places interventions much earlier, in childhood or even before that to optimise the early life environment.

As discussed above [4], midlife obesity is an important risk factor for dementia. Much research has been done in obesity and risk factors for this and we will discuss some of this work to shed light on when preventative interventions should take place. A review of 135 studies showed that early childhood markers for adult obesity included maternal obesity, maternal smoking, childhood obesity and stunted growth patterns (often an indicator for nutritional deficiency), as well as father’s employment status as a proxy for SES [9]. Poverty in childhood (low father’s employment status) is thus at least partly responsible for a higher risk of obesity in adulthood and later life cardiovascular disease and dementia. This may be because poor nutrition characterised by high fat and processed carbohydrates is relatively cheap and filling, as compared to nutritious more expensive foods, such as lean meats, fish, fruit and vegetables [10]. Recent data, however, suggest that the income-obesity gap is narrowing and one UK study found that middle-classes were more likely to have obese off-spring than lower or upper classes [11].

These data thus suggest that maternal behaviour (adverse dietary patterns and/or inactivity, smoking) as well as child behaviour (perhaps copied from the mother) predispose to adult obesity [12,13] independent of SES (model 3b). Losing weight in adulthood is often difficult and hence promoting non-smoking and a healthy weight in expecting mothers and promoting activity and healthy diets for the whole family would perhaps help promote a healthy lifetime weight in their offspring.

The review [19] also showed that growth patterns were also independently associated with childhood obesity. Stunted growth often reflects poor nutritional status, particularly in early life [14]. From model 3c it could follow that a focus on promoting pre- and early postnatal

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care for mothers may be crucial to promote better brain function in childhood to be able to benefit from good schooling to prevent later life morbidity and poor health. Prenatal care is indeed an important World Health Organisation focus. Giving combination nutritional supplements of zinc, iron, folic acid and vitamin A to deprived prenatal Nepalese mothers (as compared to vitamin A alone) produced children who had better cognitive function at age 7 [15]. In contrast, studies with single supplements showed worse cognition in children after mothers had been given zinc or iron supplements only prenatally [16]. A large European study showed that fish oil and folate given prenatally did not change cognition in childhood, although maternal DHA (from the fish oil) was associated with better cognition in their offspring [17]. There has thus been limited success of prenatal supplements to improve cognition in offspring, but this may be modified by the level of deprivation and the fact that supplements may not necessarily have linear positive effects, but rather show optimum levels to promote better health and brain function. Also perhaps prenatal nutrition is insufficient to sustain optimal brain development and other factors (childhood nutrition and exercise, healthy environments) need to be included to predict this.

More worryingly, this relative failure of prenatal nutritional supplements to make a huge impact on child IQ (and subsequent adult health) may also be caused by intergenerational transfer of risk and a potential role of epigenetics (model 3d). Data could reflect the complexity of planning health-promoting interventions, because these issues are multi-factorial. For instance, low vitamin B12 status in rural pregnant women from a developing country (India) led to a higher risk of adiposity and insulin resistance (a risk factor for diabetes) in their children at 6 years, both which are risk factors for later life morbidity, including dementia [18]. Mothers exposed to environmental pollutants promoting DNA methylation were also found to be more likely to have obese offspring [19,20], which in turn is linked to obesity in adulthood, early mortality and dementia. Epigenetics may play a role in foetal and child brain development and thus affect midlife obesity, and later life dementia. Whether inter-generational risk transfer and potential epigenetic effects can be overruled by combinations of maternal and childhood exercise and nutritional pre- and postnatal interventions, as well as promotion of better schooling and early stimulating, but stress and pollutant free environments remains to be investigated.

Conversely data may indicate a ‘preaching to the converted effect’ where healthy, well-educated, white, middle-class parents engage in clusters of health related behaviours, which are not necessarily taken up by those who have received less education and live in deprived areas, possibly because of a lack of time, money and/or knowledge. This cluster of health related behaviours includes better prenatal care, parental health promoting behaviour (exercise, diet, not smoking) and more stimulation, including access to better schools resulting in better brain development and subsequently higher IQ and higher adult SES [21].

Partly the association between childhood IQ and later life dementia then perhaps just reflects the consequences of poor social mobility and clustering of much beneficial preventative behaviour only in those who can afford this and paradoxically would need it least.

In another variant of this model (4), the emphasis is thus placed on childhood education itself. In the UK, much effort has been put into improving schooling for children from deprived areas, but top universities (which attendance increases the chances of obtaining high SES [22]) still only have a minority of children from deprived backgrounds, with the majority of students coming from middle to high SES [23].

Importantly, however, in most risk predicting models, education remains independently (from cardiovascular risk factors) protective against development of dementia. In addition, in models predicting dementia, when education and adult SES were entered in regression analyses, only education remained significant [24]. This might indicate that better SES is not necessarily the main driver for protection against dementia by promoting better health by allowing access to health promoting resources, such as medical help, less (environmental) stress and better nutrition (models 1,2 and 3). These data favour three other models where a higher obtained childhood education may lead to better SES, but by itself drives an independent effect in maintaining health and reducing risk for dementia.

This association could be explained in model 5 where education promotes knowledge of healthier lifestyles and actively pursuing health promotion (e.g. by visiting primary care providers for treatments for high blood pressure and high cholesterol and eating better diets, etc.). On the other hand, having knowledge does not necessarily transmit to engaging in healthier lifestyles. In a large multi country European cohort, men with the highest education had a 43% reduced risk for total mortality compared to men with the lowest education (for women this was 29%). However, this association was only partly explained by smoking, body weight, alcohol consumption, leisure physical activity and fruit/vegetable intake [25]. So effects of education are not only due to engaging in better health related behaviour. Possibly these data suggest success of public health promoting activities to the general public independent of education or, alternatively, that there are other factors than lifestyle associated with education that increase age at mortality.

However, as education showed independent effects in the earlier Models discussed, it could play another role in a sixth model. Education could promote cognitive resources by giving access to alternative coping styles to solve problems and a wider vocabulary. In dementia once word finding abilities and executive functions start to decline, these cognitive reserves could buffer severity of symptom display. Indeed studies have shown that a higher education is associated with later age at onset of dementia, but a steeper and faster decline [26]. This rapid decline probably occurs when pathological neuronal damage has overruled cognitive reserve capacity which is expected to be later in those with higher education. Vascular, Alzheimer’s and Lewy Body pathology are often found to co-occur at post-mortem. A combination of different dementia pathologies leading to a total pathology load combined with better education (reflecting a positive cognitive reserve balance) could then predict a later age at onset of dementia symptoms but a faster rate of decline once these are present [27].

The last model (7) is often referred to as the ‘use it or lose it model’. Here education would stimulate the lifelong active use of mental facilities, and the brain then acts as a muscle which needs to be stimulated to be maintained. To date whether mental activity can treat dementia is controversial. Staying mentally active was found to be protective in studies [28,29] but whether this is causal is questionable. Rather older people who can still read and write may continue to do so, but having dementia symptoms with word finding problems etc. might impede these activities already at an early stage of the dementia syndrome. On the other hand limited data from the US and Japan did show that engaging in reading and arithmetic exercises improved dementia symptoms after 6 months [30]. Data also indicate that older rats still express neurogenesis when exposed to novel environments.
However, rats exposed to novel environments are also more physically active, which is an important confound in these studies. In dementia we did see some limited evidence of cognitive improvement after moderate exercise but this seemed most prominent in women [4].

In sum, all likelihood the association between childhood IQ and age at dementia onset is complicated, but our models may give some pointers as to when preventative interventions are best applied. Vascular risk associated with dementia needs to be reduced at the latest in midlife, but early acquired healthy lifestyles and good education in childhood are crucial to prevent these midlife vascular risk factors (obesity, high blood pressure, high cholesterol) and subsequent dementia. Once people have developed dementia, nutritional interventions in all likelihood play a less important role [4], but more research needs to investigate whether physical and perhaps mental activity could sustain cognitive function at this stage. Preventing dementia thus requires a lifelong approach.

References


