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Letter to the Editor

Life history theory as a possible explanation for teenage pregnancy

Using data from the Office for National Statistics Longitudinal Study, Webb *et al*. (2011) showed how teenage mothers were around 30% more likely to die prematurely by any cause and around 60% more likely to die from unnatural causes. The authors mentioned that it is unclear whether these women are at high risk of a premature death because of their teenage motherhood or whether their background predisposes them to both teenage motherhood and an early death. However, there is also another possibility not mentioned by the authors. Teenage pregnancy could be an adaptive response to a reduced life expectancy.

Life history theory in biology explains how organisms try to maximise their reproductive success in response to the environment (Hill & Kaplan, 1999). Many birds produce more eggs when there is more food available and in this way they maximise the number of surviving offspring. Socio-economic deprivation in developed countries is associated with increased mortality and morbidity in mid-life (Geronimus *et al*. 1999). Teenage pregnancy might actually be a useful reproductive strategy, because by delaying pregnancy women might well not be able to conceive at all due to illness or death. Wilson & Daly (1997) showed that teenage pregnancy was most frequent in neighbourhoods with the lowest life expectancy, using demographic data from Chicago.

Geronimus *et al*. (1999) also mentioned that while 83% of black grandmothers in the USA would survive able-bodied to their grandchild's 5th birthday if their daughter bears that child when she is aged 15 years, only 64% would survive able-bodied if their daughter waited until she is aged 30 years. So, by delaying pregnancy there is less chance of having the support of one's own mother. In this context it is important to notice that a considerable number of teenage pregnancies are planned (Nettle *et al*. 2010).

Expected future lifespan may determine important decisions, such as when to reproduce, even though the factor is not necessarily conscious (Wilson & Daly, 1997). Therefore, it should be considered in further research that the underlying cause of teenage pregnancy is the reduced life expectancy.

Declaration of Interest

None.

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The authors reply

We thank Dr Hubbeling for her interest in our recently published paper on premature mortality risk among women who became teenage mothers in England and Wales. The suggestion that the association between these two phenomena at a whole-population level may be explained as an adaptive biological response to reduced life expectancy is an interesting one. However, we believe it is unlikely that such an adaptive response is the chief causal mechanism that explains our findings, for several reasons.

First, if this were the case, we would also expect to observe a population-level relationship between reduced life expectancy and earlier age at menarche. Using data from the British National Child Development Study (NCDS), Boldsen & Mascie-Taylor (1992) analysed the geographical distribution of age at menarche across Great Britain in two successive generations. They showed a mean earlier menarche age in the eastern and southern parts of the country than in northern areas, independent of social class differences. However, life expectancy is known to be

considerably lower in Scotland and in the northern regions of England than in the rest of Great Britain (Leyland, 2004).

Second, Dr Hubbeling cites evidence from the USA in support of her theory, showing that teenage pregnancy was more frequent in Chicago neighbourhoods with reduced life expectancy (Wilson & Daly, 1997). This association is no doubt to be found across many nations in the developed world, but it does not necessarily indicate the population-level adaptive mechanism in reproductivity that Dr Hubbeling has postulated. Material deprivation and reduced life expectancy are also strongly associated (Raleigh & Kiri, 1997), and girls brought up in socially deprived families are much more likely to become teenage mothers (Hobcroft & Kiernan, 2001). Therefore, we suggest that the proposed adaptive mechanism is likely to be explained by confounding factors. Other mechanisms related to deprivation may also explain why some girls actively plan to become mothers during their teens. For example, those with low levels of educational attainment and diminished expectations for their future life-chances may view motherhood as a pathway towards enhanced social status, or as a way of escaping an unhappy, dysfunctional or abusive family home (Wahn *et al*. 2005). Intergenerational effects may also have a strong influence (Seamark & Pereara Gray, 1997).

Third, we should emphasise that the absolute risk of premature death occurring many years below mean UK female life expectancy was extremely low among the women who became teenage mothers in our study. Although we did not have complete follow-up through the life course in our cohort study, almost all of these teenage mothers will survive into their sixties, seventies or eighties. Furthermore, the increase in risk that we observed, compared with age-controlled women who were not mothers, was modest (around 30%). This combination of a low absolute risk and a modest elevation in relative risk means that the effects we have reported are subtle ones in quantitative terms. As virtually all teenage mothers in the UK are long-term survivors, we feel that, although Dr Hubbeling's theory of adaptive reproductivity is interesting, it lacks the face validity and plausibility to explain our recently reported findings.

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Letter to the Editor

Mental disorders as mechanistic property clusters

In their insightful article, Kenneth Kendler, Peter Zachar and Carl Craver recommend the programmatic modelling of psychiatric disorders as kinds of mechanistic property clusters (MPC) (Kendler *et al*. 2010). According to this view, mental disorders are individuated by the whole cluster of mechanisms involved in the causation of their respective clinical syndromes. As the authors assert, 'the identity of the disease […] is grounded in the similarity of the complex, mutually reinforcing network of causal mechanisms in each case' (p. 6). However, on the same page a few lines below, they also claim that since 'the same cluster of symptoms might arise from different mechanisms', 'MPC kinds are […] "multiply realizable" '. I find this claim inconsistent with their previously cited assertion. If the identity of MPC kinds is grounded in the complex network of their causative mechanisms, then they cannot be 'multiply realizable'. What are 'multiply realizable' are not MPC kinds, but clusters of strongly similar clinical signs/symptoms. Instead, the MPC model clearly implies, however, that mental disorders sharing the same cluster of clinical signs/symptoms, emerging through