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.

References

Boldsen JL, Mascie-Taylor CGN (1992). Geographical variation in age at menarche in Britain. *International Journal of Anthropology* **7**, 1–6.

Hobcroft J, Kiernan K (2001). Childhood poverty, early motherhood and adult social exclusion. *British Journal of Sociology* **52**, 495–517.

Leyland AH (2004). Increasing inequalities in premature mortality in Great Britain. *Journal of Epidemiology and Community Health* **58**, 296–302.

Raleigh VS, Kiri VA (1997). Life expectancy in England: variations and trends by gender, health authority, and level of deprivation. *Journal of Epidemiology and Community Health* **51**, 649–658.

Seamark CJ, Pereira Gray DJ (1997). Like mother, like daughter: a general practice study of maternal influences on teenage pregnancy. *British Journal of General Practice* **47**, 175–176.

Whan EH, Nissen E, Ahlberg BM (2005). Becoming and being a teenage mother: how teenage girls in South Western Sweden view their situation. *Health Care for Women International* **26**, 591–603.

Wilson M, Daly M (1997). Life expectancy, economic inequality, homicide, and reproductive timing in Chicago neighbourhoods. *British Medical Journal* **314**, 1271–1274.

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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

different networks of causative mechanisms, constitute distinct MPC kinds, each one uniquely 'realized'. Besides, this view is already – although only implicitly and rudimentarily – endorsed in contemporary psychiatric classification systems. More precisely, the endorsement of this view is reflected in their systematic distinction of presumably 'primary' mental disorders of still unknown causation from 'secondary' mental disorders due to diagnosable general medical conditions, irrespective of the otherwise possibly strong similarities in their clinical manifestations.

Declaration of Interest

None.

Reference

Kendler KS, Zachar P, Craver C (2010). What kinds of things are psychiatric disorders? *Psychological Medicine*. Published online: 22 September 2010. doi:10.1017/S0033291710001844.

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The authors reply

Our essay addresses two closely related but distinct questions. The first question is 'What does it mean to say of a given psychiatric disorder that it is a kind of disorder rather than, for example, a more or less arbitrary collection of properties that happen to co-occur?' To this, we answer that kinds of disorders, or the things we ought to count as kinds, are not arbitrary collections of individuals but rather property clusters in which the properties tend to co-occur because there exists some underlying mechanism that explains their co-occurrence. Nothing in this view of kinds demands that there should be only one underlying mechanism, either in a single afflicted individual or across a population. Kinds are regular clusters of properties sustained by one or more mechanisms such that they are regular, repeatable, predictable, and at least potentially manipulable. The second question is: What individuates kinds of psychiatric disorders? What makes one kind different from another? How do we know when we have one kind as opposed to two or more hidden under the same label? In answer to that, it is tempting to say (beyond the answer to the first question) that kinds correspond in a one-to-one way with mechanisms: if there are two kinds of mechanism for one property cluster, then there are in fact two higher-level kinds, one for each mechanism. This answer to the second question, however, would be inconsistent with multiple realizability, as multiple realizability is just the idea that the same kind can be realized by different mechanisms. Our essay defends the MPC as an answer to the first question while trying to resist the claim that it provides a complete answer to the second. Given that differences in underlying mechanisms often do signal differences in property clusters, it makes sense to sort psychiatric disorders in part on the basis of what we learn about their biological mechanisms. Superficially similar syndromes might be underwritten by different underlying mechanisms, but slightly different mechanisms could be seen as producing highly similar outcomes. However, it was not a goal of our essay to deal more deeply with this thorny and long-debated issue – that is the relative merits and practicalities of using 'surface features (i.e. symptoms and signs) or etiology (=mechanism) to undergird a psychiatric nosology.

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