and schizophrenia may not be a causal relationship. The authors hypothesise that the association may be explained by a broad genetically determined phenotype of schizotypal personality types, which lead to high-risk lifestyle in the mother, which in turn contributes to OCs in the birth. This is a highly interestng hypothesis that warrants empirical testing.

However, a demonstration of an increased occurrence of OCs in births to schizophrenic or schizotypal mothers would of course not exclude an independent effect of perinatal and birth complications on the risk for adult schizophrenia.

Sacker *et al* also find that the associations between OCs and schizophrenia demonstrated elsewhere may be explained partly by information bias. The irregular lifestyles and lack of attendance to antenatal care of the mothers of schizophrenics may lead the midwife to expect and record more obstetric complications in this group than among controls. However, it is not clear to us how the authors have demonstrated this selective underreporting in controls in other studies, and it would appear that this is at least partly refuted in their own study. The significant variables as, for example, birth weight less than 2500 g would not seem to leave much room for personal judgement by the reporting midwife. Also in their first report of the study (Done *et al*, 1991), the high quality of the data was emphasised.

The word "negative" (page 734) is not the most precise characterisation of the prior work by Done *et al*, given the statistical power of that study, because the direction of the finding is a positive association, and the odds ratio is not trivial (1.4). Indeed, the present paper by Sacker *et al* seems to confirm and specify this positive association in more detail.

DONE, J., JOHNSTONE, E. C., FRITH, C. D., *et al* (1991) Complications of pregnancy and delivery in relation to psychosis in adult life: data from the British perinatal mortality survey sample. *British Medical Journal*, **302**, 1576–1580.

SACKER, A., DONE, J., CROW, T. J., *et al* (1995) Antecedents of schizophrenia and affective illness. Obstetric complications. *British Journal of Psychiatry*, **166**, 734–741.

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AUTHOR'S REPLY: Rifkin & Takei refer to "the lack of direct testing of the hypothesis", and Mortensen *et al* state "it is not clear to us how the authors have demonstrated this selective underreporting in controls". However we wrote (p. 739)

> ". . . previous studies have been under-reporting OCs in the controls and that this has contributed to the illusion of raised OCs in the schizophrenics. Lewis & Murray's (1987) composite scale provides rates of complications around 17% in the normal population and between 32% and 37% for the births of schizophrenics when derived from birth records (Nimgaonkar *et al*, 1988; O'Callaghan *et al*, 1992). This compares with rates of 39% for controls and 38% for schizophrenics when the same scale was used in our study. Werner and colleagues (1971) found that 44% of births in their Island of Kauai prospective cohort study incurred an obstetric complication".

Is it reasonable to suppose that midwives may systematically under report in some cases and over report in other cases? The use of guidelines, and procedures to ensure adherence, which were used in the Perinatal Mortality Study can have a marked impact (Emslie *et al*, 1993) and as such have become a priority topic in the latest NHSE R&D Implementation Programme.

Rifkin *et al* suggest that OCs that have been reported in the literature are unmissable to the midwife. This is untrue. Some of the key studies have needed to use 'equivocal OC' (sic), or 'mild complications' to obtain a significant odds ratio. Furthermore the only individual OC singled out in McNeil's (1987) review was 'prolonged labour', which is an 'equivocal OC' on rating scales, and may not even be a true OC (O'Callaghan *et al*, 1990). An equivocal measure may or may not be recorded depending on its perceived importance under the prevailing circumstances. Our conjecture is that 'equivocal OCs' are more likely to be recorded when the midwife is concerned about the competence of the mother.

Rifkin *et al* suggest that the Perinatal Mortality Survey 1958 focused on 'less salient OCs'. This is wrong. Appendix 1 provided a list of all variables examined which included all of those mentioned by Rifkin *et al*.

Contrary to the claim of Rifkin *et al* and Mortensen *et al* low birth weight has not been consistently reported in the studies of OCs in schizophrenia. We have counted five studies with negative results and four studies reporting raised OCs for an aggregated score only. Were birth weight to be reduced in the group of schizophrenics it would surely have been mentioned in these latter studies. Furthermore McNeil does not single out

https://doi.org/10.1192/S0007125000075814 Published online by Cambridge University Press

low birthweight in his 1978 review. Miller *et al* (1987) have reported that low birthweight is associated with a large number and variety of risk conditions, often acting multiplicatively, which include cigarette smoking, lack of prenatal care, physique of mother, teenage pregnancy, behavioural and environmental risks, health care risks, drug addiction and chronic alcoholism. We found that some of these were raised in the mothers of schizophrenics and in addition those not documented in the 1958 study (such as drug addiction and alcoholism) are known to be found more often in first degree relatives (i.e. parents). Thus if birthweight does turn out to be lower in people with schizophrenia it may well be due to multiple risk arising from the physique and lifestyle of the mother.

Rifkin & Takei's final point missed the analysis reported on p. 787. A logistic regression, with schizophrenia of cohort member as the dependent variable, was calculated with *any* parental psychological disorder fitted first before fitting the physique/lifestyle variables identified earlier. Even when parental psychological disorder (which included schizophrenia in parents) was removed the increased risk in the parents of schizophrenics remained on a number of measures.

We would agree with Mortensen *et al* that a debate is long overdue on alternatives to the theory that minor OCs cause a neurodevelopmental disorder later presenting as schizophrenia. The voluminous literature suggests that Lilienfeld *et al*'s 1950s concept of "a continuum of reproductive causality" has not stood the test of time, but this has been overlooked. For example psychological sequelae of pregnancy/perinatal damage are widely reported to be modulated by social and familial environment. Good environments foster and poor environments fail to provide the potential for recovery. Since early socio/environmental disadvantage does not increase the risk of schizophrenia, this diathesis is unlikely to be found. Alternative explanations for the association between OCs and schizophrenia will then be required.

EMSLIE, *et al* (1993) Do clinical guidelines improve general practice management and referral of infertile couples. *British Medical Journal*, **306**, 1728–1731.

MCNEIL, T. F. (1987) Perinatal influences in the development of schizophrenia. In: *Biological Perspectives of Schizophrenia* (eds H. Helmchen & F. A. Henn) Chichester: John Wiley.

O'CALLAGHAN, E., *et al* (1990) Obstetric complications in schizophrenia and the validity of maternal recall. *Psychological Medicine*, **20**, 89–94.

MILLER, H. C., *et al* (1987) Incidence of low birth weight infants born to mothers with multiple risk factors. *Yale Journal of Biology and Medicine*, **60**, 397–404.

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Eugenics

SIR: The editorial by Pearson (1995) concludes with the warning that opposition to the Chinese eugenicist birth policy law will be more effective if it eschews the "high moral ground", and focuses rather on the fact that such a policy is unscientific. This caveat, if indeed honoured by 'Westerners', may or may not prove to be prescient; but, either way, it is an unwise path to follow.

Policies of eugenics are bound inextricably to salient issues of morality. To pretend otherwise, or to focus on its unscientific nature to the exclusion of pressing moral concerns, constitutes an unconscionable, and improper, breach of academic candor and forthrightness. Although Pearson (1995) comments that moral arguments against eugenics may stir up, in the minds of Chinese, a spectre of cultural imperialism, it is hard to fathom how this may come about if the morality of eugenics is debated in an intellectual environment permeated by even a modicum of free expression of thought. To deliberately fail to stress, or even mention, that, historically, eugenics policies have been fettered by indissoluble bonds to pressing moral concerns appears to constitute, ultimately, a foolish, misguided attempt to grovel at the feet of those ignorant of historic truths, or else bent intentionally on doing wrong. Historically, attempts to appease those suffering from these latter ailments have failed; and they will likely fail afresh, if attempted now.

The correct path to follow is to emphasise that eugenics policies, historically, have thrived in the soil of authoritarianism and dictatorship. And that the present Chinese eugenicist policies are scions of historical roots which thrived during the barbarism of Hitler's Third Reich. Indeed, for many, the term eugenics will immediately conjure up images of Nazi Germany and widescale euthanasia. Any honest discussion of eugenics should mention also that the burgeoning number of gene tests under furious development have tentacles reaching potentially to eugenics policies, as well as policies of genetic-related discrimination extending to insurance and employment (Uzych, 1986, 1992, 1995).

China, in sum, is sliding rapidly down a path towards moral degradation; and this disturbing development should not be ignored.

https://doi.org/10.1192/S0007125000075814 Published online by Cambridge University Press