CASE REPORT/JOURNAL CLUB


This study used semi-structured interviews with 22 general practitioners and 35 practice nurses from a variety of general medical practices across Sheffield (a city in the north of the UK). The aim was to identify the barriers to talking about the discussion of sexual health. The participants had particular difficulties discussing sexual health with groups of patients where they did not know how opening up the subject would be received and where they anticipated embarrassment. They identified as particularly difficult: patients of the opposite gender to themselves, patients from ethnic minority groups, middle-aged and older patients and non-heterosexual patients. This tells us about the preconceptions of the participants and their lack of training and experience in discussing sexual health.

The other main barrier was the perception that asking about sexual health would ‘open a can of worms’, that is, that it would reveal information that would take too much time to deal with. Perhaps the description of ‘a can of worms’ also describes the feeling that ‘unsavoury’ information would be revealed? The participants felt that discussing sexual health matters in primary care created problems because of the sensitivity and complexity of the material. They also felt constrained by lack of time and expertise.

This study certainly reinforces other studies showing that the role of primary care in providing sexual health services cannot be expanded without training and education for the people who are to deliver the service. Training and education can remove the anxiety about showing that the role of primary care in discussing sexual health.

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This cohort study was undertaken in Maryland, MD, USA following up three groups of women: two groups initiating hormonal contraception (i.e. depot-medroxyprogesterone acetate (DMPA) or oral contraception (OC)) and a group of controls not using hormonal contraception but attending the same Planned Parenthood clinics for gynaecological care. Detailed and carefully thought through exclusion criteria were applied to potential participants in the study, and to time-segments of follow-up. Extensive data were collected on the baseline characteristics of the groups, and pre-existing infections were treated and confirmed cleared before entry to the study. Time-varying risk factors and clinical signs were measured prospectively, and at each follow-up appointment there was high-quality testing of chlamydial infection and careful checking of actual contraceptive use. Sophisticated statistical methods were used to model hazard of cervical infection for each of the two hormonal contraception groups (OC and DMPA), relative to controls (non-hormonal contraception), and to adjust for individual baseline and time-varying covariates.

This analysis showed that young age (15–17 years), two or more sex partners, inner-city site of clinic attended, ethnicity (non-white) and DMPA use (relative to non-hormonal contraception) were all statistically significantly associated with increased risk of acquiring cervical infection. The paper concludes that use of DMPA, but not of OC, appears to be associated with increased acquisition of cervical chlamydial and gonococcal infections. Readers should note and remember the essential qualification ‘appears’.

A necessary factor in acquisition of new cervical infection is that one of a woman’s sexual contacts is already infected. Behavioural factors can increase the likelihood of this circumstance (more partners increases the chance of an infected partner) and, where a partner is infected, behaviour such as non-use of condoms will facilitate transmission of that infection. In addition, certain physiological or hormonal factors may mediate acquisition of infection (perhaps youth, hormonal status, etc.). Conversely, if none of a woman’s partners is infected then behavioural and physiological factors are irrelevant. Unbiased comparison of rates of new infection therefore requires similar background pools of infection in the contraceptive use groups to be compared. This was not the case in this study, since the baseline (pre-existing) rate of chlamydia in the DMPA group was nearly three times that in the OC group, and nearly double that in the control group (8.9%, 3.1% and 4.6%, respectively). No amount of adjusting for facilitatory behavioural or physiological factors can compensate for fundamental differences between the groups in infectious potential. [It is irrelevant whether individual cases of chlamydia detected at baseline were treated and cleared, and were not on the whole re-infected during follow-up. The judgement being made here is about the pre-existing level of infection prevalent in the sexual contacts of the group, which is best estimated by infection rates at baseline.] An observational not a randomised design was chosen because the fact that “most participants had initiated contraception and had a particular contraceptive method in mind” made it neither ethically nor practically feasible to randomise. However, the failure to randomise runs a very strong risk of confounding of study findings by factors other than contraceptive use. The crucial potential impact of group-level pool of infection has been considered above, but in addition there were marked differences between the groups in individual behavioural and ‘physiological’ characteristics, at baseline and during follow-up. The extent and direction of these differences makes it very unlikely that the multivariable adjustment applied will have accounted for all confounding by these individual-level characteristics. No adjustment was possible to control for pre-existing between-group differences in infection pool size.

A further concern about the analysis is that the comparison made (in the model used) were of DPMA vs controls, and OC vs controls, with no direct test of DPMA vs OC. However, the latter comparison would seem to be the more obvious one to have been made, if wishing to reflect on mechanisms for increased acquisition of infection that are specific to DPMA, which is the thrust of the discussion. The comparisons actually made, of each hormonal group vs controls, are really rather trivial. It would be surprising if initiation of hormonal contraception was not associated with changes in behavioural factors facilitating infection. For example, during the follow-up controls did not report a change in prevalence of ‘always’ using condoms, but both hormonal groups showed a marked reduction in this protective behaviour – from at least 30%, down to 13%. Women with infected partners who up to study entry had always used condoms, but stopped once reliable hormonal contraception was initiated, would then become ‘at risk’ of infection. The extent of this risk in the two hormonal study groups would depend on their pre-existing infection pools, and even more so on the known infection status of current partners, which differed significantly across the two groups.

Three reasons for caution have been noted:

- uncontrolled differences in background infectivity of sexual contacts across the three groups
- marked differences between groups in individual risk factors for infection, to the extent that multivariable adjustment is unlikely to have accounted for all confounding
- failure to compare OC and DPMA directly in the model.

Readers should be aware that for these study results no inferences may be misleading.

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