# Measuring paternal discrepancy and its public health consequences

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Paternal discrepancy (PD) occurs when a child is identified as being biologically fathered by someone other than the man who believes he is the father. This paper examines published evidence on levels of PD and its public health consequences. Rates vary between studies from 0.8% to 30% (median 3.7%, n = 17). Using information from genetic and behavioural studies, the article identifies those who conceive younger, live in deprivation, are in long term relationships (rather than marriages), or in certain cultural groups are at higher risk. Public health consequences of PD being exposed include family break up and violence. However, leaving PD undiagnosed means cases having incorrect information on their genetics and fathers continuing to suspect that children may not be theirs. Increasing paternity testing and use of DNA techniques in clinical and judicial procedures means more cases of PD will be identified. Given developing roles for individual's genetics in decisions made by health services, private services (for example, insurance), and even in personal lifestyle decisions, the dearth of intelligence on how and when PD should be exposed urgently needs addressing.

> or any father, identifying that the child they are raising as their biological progeny is actually sired by another man (paternal discrepancy (PD)) can have substantial health consequences. Such knowledge can also destroy families;1 affecting the health of the child and mother as well as that of any man who is ultimately identified as the biological parent.<sup>2</sup> Typically, PD is associated with a woman having a sexual relationship (usually covertly) outside of her marriage or long term partnership. Here PD occurs when a child is believed to have been fathered by the husband (or partner) but is actually the progeny of another man. Pregnancy may be accidental but occasionally may be the reason for infidelity (for example, where sex with the long term partner has not produced children a woman might seek conception elsewhere3). PD also occurs without infidelity. Where a woman quickly changes from one sexual relationship to another, a pregnancy resulting from a previous partner can be wrongly attributed to a new partner. Rarely, PD occurs because of medical mistakes including mix ups of semen during artificial insemination and in vitro fertilisation.4

Increased understanding of human genetics<sup>5</sup> and, more recently, widespread public access to genetic identification techniques now means that almost anyone can establish the biological parentage of their children.67 Moreover, along with an increase in parentage testing<sup>8</sup> health services now use genetic techniques in diagnosis9 and treatment,10 with criminal justice organisations also using genetic techniques in crime detection.<sup>11</sup> Such techniques can inadvertently uncover inconsistencies in a family's genetics that disclose PD.12 However, while the opportunity to expose PD through paternity testing or routine health and judicial procedures has increased, little consideration has been given to the consequences. Here, we collate existing evidence on the prevalence of PD, review how increasing use of genetic techniques will continue to reveal more cases, and examine the public health consequences of people having greater need for, and access to, such knowledge.

#### **METHODS**

Titles and abstracts of peer reviewed scientific literature (PubMed 1950-2004 including Medline 1966–2004, BIDS International Bibliography of the Social Sciences 1951-2004, PsychINFO 1887-2004) were interrogated for references to the prevalence of PD, mechanisms for its detection, and the potential health consequences of PD being exposed. The key search terms used were: nonpatern\*; non and patern\*; and father matched with discrepancy, uncertainty, misattributed, false and investment. Peer reviewed papers were supplemented by reports from conference abstracts, books, and other scientific reports (table 1). As relevant literature was not associated with any particular journals hand searching13 was not undertaken on any journal's entire contents but references listed within all identified literature were examined for additional relevant papers. Using all available data we used discursive qualitative techniques to assess the evidence for PD. Thus, all papers were examined separately by two authors for references to PD, sampling characteristics, methodology for identification of PD, and potential bias inherent in studies that have measured PD but usually not been designed for that purpose (see table 1). Where authors' classifications conflicted this was resolved by a third author examining the document.

Literature reviews and, where necessary, original research literature were also examined to

Abbreviations: PD, paternal discrepancy; STI, sexually transmitted infection

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identify developments in the use of DNA techniques that have disclosed or could potentially disclose PD. Finally, although few publications deal with how demographics may affect levels of PD and we found no papers dealing directly with how exposing PD could affect health, we use a combination of extensive literature reviews and original research literature on sexual behaviour and the health correlates of different social structures to address each issue respectively.

#### RESULTS

#### How common is paternal discrepancy?

Few studies have been undertaken specifically to estimate population levels of PD<sup>14</sup> and most evidence is based on data collected for other purposes (table 1). Historically, comparisons of family members' blood groups (ABO and rhesus) either collected for blood donation or for other purposes provided some estimates of PD (table 1). More recently, investigations of familial patterns of disease inheritance have identified PD15 and led to further estimates of its prevalence (table 1). An additional source of estimates results from commercial and public organisations offering tests to fathers who already suspect PD (table 1). Such studies are no substitute for population surveys and contain biases that either exaggerate or underestimate population levels of PD. Thus, PD estimates based on men or women seeking proof of paternity can overestimate levels of PD where paternal uncertainty was usually the motivation for testing. In contrast, estimates based on genetic health screening and other studies (where confirming paternity was not the objective) may underestimate PD as people can refuse to participate or are excluded<sup>15</sup> when subjects or investigators consider paternity in doubt. Estimates can also include anomalies that seem to be PD but result from other social phenomenon. Thus, people may adopt a child or conceive through AID (artificial insemination by donor) but keep such information hidden. Equally, friends or relatives occasionally raise a child as theirs when the mother is too young, unwell, considered inappropriate, or has abandoned the child.16 Historical blood type data or even modern data identifying relatives of natural disaster and terrorist attack fatalities17 18 can include such anomalies unless family histories are available. Here, to estimate population levels of PD we have included all identified published estimates of PD except where they do not include at least basic methodological details and sample sizes or are based on historical data over multiple generations.<sup>19 20</sup> We have also excluded estimates derived solely from behavioural studies that have not included biomolecular marker testing (table 1). For the remaining studies we examine two types of PD rates. For disputed paternity tests median levels of PD across 16 studies is 26.9% (interquartile range (IQR) = 16.7%–33.4%). However, being based on cases where PD was already suspected this inevitably overestimates population levels (table 1). For studies based on populations chosen for reasons other than disputed paternity (table 1) median PD is 3.7% (IQR = 2.0%-9.6%). While this is not a measure of population prevalence it does suggest the widely used (but unsubstantiated) figure of 10% PD<sup>21</sup> may be an overestimate for most populations.

#### Who will PD affect most?

While few studies have measured demographic effects on levels of PD, higher rates have been found among people from lower socioeconomic groups.<sup>14</sup> Furthermore, existing data on sexual behaviour permit some measure of those populations most at risk.<sup>22 23</sup> Increased risk of PD is seen among people with concurrent sexual partners. As having concurrent sexual partners occurs more at earlier ages,

younger women are at highest risk (for example, British women with concurrent sexual partners in past 12 months; 16-24 years = 15.2%, 25-34 years = 7.6%<sup>22</sup>). Prevalence of women with concurrent partners has increased over the past decade (for example, Britain<sup>22</sup>). Consequently, girls who conceive at early ages may have greater chances of PD with first pregnancies having been shown to be at higher risk.<sup>24</sup> One in five women in marriages or long term relationships in the UK have had affairs and similar figures are reported from most developed countries.25 However, higher rates of infidelity are seen among pairs who are not married.<sup>26</sup> Furthermore, time spent apart in marriages or long term relationships (for example, through occupational travel) is also associated with higher levels of infidelity as is living in higher population densities.<sup>27</sup> Sexual risk taking (measured for instance by levels of sexually transmitted infections (STIs)) has also been associated with higher levels of deprivation as well as ethnic and cultural issues.<sup>28</sup> <sup>29</sup> Thus, in the USA, African Americans' rates of gonorrhoea can be 20 times higher than their white counterparts,<sup>30</sup> while Hispanic adolescents have birth rates 2.9 times those of non-Hispanic white adolescents.31 Studies in the UK also show similar ethnic differences in sexual risk<sup>29</sup> and limited analyses of PD suggest higher rates among some ethnic groups.32 Thus, ethnicity as well as lower socioeconomic class,<sup>14</sup> younger age, and higher levels of deprivation seem to be risk factors for both PD as well as other sexual health issues (for example, teenage pregnancy and STIs<sup>33</sup>).

#### Increases in techniques that identify PD

Genetic techniques are becoming increasingly central to modern medicine. Both the number of conditions thought to be related to a person's genetics (for example, cystic fibrosis<sup>34</sup>; coronary heart disease<sup>35</sup>; cancer<sup>36</sup>; obesity<sup>37</sup>) and the number of DNA molecular tests undertaken continues to increase (UK9). The role of genetics will increase as more diseases are related to genetic predispositions<sup>5</sup> and treatments become tailored to a patient's genome.<sup>38</sup> Often, genetic screening can be triggered by a child, parent, or other relative developing a genetic disease and consequently, many family members will be screened to determine who else is at risk and the exact nature of the genetics.<sup>39</sup> Such tests are essential for clinicians and patients to make vital decisions regarding lifestyle,40 terminations of pregnancy,41 whether to conceive at all and types of treatment<sup>42</sup> but will also identify PD. In these circumstances, there are clear advantages to patients understanding their actual genetic inheritance, in particular in allowing them to rule out genetic conditions experienced by their social father and instead take into account those relating to their biological father. Equally for health professionals in general, measuring PD is essential to understanding the genetics of health and ill health43 with discounted PD confusing estimates of heritability and potentially inhibiting development of genome based interventions.

Two further expanding health areas also expose PD. Organ donation, particularly when close family is screened for potential donors, can identify PD (for example, kidney donation<sup>44</sup>). Equally, examination of male fertility can identify people who are infertile and unlikely to have ever been fertile. PD is exposed when this diagnosis occurs in families where the husband (or long term partner) already believes he has fathered one or more children.<sup>45</sup>

Criminal investigations increasingly rely on DNA techniques to identify culprits and important investments have been made to develop DNA databases of criminals (for example, the National DNA Database, UK<sup>46</sup>). Such databases have already been used to identify relatives of criminal offenders<sup>47</sup> and consequently have the potential to expose

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UK USA USA USA USA USA USA South America USA New Zealand Mexica UK Erance Canada Nexica Mexica UK Behavioural estimates UK USA	Southern English families Undisputed paternity tests Wichigan white sample Additiogan black sample Caditionian white sample Caditionian trabe Southern English families Yanomana trabe Hawaina families Cesnit fahrosis screening Genetic screening (various) Haemophilia B screening Genetic screening (various) Haemophilia B screening Cystic fahrosis screening Nuevo Leon new borns Nuevo Leon new borns Multiple sclerosis screening Multiple sclerosis screening Magazine readers College undergraduates	2578 67 1417 53 523 523 6960 2000 1983 200 217 283 285 285 285 285	$\begin{array}{c} 3.7 \left( 3.0 \ \mathrm{b} \ 4.4 \right) \\ 18.0 \left( 8.5 \ \mathrm{b} \ 2.7 \left( 3 \right) \\ 10.1 \left( 7.5 \ \mathrm{b} \ 2.2 \right) \\ 10.1 \left( 7.5 \ \mathrm{b} \ 2.2 \right) \\ 2.7 \left( 2.3 \ \mathrm{b} \ 3.4 \right) \\ 30.0 \left( 2.3 \ \mathrm{b} \ 3.4 \right) \\ 9.0 \left( 4.1 \ \mathrm{b} \ 14.1 \right) \\ 9.0 \left( 4.1 \ \mathrm{b} \ 14.1 \right) \\ 7.0 \left( 4.1 \ \mathrm{b} \ 2.8 \right) \\ 4.0 \left( 3.1 \ \mathrm{b} \ 4.9 \right) \\ 7.0 \left( 3.1 \ \mathrm{b} \ 4.9 \right) \\ 2.9 \left( 0.5 \ \mathrm{b} \ 5.0 \right) \\ 1.4 \left( 0.4 \ \mathrm{b} \ 2.3 \right) \\ 2.8 \left( 1.1 \ \mathrm{b} \ 4.5 \right) \\ 2.8 \left( 1.1 \ \mathrm{b} \ 4.5 \right) \\ 2.8 \left( 1.1 \ \mathrm{b} \ 4.5 \right) \\ 2.9 \left( 0.6 \ \mathrm{b} \ 5.0 \right) \\ 1.1 \left( 8 \left( 9 - 1 \ 3 \right) \right) \\ 1.1 \left( 8 \left( 9 - 1 \ 3 \right) \right) \\ 1.1 \left( 8 \left( 9 - 1 \ 3 \right) \right) \\ 1.5 \left( 0.7 \ \mathrm{b} \ 2.5 \right) \\ 6.9 \ \mathrm{b} \ 13.8 \\ 6.9 \ \mathrm{b} \ 13.8 \\ 6.9 \ \mathrm{b} \ 13.8 \\ 13.0 \ \mathrm{b} \ 20.0 \ \mathrm{b} \ 10.0 \ \mathrm{b} $	Blood and other markers Blood and other markers DNA testing Mixed methods Blood and other markers DNA testing Mixed methods Blood and other markers Blood and other markers	not known not known not known not known not known not known nor-participation in sample (-) some suspected non-paternity (+) not known non-participation in sample (-) non-participation in sample (-)	Edwards, 1957 <sup>74</sup> Sussman and Schatkin, 1957 <sup>75</sup> Schadrt and Gershowitz, 1963 <sup>76</sup> Schadrt and Gershowitz, 1963 <sup>76</sup> Peritz and Rust, 1972 <sup>77</sup> Philipp, 1973 <sup>86</sup> Neel and Weiss, 1975 <sup>76</sup> Asthon, 1980 <sup>86</sup> Samon <i>et al</i> , 1980 <sup>81</sup> Lathrop <i>et al</i> , 1980 <sup>81</sup> Lathrop <i>et al</i> , 1982 <sup>84</sup> Poon <i>et al</i> , 1992 <sup>84</sup> Poon <i>et al</i> , 1992 <sup>84</sup> Sasse <i>et al</i> , 1992 <sup>84</sup> Cerda-Flores <i>et al</i> , 1999 <sup>85</sup> Bellis and Baker 1990 <sup>88</sup> Bellis and Baker 1990 <sup>88</sup>
* All populations in "other testing" art the table. However, this does not take markers methods usually rely on ABC remain undetected. With DNA tests (+) = likely to overestimate PD and (-) avoided by those concerned that PD biomolecular markers to estimate PD	r testing" are after birth. FCI, confidence interva does not take into account sampling and other r rely on ABO and rhesus blood groupings or hu DNA tests polymerase chain reaction and rest a PD and (-) = likely to underestimate PD. All di ned that PD will be exposed and consequently estimate PD.	<li>Is. 95%Cls wer nethodologica man leucocyte riction fragme puted paternit may underesti</li>	e not included in most p l'variations between stu a antigen differences. In nt length polymorphism by testing is likely to rear mate PD. Not known is	apers reporting levels of PD. He dies. 95%CIs have not been cal studies using these methodoog or are commonly used and PD d uit individuals who already sust uit individuals who already sust	re, we have calculated all confidence inter culated for behaviour based estimates as jies calculations of PD prevalence often in prevalence area are usually sensitive enough effection rates are usually sensitive enough meet PD and results exaggerate population direction of any bias is unclear. ¶ Behavi	* All populations in "other testing" are after birth. FCI, confidence intervals. 95%CIs were not included in most papers reporting levels of PD. Here, we have calculated all confidence intervals based on the sample size and percentage included in the table. However, this does not take into account sampling and other methodological variations between studies. 95%CIs have not been calculated for behaviour based estimates as these have been published as ranges. ‡ Blood and other mackers methods usually rely on ABO and thesus blood groupings or human leucocyte antigen differences. In studies using these methodologies calculations of PD prevalence often include a corrective factor to account for discrepancies that manimulated test behaviour based estimates as these have been published as ranges. ‡ Blood and other methodologies calculations of PD prevalence often include a corrective factor to account for discrepancies that manimulated test with the exposed and and externation fragmant length polymorphism are commonly used and PD detection rates are usually rely to underestimate PD. All disputed paternity testing is likely to recruit individuals who already suspect PD and results exaggered population levels. Cenetic gor health reasons is likely to be avoided by those contermed that PD will be exposed and consequently may underestimate PD. Not known is entered next to studies where direction of any bias is unclear. ¶ Behaviour based estimates rely an questionnaires rely to the stander PD will be exposed and consequently may underestimate PD. Not known is entered next to studies where direction of any bias is unclear. ¶ Behaviour based estimates rely on questionnaires rather than biandecular and these contented PD and test.

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### **Policy implications**

- As advances in genetic techniques allow paternal discrepancy to be identified, clear guidance is necessary on when and how it is disclosed.
- Individual and family support services need to be integrated into the paternity testing service and supported by appropriate training.
- Sufficient evidence is already available to suggest paternal discrepancy affects the health of many people. Appropriately designed studies are now required to accurately measure its demographics and quantify its direct and indirect costs.
- Health and judicial procedures that can identify paternal discrepancy should have guidance on when and how paternal discrepancy should be exposed and such guidance should be publicly available.

unexpected anomalies including PD. Furthermore, both health and judicial systems increasingly rely on genetic testing in major disasters (for example, environmental catastrophes and terrorist attacks) to confirm the identity of those who may have perished; especially where bodies have been damaged beyond recognition.<sup>17 18</sup> Here, genetic sampling can expose PD where DNA results (matched to a parent, child, or other relative) conflict with other evidence (for example, clothing, jewellery found on corpses).

By far the most common means available for most people to test PD is through use of commercial testing kits with multiple web sites already advertising this service. People (usually concerned fathers) visit a clinic or send off for a testing kit and provide samples (check swabs, hair follicle samples) from themselves and the child for testing.<sup>6</sup> The number of tests undertaken annually continues to increase (USA, 1991 = 142 000, 2001 = 310 490<sup>48</sup>). Although some countries are considering changing legislation to try to stop fathers testing children without the permission of the mother, such legislation is unlikely to affect testing patterns as using services based abroad is comparatively simple.

#### Public health consequences of PD

Despite increasing use of, and access to, techniques that can identify PD, very little consideration has been given to the consequences of a family becoming aware of PD or what services and support are required when PD is exposed. Furthermore, even when PD is inadvertently identified by public agencies, a public health perspective is necessary to assess how such information should be used and if and when those affected should be informed.

A 4% PD would affect far more than 1 in 25 families. Given an average of two children per family, more families will be affected within just a single generation; although it is probable that PD will cluster in some family groups.<sup>25</sup> Typically however, many families have three or more living generations. Consequently, the proportion of families affected will increase further when other relationships (for example, between parents and grandparents) are also considered.

In addition, for each child resulting from PD there is also a biological father elsewhere and such people are often part of other long term relationships involving marriages and children.<sup>49</sup>

An important consequence of discovering infidelity in a marriage or other relationship is the eventual breakdown of that partnership.<sup>50</sup> Around 20% of divorces feature claims of infidelity by one or both partners (England and Wales<sup>51</sup>). The

#### What this paper adds

- Provides a broad review of paternal discrepancy rates and population characteristics related to its prevalence.
- Reviews the new methodologies used by health and judicial systems that have increased the likelihood of detecting paternal discrepancy.
- Examines the public health consequences both of disclosing paternal discrepancy and of keeping it undisclosed.
- Identifies the urgent need for better intelligence on demographics of paternal discrepancy and its effects on family structure and health.

effects of breakdowns in relationships include increased mental health problems for both partners52 while children can experience low self esteem, anxiety, and increased involvement in antisocial behaviour such as aggression.53 Other issues related to separations such as relocation of one parent and children can also have detrimental effects.<sup>54</sup> Not all disclosures of PD will result in relationships ending.44 However, those that continue must cope with a child in the family structure who is related to only one parent and sometimes the result of infidelity. Despite many mixed family structures working well, fathers spend more time and other resource on their biological children and, at worst, children in families where the father is not their own may be at greater risk of paternal violence.55 Suspected infidelity is also a trigger for domestic violence against women.56 Furthermore, people outside the family who are ultimately identified as true biological fathers may experience breakdown in their own relationships. With such outcomes relating to the results of paternity tests it is vital that they are accurate. However, some commercial companies have already been known to provide false results.57

Minimising the negative consequences of PD disclosure requires services and support to be immediately available. However, with PD testing even basic counselling is not always provided and those receiving results by letter, email, or over a web site can be effectively isolated. Although people might approach generic support services (for example, marriage guidance, general practice) in general these have little or no research regarding PD on which to base practice or advice. Effective practice and available support can be even scarcer for the mother, child, and for the man eventually identified as the biological father.

Although restricting access to commercial testing may seem appropriate, the public health impact of restrictions could also have negative consequences. Here we estimate that only around one in every four elective tests identify PD; the remainder confirm the father and child are biologically related (table 1). Again little is understood about the consequences to parents or children of the father suspecting PD but not having this established or refuted. Many are likely to be similar to having PD confirmed (that is, stress, possible family breakdown, and abuse). For three quarters of individuals, PD tests will allay their suspicions and may improve relationships.

The issues surrounding accidental disclosure of PD through health or judicial activity are no more clear cut. To date inadvertent identification of PD has usually been kept from those affected. However, more links between genetics and individuals' health are identified every day and consequently the case for the child to be informed is strengthened. Increasingly, the knowledge of genetic inheritance is not just

of use to clinicians but informs the lifestyle choices of the person,40 the decision to procreate,58 and in some cases access to insurance.<sup>59</sup> Consequently, a person left wrongly believing they are related to a father with a heritable condition will suffer some disadvantage. Disclosing PD in a controlled health care environment may also have substantially fewer health consequences than if later uncovered independently through commercial tests. Equally, as public understanding of heritability increases, inheritance patterns in families will allow people to identify (or suspect) PD themselves. Furthermore, the same increase in understanding will discourage people from using modern genetic techniques in case PD is disclosed.15 Overall, the health consequences of either revealing PD or maintaining confidentiality are strongly linked to the rights of the child, father, and mother and recent developments in assisted fertility (for example, in Sweden and the UK) now place the child's right to know their biological father above that of the donor (biological parent) to remain anonymous.60

#### CONCLUSIONS

Modern genetic techniques continue to open a Pandora's box on hitherto hidden aspects of human sexual behaviour. No clear population measures of PD are currently available. However, recent trends in sexual health suggest unprotected sex and multiple sexual partners (two key requirements for PD) are comparatively common occurrences<sup>21</sup><sup>22</sup> with a large proportion of conceptions still unplanned (around a third in the UK<sup>61</sup>).

Efforts to reduce PD may meet with some success. Improved contraception in at risk groups such as young people, who may be switching sexual partners, should help not only with STIs and unwanted pregnancies but also PD rates. Furthermore, PD offers another important reason to develop sexual health messages for older age groups, some of whom are still accumulating new sexual partners but sometimes in a more covert fashion. The availability of paternity testing kits themselves may also be used to convince some men that carefree sex and denial of paternity is no longer a viable option. However, no intervention will completely eliminate infidelity where historically even laws to make it punishable by death have failed (England, Adultery Act 1650). Equally, it is unlikely that any legislation will stop people purchasing and exploiting paternity testing technologies. Consequently, we must develop a better understanding of the prevalence and distribution of PD, the consequences of its disclosure or non-disclosure, and the interventions necessary to protect health when PD is disclosed.

Methods used in this paper identified a distinct lack of well designed population surveys. However, the lack of a disciplinary focus for PD studies (which appear in biological, behavioural, medical, and genetic literature) means despite extensive efforts some studies may have been missed especially if they were not catalogued on health and social literature databases.13 Equally, those sexual risk factors for PD presented here are not based on genetic studies but on, for instance, possible consequences of having more and concurrent partners. The strength of such risk factors will inevitably depend on patterns of contraception use and terminations of pregnancy. Regardless however of the level of PD within any population, exposing such people will inevitably affect not only their health but that of their family and potentially that of the biological father. With increasing levels of organ donation, male infertility treatment, screening for diseases, and DNA profiling featuring in police and emergency investigations, opportunities to identify PD are also increasing. Decisions on what should be done with such information are currently poorly researched. Consequently,

most inadvertently identified PD is ignored along with the associated consequences to people of not knowing the correct parentage and the possibility that PD may be discovered later. However, in a society where services and life decisions are increasingly influenced by genetics, our approach to PD cannot be simply to ignore this difficult issue but must be informed by what best protects the health of those affected.

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#### REFERENCES

- 1 Lucassen A, Parker M. Revealing false paternity: some ethical considerations. Lancet 2001;357:1033-5.
- 2 Brown R. Does Res Judicata bar tort claims for misrepresenting paternity? Am J Fam Law 2003;17:179
- Hughes SM, Harrison MA, Gallup GG. Sex differences in mating strategies: mate guarding, infidelity and multiple concurrent sex partners. Sexualities, Evolution and Gender 2004;6:3–13.
- 4 Spriggs M. IVF Mix up: white couple have black babies. J Med Ethics 2003:29:65.
- 5 Chiche J, Cariou A, Mira J. Bench-to-bedside review: fulfilling promises of the Human Genome Project. Crit Care 2002;6:212–15. 6 Department of Health. Code of practice and guidance on genetic paternity
- testing services. London: Department of Health, 2001.
- Australian Law Reform Commission. Essentially yours: the protection of human genetic information in Australia. Sydney: Australia Law Reform Commission, 2003,
- 8 Human Genetics Commission. Inside information: balancing interests in the use of personal genetic data. London: Human Genetics Commission, 2002. 9 Parliamentary Office of Science and Technology. NHS genetic testing,
- Postnote 227. London: Parliamentary Office of Science and Technology, 2004
- Wilson JF, Weale ME, Smith AC, et al. Population genetic structure of variable drug response. Nat Genet 2001;29:265–9.
   Jobling MA, Gill P. Encoded evidence: DNA in forensic analysis. Nat Rev
- Genet 2004;5:739-5
- **Ross LF**. Disclosing misattributed paternity. *Bioethics* 1996;**10**:114–30. In: Higgins JPT, Green S, eds. Cochrane handbook for systematic reviews of 13 interventions 4.2.4 (updated March 2005). Cochrane Library, Issue 2. Chichester: Wiley, 2005. 14 **Cerda-Flores RM**, Barton SA, Marty-Gonzalez LF, *et al.* Estimation of
- nonpaternity in the Mexican population of Nuevo Leon: a validation study with blood group markers. *Am J Phys Anthropol* 1999;**109**:281–93.
- 15 Brock DJH, Shrimpton AE. Non-paternity and prenatal genetic screening. Lancet 1991;338:1151.
- 16 Dowdell EB. Grandmother caregivers and caregiver burden. MCN Am J Matern Child Nurs 2004;29:299–304.
- Brenner CH, Weir BS. Issues and strategies in the DNA identification of World 17 Trade Center victims. Theor Popul Biol 2003;63:173-8. Hsu CM, Huang NE, Tsai LC, et al. Identification of victims of the 1998
- 18 Taoyuan airbus crash accident using DNA analysis. Int J Legal Med 1999:113:43-6.
- Sykes B, Irven C. Surnames and the Y chromosome. Am J Hum Genet 2000;**66**:1417–19
- 20 Helgason A, Hrafnkelsson B, Gulcher JR, et al. A population wide coalescent analysis of Icelandic matrilineal and patrilineal genealogies: evidence for a faster evolutionary rate of mtDNA lineages than Y chromosomes. *Am J Hum Genet* 2003;**72**:1370–88.
- 21 Macintyre S, Sooman A. Non-paternity and prenatal genetic screening. Lancet 1991;338:869-71.
- Johnson AM, Mercer CH, Erens B, et al. Sexual behaviour in Britain: 22
- partnerships, practices, and HIV risk behaviours. Lancet 2001;358:1835-42. 23 Finer LB, Darroch JE, Singh S. Sexual partnership patterns as a behavioral risk factor for sexually transmitted diseases. Fam Plann Perspect 1999;31:228-36
- 24 Chagnon N. (Cited in Smith RL). Sperm competition and the evolution of
- animal mating systems. London: Academic Press, 1984. 25 **Cherkas LF**, Oelsner EC, Mak YT, *et al*. Genetic influences on female infidelity. and number of sexual partners in humans: a linkage and association study of

the role of the vasopressin receptor gene (AVPR1A). Twin Res

2004;7:649-58

- 26 Forste R, Tanfer K. Sexual exclusivity among dating, cohabiting, and married women. J Marriage Fam 1996;58:33-47
- 27 Traeen B, Stigum H. Parallel sexual relationships in the Norwegian context. J Community Appl Soc Psychol 1998;8:41–56.
   Schofield MJ, Minichiello V, Mishra GD, et al. Sexually transmitted infections
- and use of sexual health services among young Australian women: women's health Australia study. *Int J STD AIDS* 2000;**11**:313–23.
- Low N, Sterne JAC, Barlow D. Inequalities in rates of gonorrhoea and 29 chlamydia between black ethnic groups in south east London: cross sectional study. Sex Transm Infect 2001;77:15–20.
- Centers for Disease Control and Prevention. STD Surveillance 2003: special focus profiles: racial and ethnic minorities. Atlanta, GA: Centers for Disease 30
- Control and Prevention, 2003. Martin JA, Hamilton BE, Sutton PD, et al. Births: final data for 2002, National 31 Vital Statistics Reports, 52(10). Atlanta, GA: Centers for Disease Control and Prevention, 2003
- 32 Du Toit ED, May RM, Halliday IL, et al. Paternity exclusion using 18 genetic systems in 2124 cases in four South African population groups. S Afr Med J 1989:75:103-5.
- Bellis MA, Hughes K, Ashton JR. The promiscuous 10%? J Epidemiol Community Health 2004;58:889–90. 33
- Steen CD. Cystic fibrosis: inheritance, genetics and treatment. Br J Nurs 34 1997;6:192-9.
- Winkelmann BR, Hager J, Kraus WE, et al. Genetics of coronary heart disease: current knowledge and research principles. Am Heart J 2000;140:S11-26.
- 36 Frank SA. Genetic predisposition to cancer insights from population genetics. Nat Rev Genet 2004;5:764–72.
- 37 Cummings DE, Schwartz MW. Genetics and pathophysiology of human obesity. Annu Rev Med 2003;54:453-71.
- Department of Health. Our inheritance, our future-realising the potential of 38 genetics in the NHS. Cm5791. London: Department of Health, 2003. Evans JP, Skrzynia C, Burke W. The complexities of predictive genetic testing
- 39 BMJ 2001;**322**:1052-6
- 40 Marteau TM, Lerman C. Genetic risk and behavioural change. BMJ 2001;322:1056-9.
- Mansfield C, Hopfer S, Marteau TM. Termination rates after prenatal 41 diagnosis of Down syndrome, spina bifida, anencephaly, and Turner and Klinefelter syndromes: a systematic literature review. European Concerted Action: DADA (decision-making after the diagnosis of a fetal abnormality). Action: DADA (decision making differing dognosis of a lotar denomically), Prenat Diagn 1999;19:808–12.
  42 Burke W. Genetic testing. N Engl J Med 2002;347:1867–75.
  43 Ashton JR. Pater semper. Lancet 1973;ii:451.
  44 Soderdahl DW, Rabah D, McCune T, et al. Misattributed paternity in a living

- Ritter MM. Genetic testing and paternity. Lancet 2001;358:241.
   Forensic Science Service. The National DNA database (NDNAD): fact sheet.
- 46 Birmingham: Forensic Science Service, 2004. (accessed 4 Feb 2005).
- 47 Forensic Science Service. Familial Searching: fact sheet. Birmingham: Forensic Science Service, 2003. http://www.forensic.gov.uk/forensic\_t/inside/news/ docs/Familial\_Searching.doc (accessed 4 Feb 2005). American Association of Blood Banks. Annual report summary for testing in
- 48 2001. Bethesda MD: American Association of Blood Banks, 2002.
- 49 Callaghan G. Who's your daddy? The Weekend Australian Magazine 2004;6-7 Nov:28-9.
- Amato PR, Previti D. People's reasons for divorcing: gender, social class, the life course and adjustment. J Fam Issues 2003;24:602–26. 50
- Office for National Statistics. Marriage, divorce and adoption statistics: 51 review of the Registrar General on marriage, divorce and adoptions in England and Wales, 2001. Series FM2 no 29. London: Office for National atistics, 2003.
- 52 Wade TJ, Pevalin DJ. Marital transitions and mental health. J Health Soc Behav 2004;45:155-70
- Amato PR. Children of divorce in the 1990s: an update of the Amato and 53 Keith (1991) meta-analysis. *J Fam Psychol* 2001;**15**:355–70. **Braver SL**, Ellman IM, Fabricius WV. Relocation of children after divorce and
- 54 children's best interests: new evidence and legal considerations. J Fam Psychol 2003:17:206-19
- Daly M, Wilson M. The truth about Cinderella: a Darwinian view on parental 55 love. London: Weidenfeld and Nicolson, 1998.
- 56 Krug EG, Dahlberg LL, Mercy JA, et al. World report on violence and health.
- Bec. Jail term for fake DNA tests boss. BBC News 2004, 24 Sep 2004. http://news.bbc.co.uk/1/hi/england/dorset/3686864.stm (accessed 4 Feb 57 2005)
- Asch DA, Hershey JC, Dekay ML, et al. Carrier screening for cystic fibrosis: 58 costs and clinical outcomes. Med Decis Making 1998;18:202–12.

- 59 Raithatha N, Smith RD. Disclosure of genetic tests for health insurance: is it ethical not to? Lancet 2004;363:395-6
- 60 Johnson M. Donor anonymity and review: Keynote address. Proceedings of the Human Fertilisation and Embryology Authority annual conference 2004, 21 January 2004. London: Human Fertilisation and Embryology Authority, 2004.
- Allaby MAK. Risks of unintended pregnancy in England and Wales in 1989. 61 Br J Fam Plann 1995;21:93-4
- 62 Marsters RW. Determination of nonpaternity by blood groups. J Forensic Sci 1957:2:15-37
- Valentin J. Exclusions and attributions of paternity: practical experiences of forensic genetics and statistics. *Am J Hum Genet* 1980;**32**:420–31. 63
- Houtz TD, Wenk RE, Brooks MA, et al. Laboratory evidence of unsuspected 64 parental consanguinity among cases of disputed paternity. Forensic Sci Int 1982;**20**:207-15
- Mickey MR, Gjertson DW, Terasaki PI. Empirical validation of the Essen-65 Möller probability of paternity. Am J Hum Genet 1986;39:123-32. Helminen P, Ehnholm C, Lokki M, et al. Application of DNA "fingerprints" to
- paternity determinations. Lancet 1988;i:574-6.
- Jeffreys AJ, Turner M, Debenham P. The efficiency of multilocus DNA 67 fingerprint probes for individualization and establishment of family relationships, determined from extensive casework. Am J Hum Genet 1991;**48**:824–40.
- Helminen P, Sajantila A, Johnsson V, et al. Amplification of three 68 hypervariable DNA regions by polymerase chain reaction for paternity determinations: comparison with conventional methods and DNA
- fingerprinting. *Mol Cell Probes* 1992;**6**:21–6. **Krawczak M**, Böhm I, Nürnberg P. Paternity testing with oligonucleotide multilocus probe (CAC)<sub>5</sub>/(GTG)<sub>5</sub>: a multicentre study. *Forensic Sci Int* 1993;**59**:101–17.
- Strom CM, Rechitsky S, Ginsberg N, *et al.* Prenatal paternity testing with deoxyribonucleic acid techniques. *Am J Obstet Gynecol* 1996;**174**:1849–53. Molyaka YK, Ovchinnikov IV, Shlenskii AB, *et al.* DNA genotypescopy in 70
- paternity testing: use of hybridisation probes. Genetika 1997;33:831–5.
- 72 Boardman F. Letter included in: House of Commons Hansard Written Answers for 19 Feb 1998: Child Support Agency. London: House of Commons, 1998.
- Geada H, Brito RM, Ribeiro T, et al. Portuguese population and paternity investigation studies with a multiplex PCR—the AmpFISTR Profiler Plus. 73 Forensic Sci Int 2000;108:31-7
- Edwards JH. A critical examination of the reputed primary influence of ABO phenotype on fertility and sex ratio. Br J Prev Soc Med 1957;11:87-9.
- 75
- Sussman LN, Schatkin SB. Blood-grouping tests in undisputed paternity proceedings. JAMA 1957;164:249–50. Schacht LE, Gershowitz H. Frequency of extra-marital children as determined by blood groups. In: Gedda L, ed. Proceedings of the second international 76 congress on human genetics. In: Rome: G Mendel, 1963:894-7.
- 77 Peritz E, Rust PF. On the estimation of the nonpaternity rate using more than one blood-group system. Am J Hum Genet 1972;24:46–53 Philipp EE. Discussion: moral, social and ethical issues. In:
- 78 Wolstenholme GEW, Fitzsimons DW, eds. Law and ethics of AID and embryo transfer. Ciba Foundation symposium, Vol 17. London: Associated Scientific, 1973.63-6
- 79
- Neel JV, Weiss KM. The genetic structure of a tribal population, the Yanomama Indians. *Am J Phys Anthrop* 1975;**42**:25–52. Ashton GC. Mismatches in genetic markers in a large family study. *Am J Hum* Genet 1980;32:601-13.
- Salmon D, Seger J, Salmon C. Expected and observed proportion of subjects excluded from paternity by blood phenotypes of a child and its mother in a sample of 171 families. *Am J Hum Genet* 1980;**32**:432–44. Lathrop GM, Hooper AB, Huntsman JW, *et al.* Evaluating pedigree data. I.
- The estimation of pedigree error in the presence of marker mistyping. Am J Hum Genet 1983;**35**:241–62
- 83 Peñaloza R, Núñez C, Silvia A, et al. Frequency of illegitimacy in a sample of the Mexican population. La Rev Invest Clin (Méx) 1986;**38**:287–91.
- Le Roux M, Pascal O, Andre M, et al. Non-paternity and genetic counselling Lancet 1992;340:607.
- Poon M, Anand S, Fraser BM, et al. Hemophilia B carrier determination based 85 on family-specific mutation detection by DNA single-strand conformation analysis. J Lab Clin Med 1993;**122**:55–63.
- Sasse G, Müller H, Chakraborty R, *et al.* Estimating the frequency of nonpaternity in Switzerland. *Hum Hered* 1994;**44**:337–43. 86
- Chataway J, Sawser S, Feakes R, et al. A screen of candidates from peaks of 87 linkage: evidence for the involvement of myeloperoxidase in multiple sclerosis. J Neuroimmunol 1999;**98**:208–13.
- Bellis MA, Baker RR. Do females promote sperm competition? Data for humans. Anim Behav 1990;40:997–9. 88
- Gaulin SJC, McBurney DH, Brakeman-Wartell SL. Matrilateral biases in the 89 investment of aunts and uncles: a consequence and measure of paternity uncertainty. Hum Nat 1997;8:139-51.