

## REVIEW

# 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.

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.<sup>6,7</sup> Moreover, along with an increase in parentage testing<sup>8</sup> health services now use genetic techniques in diagnosis<sup>9</sup> and treatment,<sup>10</sup> 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.<sup>12</sup> 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, mis-attributed, 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 searching<sup>13</sup> 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

For 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;<sup>1</sup> 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 elsewhere<sup>3</sup>). 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.<sup>4</sup>

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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 PD<sup>15</sup> 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.<sup>16</sup> Historical blood type data or even modern data identifying relatives of natural disaster and terrorist attack fatalities<sup>17 18</sup> 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.<sup>25</sup> 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 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.<sup>31</sup> 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.<sup>32</sup> 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 (UK<sup>9</sup>). 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,<sup>40</sup> terminations of pregnancy,<sup>41</sup> 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 health<sup>43</sup> 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

**Table 1** Summary of studies providing measures of paternal discrepancy stratified into disputed paternity tests and those undertaken for other reasons

Country	Population*	Sample Size	PD estimate % (95% CI)†	Method‡	Bias§	Reference
<b>Disputed paternity testing</b>						
USA	After birth	200	29.0 (22.7 to 35.3)	Blood and other markers		Marsters, 1957 <sup>62</sup>
Sweden	After birth	3913	26.1 (24.7 to 27.5)	Blood and other markers		Valentin, 1980 <sup>63</sup>
USA	After birth	2500	25.5 (23.8 to 27.2)	Blood and other markers		Houtz <i>et al</i> , 1982 <sup>64</sup>
USA	After birth	1393	25.8 (23.5 to 28.1)	Blood and other markers		Mickey <i>et al</i> , 1986 <sup>65</sup>
Finland	After birth	26	34.6 (15.0 to 54.2)	DNA testing		Helminen <i>et al</i> , 1988 <sup>66</sup>
South Africa	After birth	2124	38.2 (36.1 to 40.3)	Blood and other markers		Du Toit <i>et al</i> , 1989 <sup>62</sup>
Mostly UK	After birth	1702	16.6 (14.9 to 18.4)	DNA testing		Jeffreys <i>et al</i> , 1991 <sup>67</sup>
Finland	After birth	35	15.2 (2.1 to 26.5)	DNA testing	suspected non-paternity (+)	Helminen <i>et al</i> , 1992 <sup>68</sup>
Germany	After birth	256	16.8 (12.2 to 21.4)	DNA testing		Krawczak <i>et al</i> , 1993 <sup>69</sup>
USA	Prenatal	37	53.0 (37.2 to 70.9)	DNA testing		Siron <i>et al</i> , 1996 <sup>70</sup>
USA	After birth	753	37.0 (33.6 to 40.5)	DNA testing		Siron <i>et al</i> , 1996 <sup>70</sup>
Russia	After birth	21	14.0 (0 to 30.6)	DNA testing		Malyaka <i>et al</i> , 1997 <sup>71</sup>
UK	After birth	16122	13.0 (12.5 to 13.5)	DNA testing		Boordman F, 1998 <sup>72</sup>
Portugal	After birth	83	27.7 (17.9 to 37.5)	DNA testing		Geada <i>et al</i> , 2000 <sup>73</sup>
Portugal	After birth	790	29.8 (26.6 to 32.9)	DNA testing		Geada <i>et al</i> , 2000 <sup>73</sup>
USA and European	After birth	310490	29.1 (28.9 to 29.3)	Mixed methods		American Association of Blood Banks, 2002 <sup>68</sup>
<b>Other testing</b>						
UK	Southern English families	2578	3.7 (3.0 to 4.4)	Blood and other markers	not known	Edwards, 1957 <sup>74</sup>
USA	Undisputed paternity tests	67	18.0 (8.5 to 27.3)	Blood and other markers	not known	Sussman and Schalkin, 1957 <sup>75</sup>
USA	Michigan white sample	1417	1.4 (0.8 to 2.0)	Blood and other markers	not known	Schacht and Gershowitz, 1963 <sup>76</sup>
USA	Michigan black sample	523	10.1 (7.5 to 12.7)	Blood and other markers	not known	Schacht and Gershowitz, 1963 <sup>76</sup>
USA	Californian white sample	6960	2.7 (2.3 to 3.1)	Blood and other markers	not known	Peritz and Rust, 1972 <sup>77</sup>
UK	Southern English families	200	30.0 (23.6 to 36.4)	Blood and other markers	poor test sensitivity (-)	Philipp, 1973 <sup>78</sup>
South America	Yanomama tribe	132	9.0 (4.1 to 14.1)	Blood and other markers	not known	Neel and Weiss, 1975 <sup>79</sup>
USA	Hawaiian families	2839	2.3 (1.7 to 2.8)	Blood and other markers	non-participation in sample (-)	Ashton, 1980 <sup>80</sup>
France	Screening and paternity tests	300	7.0 (4.1 to 9.9)	Blood and other markers	some suspected non-paternity (+)	Salmon <i>et al</i> , 1980 <sup>81</sup>
New Zealand	Tokelau families	1983	4.0 (3.1 to 4.9)	Blood and other markers	not known	Penhaloza, 1986 <sup>81</sup>
Mexico	Families with new births	217	2.9 (0.6 to 5.0)	Blood and other markers	not known	Penhaloza, 1986 <sup>81</sup>
UK	Cystic fibrosis screening	521	1.4 (0.4 to 2.3)	DNA testing	non-participation in sample (-)	Brock and Shrimpton, 1991 <sup>15</sup>
France	Genetic screening (various)	362	2.8 (1.1 to 4.5)	DNA testing	non-participation in sample (-)	Le Roux <i>et al</i> , 1992 <sup>84</sup>
Canada	Haemophilia B screening	25	4.0 (0 to 12.3)	DNA testing	non-participation in sample (-)	Poon <i>et al</i> , 1993 <sup>85</sup>
Switzerland	Cystic fibrosis/bone marrow screening	1607	0.8 (0.4 to 1.3)	Mixed methods	non-participation in sample (-)	Sasse <i>et al</i> , 1994 <sup>86</sup>
Mexico	Nuevo Leon new births	396	11.8 (8.7 to 15.1)	Blood and other markers	not known	Cerda-Flores <i>et al</i> , 1999 <sup>4</sup>
UK	Multiple sclerosis screening	744	1.6 (0.7 to 2.5)	DNA testing	non-participation in sample (-)	Chataway <i>et al</i> , 1999 <sup>87</sup>
<b>Behavioural estimates¶</b>						
UK	Magazine readers	2708	6.9 to 13.8	Behaviour based estimate	sample composition (+)	Bellis and Baker 1990 <sup>88</sup>
USA	College undergraduates	285	13.0 to 20.0	Behaviour based estimate	not known	Gaulin <i>et al</i> , 1997 <sup>89</sup>

\* All populations in "other testing" are after-birth. †CI, confidence intervals. ‡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%CI§ have not been calculated for behaviour based estimates as these have been published as ranges. † Blood and other markers methods usually rely on ABO and rhesus 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 remain undetected. With DNA testing, polymorphism in the restriction fragment length polymorphism (RFLP) test are usually sensitive enough to require little or no correction. § Bias is identified as overestimate PD (–) and underestimate PD (+). ¶ All disputed paternity testing (individuals recruited to study) is entered next to studies where direction of any bias is unclear. †† Behaviour based estimates rely on questionnaires rather than biomolecular markers to estimate PD.



### 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 (cheek 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 partners<sup>52</sup> while children can experience low self esteem, anxiety, and increased involvement in antisocial behaviour such as aggression.<sup>53</sup> 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.<sup>44</sup> 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.<sup>55</sup> Suspected infidelity is also a trigger for domestic violence against women.<sup>56</sup> 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.<sup>57</sup>

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,<sup>40</sup> the decision to procreate,<sup>58</sup> 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.<sup>15</sup> 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.<sup>60</sup>

## 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 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.<sup>13</sup> 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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