Review

The neurodevelopment of human sexual orientation

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Abstract

One of the most enduring and controversial questions in the neuroscience of sexual behaviour surrounds the mechanisms which produce sexual attraction to either males or females. Here, evidence is reviewed which supports the proposal that sexual orientation in humans may be laid down in neural circuitry during early foetal development. Behaviour genetic investigations provide strong evidence for a heritable component to male and female sexual orientation. Linkage studies are partly suggestive of X-linked loci although candidate gene studies have produced null findings. Further evidence demonstrates a role for prenatal sex hormones which may influence the development of a putative network of sexual-orientation-related neural substrates. However, hormonal effects are often inconsistent and investigations rely heavily on 'proxy markers'. A consistent fraternal birth order effect in male sexual orientation also provides support for a model of maternal immunization processes affecting prenatal sexual differentiation. The notion that non-heterosexual preferences may reflect generalized neurodevelopmental perturbations is not supported by available data. These current theories have left little room for learning models of sexual orientation. Future investigations, across the neurosciences, should focus to elucidate the fundamental neural architecture underlying the target-specific direction of human sexual orientation, and their antecedents in developmental neurobiology.
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Contents

1. Introduction

Sexual orientation refers to a dispositional sexual attraction towards persons of the opposite sex or same

sex. Sexual orientation appears 'dispositional' in that it comprises a target selection and preference mechanism sensitive to gender, motivational approach behaviours towards the preferred target, and internal cognitive processes biased towards the preferred target (such as sexual fantasies). In contrast, sexual orientation does not appear to be a matter of conscious self-labelling or past sexual activity because these are subject to contingent social pressures, such as the presence of linguistic descriptors and visible sexual minorities within an individual's culture,

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and the availability of preferred sexual partners (Bailey, 2003). Therefore, in human investigations, sexual orientation is often assessed using self-report measures of 'sexual feelings' (i.e. sexual attraction and sexual fantasies) rather than self-labelling or past hetero- or homosexual activity.

Sexual orientation appears to be a dichotomous trait in males, with very few individuals demonstrating an intermediate (i.e. 'bisexual') preference. This is borne out by fine-grained analyses of self-reported heterosexual and homosexual orientation prevalence rates (using measures of sexual feelings) in population-level samples, and work on physiological genital arousal patterns (e.g. using penile plethysmography) in response to viewing preferred and non-preferred sexual imagery. Both lines of evidence consistently demonstrate a bimodal sexual orientation among men—heterosexual or homosexual, but rarely 'bisexual' (Chivers et al., 2004; Dickson et al., 2003; Erens et al., 2003; Sakheim et al., 1985; Wellings et al., 1994). This is less so in the case among women. For example, Chivers et al. (2004) demonstrated a 'bisexual' genital arousal pattern among both heterosexual and lesbian women, suggesting a decoupling of self-reported sexual feelings (which appears broadly bimodal) from peripheral sexual arousal in women.

If sexual orientation among humans is a mostly bimodal trait, this implicates a canalization of development along a sex-typical route (heterosexual) or a sex-atypical (homosexual) route. Statistical taxometric procedures have confirmed this by demonstrating that latent taxa (i.e. non-arbitrary natural classes) underlie an opposite-sex, or same-sex, orientation in both men and women (Gangestad et al., 2000). Less well established are the factors that may be responsible for this 'shunting' of sexual orientation along two routes (the edges of which are fuzzier in women). These factors are the subject of the remaining discussion and it is suggested that they probably operate neurodevelopmentally before birth.

2. Behavioural and molecular genetics

A natural starting point for the neurodevelopment of physiological and behavioural traits must begin with the genetic level of investigation. Several family and twin studies provide clear evidence for a genetic component to both male and female sexual orientation. Family studies, using a range of ascertainment strategies, show increased rate of homosexuality among relatives of homosexual probands (Bailey and Pillard, 1995). There is also evidence for elevated maternal line transmission of male homosexuality, suggestive of X linkage (Camperio-Ciani et al., 2004; Hamer et al., 1993), but other studies have not found such elevation relative to paternal transmission (Bailey et al., 1999). Among females, transmission is complex, comprising autosomal and sex-linked routes (Pattatucci and Hamer, 1995). Twin studies in both community and population-level samples report moderate heritability

estimates, the remaining variance being mopped up by non-shared environmental factors (Bailey et al., 2000; Kendler et al., 2000; Kirk et al., 2000). Early attempts to map specific genetic loci responsible for sexual orientation using family pedigree linkage methods led to the discovery of markers on the Xq28 chromosomal region (Hamer et al., 1993), with one subsequent replication limiting the effect to males only (Hu et al., 1995). However, there is at least one independent study which produced null findings (Rice et al., 1999), while a recent genome wide scan revealed no Xq28 linkage in a new sample of families but identified putative additional chromosomal sites (on 7q36, 8p12 and 10q26) which now require denser mapping investigations (Mustanski et al., 2005). These studies are limited by factors such as the unclear maternal versus paternal line transmission effects, possible autosomal transmission and measurement issues. Two candidate gene studies which explored the putative hormonal pathways in the neurodevelopment of sexual orientation (see Section 3): one on the androgen receptor gene and another on aromatase (CYP19A1) both produced null findings (DuPree et al., 2004; Macke et al., 1993).

3. The prenatal androgen model

Several decades of research in animal models have demonstrated a major role for gonadal steroidal androgens in accounting for almost all known sexual dimorphism in brain and behaviour among vertebrates (Morris et al., 2004). These have guided investigators to search for the possible origins of human sexual orientation in androgens and their target neural substrates. Within this framework, it has become a cliché to suggest that heterosexual preference in men are due to typical degrees of prenatal exposure to androgens (primarily testosterone), and that heterosexual preference in women is due to default mammalian development along female lines (due to very little prenatal androgen exposure). Conversely (the cliché continues), homosexuality in men is due to under-exposure to prenatal androgens and in women, due to over-exposure (Ellis and Ames, 1987). This classic model of the origins of sexual orientation had some early support from experimental manipulation of prenatal sex hormone levels in animal models, and the prevalence of variant sexual orientation (in line with purported prenatal sex-typical or sexual-atypical hormonal exposure) among human inter-sex cases or in those with endocrine disorders, such as congenital adrenal hyperplasia (Bailey, 2003; Morris et al., 2004).

More recently, research in this area has moved to focusing on 'proxy markers' for prenatal hormonal exposure that can be easily, and non-invasively, explored in otherwise endocrinologically normal populations. These 'proxy markers' comprise somatic features which are known to be influenced by sex hormones prenatally. Thus showing variation in these traits between adult heterosexuals

and homosexuals may provide a 'window' into the early neurodevelopment of sexual preferences under the actions of prenatal hormones. These markers are certainly imperfect tools, but in the absence of prospective research, they have provided some intriguing insights. The best known proxy marker of prenatal hormonal exposure is the ratio of the second to fourth finger lengths (or 2D:4D ratio) (Manning et al., 1998). 2D:4D is sexually dimorphic, with males showing lower ratios than females. Evidence for lower ratios in individuals with androgen over-exposure (such as in the condition congenital adrenal hyperplasia) strongly implicates prenatal androgens in modulating 2D:4D (Brown et al., 2002a; Ökten et al., 2002; Buck et al., 2003). 2D:4D is also linked to variation in the androgen receptor gene (Manning et al., 2003a) and the ratio of testosterone to estrogen taken from amniotic fluid during gestation is negatively associated with 2D:4D at 2 years of age (Lutchmaya et al., 2004). Although ultimately correlational, these data suggest strongly that excess androgen exposure can alter the relative lengths of the second and fourth finger digits.

Four independent studies have shown that homosexual women have significantly masculine (lower) 2D:4D ratios compared to heterosexual women, although these appear to be hand-specific (Rahman and Wilson, 2003a; Williams et al., 2000; Rahman, 2005; McFadden and Schubel, 2002) but one study reported no such difference (Lippa, 2003). In homosexual men, three reports show more *male-like* (i.e. 'hyper-masculinized') 2D:4D ratios compared to heterosexual men (Rahman and Wilson, 2003a; Rahman, 2005; Robinson and Manning, 2000), while another two demonstrated more *female-like* 2D:4D ratios in homosexual men (Lippa, 2003; McFadden and Schubel, 2002). A further report showed that only homosexual men with two or more elder brothers had hyper-masculinized right-hand 2D:4D ratios (Williams et al., 2000). Evidence for possible 'within-sexual orientation' variations was reported by one study showing lower 2D:4D in self-identified 'butch' compared to 'femme' homosexual women (Brown et al., 2002b), while another did not find this in both homosexual men and women (Rahman and Wilson, 2003a). Overall, these data strongly suggest that lesbians are exposed to a greater degree of masculinization by prenatal androgens than heterosexual women. However, the reports for men are confusing—some showing 'hyper-male' 2D:4D and others female-like patterns. A possible solution for the male findings may be found in the demonstration of a 'uniform mean' 2D:4D ratio among homosexual men of between 0.96 and 0.97, contrasted with substantial variation among heterosexual populations (Manning and Robinson, 2003). This 'uniform mean' may also be population-specific as the available data indicate that only Caucasian ethnic groups manifest it (McFadden et al., in press; Voracek et al., in press). The narrow range (a masculinized value) may indicate the prenatal androgen level that maximizes the chances of a homosexual orientation. Alternatively, the overall evidence might simply suggest that both lower-than-average and higher-than-average androgen exposure increases the probability of developing male homosexuality.

Nevertheless, the 'uniform mean hypothesis' is still controversial, partly because in some respects it is simply a restatement of the findings observed thus far. It is possible that there is no real difference between heterosexual and homosexual men in 2D:4D and that the observed differences merely reflect sampling error. Secondly, there is, as yet, no known biological mechanism whereby a constant value in this particular trait should occur in the minority population (i.e. homosexual men) while the majority population shows greater variation.

One additional 'hand-related' trait that differentiates early in gestation is fingerprint patterns, or dermatoglyphics. Although an early study reported that homosexual men possess a female-typical dermatoglyphic pattern (an asymmetry with more ridges on left-hand fingers) than heterosexual men, subsequent independent reports have not demonstrated any sexual-orientation-related variations (Forastieri et al., 2003; Mustanski et al., 2002; Slabbekoorn et al., 2000). Thus dermatoglyphic patterns are almost invariably a poorer window on early prenatal differentiation of sexual orientation compared to finger length ratios.

Studies of auditory mechanisms also show specific hyper-masculinization among homosexuals compared to heterosexuals. Oto-acoustic emissions (OAEs) are tiny sounds emitted by the cochlea and can occur spontaneously or be evoked by 'clicking' sounds. OAEs of both varieties are more numerous in females than in males, and in the right ear—this patterning apparent in infants, children and adults. Evidence that OAEs are influenced by prenatal androgens come from the finding that females with male co-twins have masculinized OAE patterns (McFadden, 1993). Two reports have shown less numerous and weaker OAEs in homosexual and bisexual women compared to heterosexual women, but no variation between homosexual and heterosexual men (McFadden and Pasanen, 1998, 1999). While there is no difference between male groups with respect to auditory mechanisms on the periphery, there is centrally. This was discovered by examining the auditory evoked potentials (AEPs) produced in response to click-stimuli. On 5 of 19 AEP outcome measures, homosexual women showed masculinized responses and homosexual men demonstrated hyper-masculinized responses (McFadden and Champlin, 2000).

Further work under the prenatal androgen framework has reported sexual-orientation-related differences in physical growth markers. Homosexual men consistently report earlier pubertal onset on physical and behavioural indices (e.g. age of first ejaculation or age of first sexual experience) compared to heterosexual men (Bogaert and Blanchard, 1996; Bogaert and Friesen, 2002; Bogaert et al., 2002), whereas homosexual and heterosexual women do not differ in pubertal milestones (Bogaert and Friesen, 2002; Bogaert et al., 2002; Tenhula and Bailey, 1998). There have also

been inconsistent reports for sexual-orientation-related variations in self-reported height and weight (Bogaert and Friesen, 2002; Bogaert et al., 2002) but it is far from clear whether these reflect solely the actions of the prenatal sex steroids or multiple postnatal factors. However, one recent study, which objectively measured skeletal growth in a large community sample, reported that homosexual men have less long-bone growth in the arms, legs and hands compared to heterosexual men, while the converse was found for homosexual compared to heterosexual women (Martin and Nguyen, 2004). As these bones become sexually dimorphic in childhood but not after puberty, these data suggest that homosexual men are partially feminized and homosexual women are masculinized, in specific anthropometric measures before the pubertal increase in sex steroid levels (Martin and Nguyen, 2004).

Overall, all the available evidence points to homosexual women being, on average, exposed to more prenatal androgens than heterosexual women, as predicted. However, it is important to note that there is considerable overlap between the two female groups, indicating that prenatal androgens do not act in isolation. The findings with respect to homosexual men are even more surprising with indications of both elevated and reduced prenatal androgen exposure. This appears inconsistent with the central prediction of prenatal androgen theory that homosexual males should show evidence of lower prenatal androgen levels. However, some resolution might be found if it is supposed that the requisite neural circuitry responsible for same-sex orientation in men is un-masculinized (e.g. perhaps because of genetic factors), which leads to excessive androgenic activity in the development of other somatic features. This might explain the observed (albeit unreliably) hyper-masculinized features among homosexual men. While this suggestion is certainly speculative, it is in accord with known observations for non-monotonic effects of sex steroids in some animal models (Clark et al., 1996).

The point to bear in mind is that male homosexuality may appear as a mosaic of traits (some sex-*typical*, others sex-*atypical* and yet others that are sex-*exaggerated*). This might be produced by differences in the timing and/or concentration of androgen exposure (e.g. lower-than-average and higher-than-average levels) in heterosexual and homosexual males. For example, Geschwind and Galaburda (1985) have suggested that homosexual men are exposed to particularly high androgen levels *very early* in development, explaining both their tendency to be less right-handed (see Section 5) and, by extension, the hyper-masculinized traits observed in this group. Interestingly, these possible temporal and localized variations in androgen exposure might suggest that their actions occur further 'up stream' in the developmental pathway, perhaps explaining the null findings of candidate gene studies above (Section 2) regarding the androgen receptor and aromatase gene.

4. The fraternal birth order effect and maternal immunity

The maternal immunity hypothesis is certainly the most revolutionary neurodevelopmental model of human sexual orientation. Empirically, it rests on one very reliable finding—the fraternal birth order effect (FBO): that is, homosexual men have a greater number of older brothers than heterosexual men do (and relative to any other category of sibling), in diverse community and population-level samples, and as early as they can be reliably surveyed (Blanchard, 2004). The estimated odds of being homosexual increase by around 33% with each older brother, and statistical modelling using epidemiological procedures suggest that approximately 1 in 7 homosexual men may owe their sexual orientation to the FBO effect (Cantor et al., 2002). It has been suggested that the remaining proportions of homosexual men may owe their sexual orientation to other causes, such as differential prenatal androgen levels (Blanchard, 2004; Cantor et al., 2002). Homosexual and heterosexual women do not differ in sibling sex composition or their birth order, thus any neurodevelopmental explanation for the FBO effect is limited to males (Bogaert, 1997). Importantly, recent work has demonstrated that homosexual males with older brothers have significantly lower birth weights compared to heterosexual males with older brothers (Blanchard and Ellis, 2001; Blanchard et al., 2002). As birth weight is undeniably prenatally determined, some common developmental factor operating before birth must underlie FBO and sexual orientation among human males.

Specifically, investigators have proposed a role for the progressive immunization of some mothers to male-linked antigens produced by carrying each succeeding male foetus. That is, the maternal immune system 'sees' male-specific antigens as 'non-self' and begins producing antibodies against them (Blanchard, 2004). One possible group of antigens are the Y-linked minor histocompatibility antigens, specifically *H–Y*. The accumulating H–Y antibodies may divert male-typical sexual differentiation of the foetal brain, leading the individual to be sexually attracted to males (Blanchard and Bogaert, 1996). For example, male-specific antibodies may bind to, and inactivate, male-differentiating receptors located on the surface of foetal neurons thus preventing the morphogenesis of masculinized sexual preferences.

The maternal immunity theory is consistent with a number of observations: the number of older sisters is irrelevant to sexual orientation in later born males; the H–Y antigen is expressed by male foetuses only and thus the maternal immune system 'remembers' the number of males carried previously and may modulate its response; and H–Y antigens are strongly represented in neural tissue (Blanchard, 2004; Blanchard and Bogaert, 1996). Nonetheless, there is no data specifying a role for these particular antigens in sexual preferences among humans. There are

several alternative candidate antigens to H–Y, including the distinct Y-linked protein families' *protocadherin* and *neuroligin*, both which have been found in humans. These cell adhesion proteins are thought to influence cell–cell communication during early male-specific brain morphogenesis and may have male-typical behavioural consequences (Blanco et al., 2000). Consistent with these studies is neurogenetic evidence for the direct transcription of Y-linked sex determination genes *SRY* and *ZFY* in the male human brain (including hypothalamus) (Mayer et al., 1998). The maternal immunity model may also explain the link between birth weight and sexual preferences: mouse models show that maternal immunization to male-derived antigens can affect foetal weight (Gentile et al., 1992; Lu and Dawson, 1986). Furthermore, male mice whose mothers are immunized to H–Y prior to pregnancy show reduced male-typical consummatory sexual behaviour towards receptive females (Singh and Verma, 1987).

The maternal immunity model implicitly relies on a non-hormonal immunologic neurodevelopmental explanation and thus cannot immediately explain the hyper-male features (e.g. 2D:4D and AEPs) associated with male homosexuality. It is possible that male-specific antibodies may interact with sexual differentiation processes controlled by sex hormones or be completely independent of them—this is unknown as yet.

5. Developmental instability and sexual orientation

There has been some argument recently that the prenatal androgen theory does not adequately explain the robust association between sexual orientation and handedness. Homosexual men have an approximate 34% odds ratio of being non-right-handed and homosexual women have approximately 91% odds of being so, compared to heterosexuals (of whom men are more non-right-handed than women) (Lalumiere et al., 2000). As the classic version of prenatal androgen theory predicts that homosexual men should show *less* non-right-handedness and homosexual women *more*, the observation holds true for women but not men (Lalumiere et al., 2000). Thus, perhaps a more domain-general developmental explanation for variation in human sexual orientation is needed. This may be found in *developmental instability* (DI) which refers to an organism's level of vulnerability to environmental and genetic stresses during development. In this view, same-sex orientation is due to generalized developmental insults that shift erotic preferences away from the species-typical pattern of opposite-sex attraction (Lalumiere et al., 2000). Again, proxy somatic measures of these 'insults' or 'instability' are proposed to provide a window on the developmental history of the organism—handedness being one such proxy. At first inspection the handedness data does appear to provide support but its explanation is, in fact, more parsimonious within prenatal androgen theory.

The apparent hyper-masculinized handedness of homosexual men can be squared with the explanation for hyper-masculinization in other somatic features such as finger length ratios, as detailed earlier. In support, two studies have reported a robust association between low 2D:4D and left-hand preference (Fink et al., 2004; Manning et al., 2000).

The most commonly utilized proxy measure of DI involves measuring random deviations from perfect symmetry in bilateral bodily features (e.g. dermatoglyphics, and lengths of ears, fingers, wrists and feet) and is referred to as fluctuating asymmetry (FA). FA is thought to reflect differential genomic robustness. Individuals with genomes which are less sensitive to stress-induced disruption may show suppression in phenotypic variation and thus be reproductively 'fitter' (i.e. produce the 'ideal' phenotype, such as heterosexuality). Therefore, a central prediction from DI theory is that heterosexuals of both sexes should show low FA values compared to homosexuals. Several reports have found no significant differences in FA between heterosexuals and homosexual (Rahman and Wilson, 2003a; Rahman, 2005; Mustanski et al., 2002) suggesting that a homosexual orientation does not necessarily reflect a 'less than optimal' phenotypic sexual orientation. Therefore, perhaps the canalization of the sexual-orientation trait is more likely due to specific, rather than general-purpose, neurodevelopmental mechanisms (such as the actions of prenatal androgens).

6. Neural circuitry

Neurodevelopmental mechanisms must wire neural circuits differently in those with same-sex attractions from those with opposite-sex attractions, but we still know very little about this circuitry. The first indication for neural correlates of sexual partner preference came from Simon LeVay (1991) autopsy study of the third interstitial nucleus of the anterior hypothalamus (INAH-3) which he found to be smaller in homosexual men than in presumed heterosexual men, and indistinguishable from presumed heterosexual women. Another study found a non-significant trend for a female-typical INAH-3 among homosexual men (and confirmed the heterosexual sex difference), but this was not evidenced the main sexually dimorphic parameter reported by this study (the total number of neurons) (Byne et al., 2001). This preceding finding is noteworthy as a prediction from the prenatal androgen theory would be that a parameter which shows significant sexual dimorphism should also demonstrate within-sex variation attributable to sexual orientation. A conservative conclusion regarding these data is that while INAH-3 is larger in heterosexual men than in heterosexual women, and possibly smaller in homosexual men, structurally speaking this within-sex difference may not be very large at all.

One recent positron emission tomography study has demonstrated stronger hypothalamic response to serotonergic challenge in heterosexual than in homosexual men (Kinnunen et al., 2004), and neuroimaging studies comparing heterosexual men and women while viewing preferred sexual imagery show significantly greater hypothalamic activation in heterosexual men (Karama et al., 2002). These findings, coupled with the anatomical findings described earlier, could be taken to suggest that there is a functionally distinct anterior hypothalamic substrate to sexual attraction towards women. This supposition is further supported by mammalian lesion models of the preoptic area (POA) of the anterior hypothalamus showing reduced appetitive responses towards female by male animals (Hull et al., 2002). Nevertheless, investigations comparing heterosexual and homosexual women are needed to support a role for this region in sexual preference towards females among humans.

While animal models point to a role for prenatal androgens in producing sexual variation in hypothalamic regions (Morris et al., 2004), a similar relationship in humans is unclear. One study found no sexual-orientation-related differences in the distribution of androgen receptors in sexually dimorphic hypothalamic regions (Kruijver et al., 2001). However, one animal model often overlooked by scientists may provide some guidance. Some males of certain species of sheep show an exclusive same-sex preference, and also show reduced aromatase activity and smaller ovine sexually dimorphic nuclei (a possible homolog to the human INAH-3) compared to female-oriented sheep (Roselli et al., 2004). A role for aromatized metabolites of testosterone in underscoring possible hypothalamic variation related to human sexual orientation requires further study in light of these findings (Roselli et al., 2004). Moreover, putative sexual orientation differences in aromatase activity in human males may go some way to explaining the 'mosaic' profile of hypo- and hyper-masculinized traits described earlier. For example, a reduction in aromatase activity in homosexual compared to heterosexual men (predicted from the Roselli findings) may lead to reduced availability of aromatized testosterone (i.e. estradiol) which typically masculinizes the male mammalian brain (Morris et al., 2004). This may lead to hypo-masculinized hypothalamic circuitry and yet leave excess non-aromatized testosterone to hyper-masculinize additional androgen sensitive traits (e.g. 2D:4D) through other metabolic pathways, such as 5-alpha reductase. Note, one mitigating piece of evidence with respect to these suggestions is the null finding of DuPree et al. (2004) regarding sexual-orientation-related variation in the aromatase gene.

The possibility that sexual-orientation-related neural variation extend to higher cortical regions has been evidenced by neurocognitive investigations. Several independent studies consistently demonstrate low scores (female-typical) by homosexual men in basic spatial ability tests (such as mental rotation and spatial perception) compared to heterosexual men (Rahman and Wilson,

2003b). Homosexual men also show better spatial location memory, improved recall of spatial landmarks during navigation, and better phonological and semantic fluency (all female-typical responses) compared to heterosexual men (Rahman et al., 2003a,b, 2005). These data tentatively suggest sexual variation in parietal, hippocampal-temporal, and prefrontal brain regions known to underlie these cognitive skills. Behavioural and structural sexual variation in inter-hemispheric pathways may contribute to these cognitive differences, but are not well replicated (Allen and Gorski, 1992; Wegesin, 1998a). Independent investigations utilizing several neurophysiological measures also support parietal and temporal lobe involvement, depending on the probe used: sexual, auditory or cognitive (Howard et al., 1994; Reite et al., 1995; Wegesin, 1998b). Parietal lobe involvement is likely as this region is part of the neural architecture of heterosexual sexual arousal, and possibly involved in visual-configural processing of preferred sexual 'targets' (Howard et al., 1994; Waisman et al., 2003).

Thus far, almost nothing is known about the neural basis of sexual orientation in women. One sexually dimorphic neurobehavioural probe—pre-pulse inhibition of the startle response (whereby there is a reduction in the eye-blink reflex to a loud noise if preceded by a by a quieter noise)—is strongly masculinized in homosexual compared to heterosexual women, and indicates the involvement of pallido-striato-thalamic limbic circuitry (Rahman et al., 2003c). Cognitive studies demonstrate better verbal fluency among homosexual women, pointing to prefrontal cortical involvement, while neurophysiological studies reveal no differences (Rahman et al., 2003a; Wegesin, 1998b), other than those in the auditory regions revealed by AEPs (McFadden and Champlin, 2000).

The available evidence gives us clues as to the neural network underlying a sexual orientation in men, including anterior hypothalamic regions, and cortical regions such as the parietal lobes. As far as sexual orientation in females is concerned, there is some indication for the involvement of limbic circuitry but little else. The functional neuroendocrinology herein is unknown but clues from the animal literature point to developmental processes under the control of prenatal sex steroids. Further investigation of such processes, such as potential androgenic modulation of apoptosis in the requisite neural circuitry, is needed (Morris et al., 2004; Chung et al., 2000).

7. Is there a role for learning in the development of human sexual orientation?

The role of learning in the development of human sexual orientation has been the subject of much debate and controversy, most likely because it is erroneously believed to result in particular socio-political consequences associated with homosexuality (Bailey, 2003). While data are a little thin on the ground, several lines of evidence mitigate

the involvement of learning mechanisms. In animal models, there are documented effects of conditioning on sexual arousal, approach behaviour, sexual performance and strength of sexual preference towards opposite-sex targets, but no robust demonstrations of learning in the organization of same-sex preferences among males (Pfaus et al., 2001; Woodson, 2002). Interestingly, one study in female rats demonstrated that the volume of the sexually dimorphic nucleus of the preoptic region was increased (male-typical) by testosterone administration coupled with same-sex sexual experience (Woodson et al., 2002). This suggests that sexual experience may interact with steroid exposure to shape sexual partner preferences in females.

In humans, the extent of childhood or adolescent homosexual versus heterosexual activity does not appear to relate to eventual adult sexual orientation. Documented evidence regarding the situational or cultural 'initiation' of juvenile males into extensive same-sex experience (for example, in single-sex public schools in Britain or the obligatory homosexual activity required of young males in the Sambia tribe of New Guinea) does not result in elevated homosexuality in adulthood (Bailey, 2003; Wellings et al., 1994).

An alternative explanation for the FBO effect is that sexual interaction with older brothers during critical windows of sexual development predisposes towards a homosexual orientation. Studies in national probability samples show that sibling sex-play does not underscore the link between FBO and male sexual orientation (Bogaert, 2000), and that the sexual attraction component of sexual orientation, but not sexual activity, are best predicted by frequency of older brothers (Bogaert, 2003). In further support, same-sex play between pairs of gay brothers is also unrelated to adult homosexual attraction (Dawood et al., 2000).

Perhaps parent–child interactions influence the sexual orientation of children? An informative test here is to examine the sexuality of children of homosexual parents because this type of familial dynamic could promote same-sex preferences through observational learning mechanisms. However, evidence from retrospective and prospective studies provides no support for this supposition (Bailey et al., 1995; Golombok and Tasker, 1996). Nonetheless, one must bear in mind that if parental behaviour does determine offspring sexual orientation, it could be equally common in homosexual and heterosexual parents.

While a role for learning factors can never be entirely omitted, it is perplexing that several of the key routes by which these could have their effect, such as through sexual experience during childhood or adolescence, or through parental socialization, are not supported. Almost certainly the expression of homosexual *behaviour* has varied over time and across cultures, but there is little reason to think that dispositional homosexuality varies greatly cross-culturally or even historically (Bailey, 2003).

8. Conclusion: The future of biobehavioural research on human sexual orientation

The literature thus far provides a rough outline of the neurodevelopmental mechanisms underlying human sexual orientation. As further work from several fields accumulates it is likely we will produce improved mechanistic explanations. Proxy markers will only ever be useful insofar as they truly index the underlying developmental mechanism. One informative test of the prenatal androgen model would be to examine amniotic sex steroid levels and sexual orientation (and its neurobehavioural correlates) prospectively. Nonetheless, it is also possible that sex steroid levels differ in the *brains* of pre-homosexual and pre-heterosexual foetuses but are not reflected in levels in their uterine environment (as indexed by amniocentesis). For the time being, work can clarify the utility of proxy markers, as well as focusing on other reliable 'windows' into early development e.g. 2D:5D and 3D:4D (McFadden and Schubel, 2002; Manning et al., 2003b).

Future investigations must clarify the relationship between neurodevelopmental markers and other neurobehavioural features associated with sexual orientation. Our group recently demonstrated no link between 2D:4D, the number of older brothers, and the neurocognitive variation between heterosexuals and homosexuals (Rahman et al., 2004). This study shows the potential for investigations to narrow the number of potential neurodevelopmental explanations for sexual orientation and its correlates. In this case one domain (i.e. cognitive) linked to sexuality is not necessarily attributable to a common prenatal mechanism (insofar as 2D:4D and number of older brothers reflect this). Progression of the maternal immunity model requires evidence for maternal immune responses in homosexual subjects with older brothers and those without (and their mothers). Studies using serological measures should reveal whether male-specific antigens, cell-surface proteins or even maternal cytokines are involved, while neuroimmunologic analyses of brain material can elucidate non-hormonal possibilities, such as differential sex-linked gene expression in the brains of heterosexuals and homosexuals (Mayer et al., 1998). There is no doubt that such investigations will also benefit from further linkage and candidate gene studies.

The primary challenge at this stage is to elucidate the precise neural circuitry underlying direction of sexual preference, requiring research across the neurosciences. This may require the definitional fractionation of sexual orientation into discrete behavioural components, as derived from animal models of the formation of sexual partner preferences, in order help frame research questions. Example components could include the detection and orientation toward potential 'targets' and the sensory modalities in which these operate (e.g. visual orienting or olfactory detection). Neuroimaging techniques will need to quantify putative sexual-orientation-related volumetric

differences in limbic substrates in vivo, while functional methods could be exploited to elucidate the subcortical–cortical networks responsible for sexual attraction to male and female targets. Neurochemical imaging studies could investigate potential roles of sex steroids upon these neural mechanisms. The psychological sciences can assist here also. For example, researchers could test whether the known attenuation of the human startle response (e.g. eye-blink patterns) to aversive stimuli is apparent for non-preferred sexual stimuli (compared to preferred sexual stimuli) in heterosexual and homosexual adults. Together, these investigations may clarify the inhibitory mechanisms underlying human sexual appetitive responses immensely. Ultimately, work in those with healthy sexual orientations may pave the way for work on abnormal or 'paraphilic' sexual preferences (Waisman et al., 2003).

It is commonly asked of researchers in this controversial field why the biobehavioural sciences should be concerned with a trait that is so skewed—that vast majority of individuals are attracted to the opposite sex after all. Herein lies the irony—elucidating the neurobiology of same-sex orientation will provide important insights into the far greater mystery regarding the proximate neurodevelopmental mechanisms which produce heterosexuality.

Acknowledgements

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