

Fluctuating asymmetry, second to fourth finger length ratios and human sexual orientation

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Summary Sexual orientation in humans may be influenced by levels of prenatal sex steroids which canalise neurodevelopment along sex-typical (heterosexual) or sexatypical (homosexual) lines. Some evidence for sexual-orientation-related differences in putative somatic markers of prenatal sex hormones supports this view. A competing theory asserts that human homosexuality is due to developmental instability (DI) because it represents a shift from the species-typical pattern of heterosexual orientation. Evidence for elevated rates of non-right handedness among homosexuals provides limited support for this account. The current study tested both theories by examining nine bilateral somatic traits in 120 healthy heterosexual and homosexual men and women in order to compute second to fourth finger length ratios (2D:4D), a measure ascribed to levels of prenatal sex steroids, and fluctuating asymmetry (FA), a measure of DI. Homosexual men and women had significantly lower right hand 2D:4D ratios (even after controlling for handedness, height and weight differences) in comparison to heterosexuals, but sexual orientation did not relate to composite FA scores. The findings constrain the number of possible neurodevelopmental pathways responsible for sexual orientation in humans.

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1. Introduction

Biological research into human sexual orientation has attracted a great deal of scientific research as well as its fair share of controversy (for a full review see Rahman and Wilson, 2003a). Although there is no single etiogenic account of sexual orientation in humans, the dominant theory has been the prenatal

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hormone theory (Ellis and Ames, 1987). This proposes that variation in human sexual orientation is under the control of prenatal sex steroids (probably interacting with genetic factors: see Hamer et al., 1993; Bailey et al., 2000) which canalise neurodevelopment in a sex-typical (heterosexual) or sex-atypical (homosexual) fashion. This theory predicts that homosexuals of both sexes should show cross-sex shifts in neurobehavioural domains in line with the atypical shift in their sexual partner preference (Ellis and Ames, 1987). An array of evidence from neuroanatomical and behavioural domains tends to support this notion. Among these

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include findings that homosexual men have larger neuronal populations in the suprachiasmatic nucleus (Swaab and Hofman, 1990), a trend for smaller interstitial nuclei of the anterior hypothalamus (LeVay, 1991, c.f. Byne et al., 2001), female-typical performance on sexuallydimorphic neurocognitive tests such as mental rotation, spatial perception and verbal fluency (McCormick and Witelson, 1991; Wegesin, 1998a; Rahman and Wilson, 2003b; Rahman et al., 2003a) and female-typical neurophysiological patterns (Reite et al., 1995; Wegesin, 1998b). However, homosexual men have also been found to show sextypical or even 'hyper-male' traits in some domains. For example, McFadden and Pasanen (1998, 1999) have found no differences between heterosexual and homosexual men in sexuallydimorphic otoacoustic emissions (or OAE's, which are weak sounds produced by the inner ear and are more numerous, and stronger, in females than in males), whilst McFadden and Champlin (2000) reported that homosexual men showed hypermasculinised auditory evoked potentials (AEPs) compared to heterosexual men. Homosexual men also report larger (more male-like) genital size in two studies (one measuring genital size using physician's examinations, the other using selfreport measures; Nedoma and Freund, 1961; Bogaert and Herschberger, 1999). Homosexual women, on the other hand, appear to be show rather more consistent cross-sex shifts (in the maletypical direction). These are evidenced in two neurocognitive tests-visuo-motor ability and verbal fluency (Hall and Kimura, 1995; Rahman et al., 2003a), in OAE's, and in AEPs (McFadden and Pasanen, 1998, 1999; McFadden and Champlin, 2000).

Some evidence suggests that these differences may arise, in part, from prenatal factors, primarily the levels of sex hormones experienced in utero. Such evidence relies almost entirely on 'proxy markers', which are somatic in nature, and ascribed to the organising effects of prenatal sex hormones. The second to fourth finger length ratio (or 2D:4D) is thought to be a negative correlate of prenatal testosterone and a positive correlate of prenatal estrogen (Manning, 2002). Homosexual women show reduced (i.e. masculinised) 2D:4D ratios compared to heterosexual women (Williams et al., 2000; McFadden and Schubel, 2002; Rahman and Wilson, 2003c). Studies in homosexual men have yielded inconsistent results; some reports demonstrate hyper-masculinised (lower) 2D:4D ratios (Robinson and Manning, 2000; Rahman and Wilson, 2003c) and others show feminised (higher) ratios (McFadden and Schubel, 2002; Lippa, 2003).

In analysis of data from heterosexuals and homosexual men of several ethnic groupings, Manning and Robinson (2003) suggest that this inconsistency may be due to greater variance in 2D:4D among heterosexuals compared to homosexual men. They suggest that homosexual men may show a 'conconstant' mean 2D:4D across ethnicity of roughly 0.96, although the exact mechanistic explanation for this is not apparent. Manning (2002) has recently summarised the evidence that androgens stimulates prenatal growth of the fourth finger whilst prenatal estrogen stimulates the growth of the second finger-a low 2D:4D ratio being indicative of greater exposure to male sex steroids (androgens) in the uterine environment. Thus, on the basis of the studies reviewed above, homosexuals of both sexes could be argued to be showing evidence of greater exposure to prenatal androgens. In support, the 2D:4D is sexually dimorphic with males showing lower ratios compared to females, a pattern which appears to be established by 2 years of age and is correlated with testosterone levels (Manning et al., 1998; Ronalds et al., 2002). A low 2D:4D has previously been associated with male-typical traits such as mental rotation ability, assertiveness, lefthandedness, and a predisposition towards autism (Wilson, 1983; Manning et al., 2000, 2001; Manning and Taylor, 2001). However, Putz et al. (2004) reported no associations between 2D:4D and several sex-linked traits in a large sample of male and female undergraduates, although lower ratios were related to non-heterosexual orientation in both sexes. This study employed uncorrected correlational analyses. Rather stronger evidence for prenatal hormonal influences on 2D:4D comes from individuals exposed to elevated levels of androgens before birth (such as in the condition congenital adrenal hyperplasia, or CAH). Brown et al. (2002) and Okten et al. (2002) reported that the 2D:4D ratio was masculinised (low values) in CAH females and hyper-masculinised in CAH males as compared to same-sex controls, in line with the notion that prenatal androgen exposure reduces the 2D:4D ratio. However, Buck et al. (2003) found no differences in 2D:4D between girls with CAH and control girls. That study differed from Brown et al. and Ökten et al. in that only the left hand was measured, and the measurements were made from radiographs. Radiographic techniques may omit relevant aspects of finger digits (such as the fat pads at the fingertips) that contribute to the sex difference in relative length. 2D:4D is also linked to variation in the androgen receptor gene (Manning et al., 2003) and the ratio of testosterone to estrogen taken from amniotic fluid during gestation are negatively associated with 2D:4D at 2 years of

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